FOURTH EDITION

EQUINE NUTRITION & FEEDING

DAVID FRAPE



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Equine Nutrition and Feeding

Fourth Edition

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Foreword

This is the fourth edition of *Equine Nutrition and Feeding* and represents a tremendous achievement of scholarship, containing an enormous amount of clearly presented detail, fully referenced and brought up-to-date with all the relevant research published in the last six years – a huge task.

Yet it remains very readable and will be of interest to all horse keepers across the world, for it covers all types of horse, all ages and conditions, in sickness and in health, kept for whatever purpose, sport, work, show or companionship.

David Frape has managed to combine details of all the relevant science with practical advice on the prevention and treatment of disease, the control of pests and parasites, and even the design of facilities, including horse boxes!

The book is well titled 'Nutrition and Feeding' because these are not the same thing and there are important practical issues in translating the one into the other. Thus it is designed to inform and help the whole range of those concerned, from the student to the most practical, from the rider to the breeder and all those involved in the care of the horse.

Such a comprehensive treatment would be a challenge for any author and it is a remarkable achievement to have kept a standard text easily assimilable and up-to-date over some 24 years, during which the science has advanced and the horse world has changed greatly.

The book can be used for easy reference or read as a narrative, helped by clear diagrams and tables.

Above all, the author recognises the individuality of horses: they, like the readers, are all different.

Professor Sir Colin Spedding
Chairman of the UK National Equine Forum

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Introduction to the Fourth Edition

During the past six years there has been a surprising amount of work on the dietary requirements and husbandry needs of the horse. This will undoubtedly lead to improvements in equine management in an increasingly competitive world where resources of raw materials, from land space to spices, become scarcer. The mass of work has encouraged me to revise the Third Edition in order to bring the evidence up to date as of September 2009. All chapters and sections of the book have been revised, with, in particular, clarification of the causes and control of several metabolic diseases. In addition, I trust that the principles of equine nutrition, which evolve gradually with time, and are based upon the best experimental evidence, are adequately covered.

The horse plays many roles throughout the world making difficult the determination of improvements in performance by experimental treatment. As Ralston (2007) states: "one of the most difficult problems in equine nutrition research is often the lack of objective and clinically relevant end points". However, adequately controlled experiments are much easier to conduct in equine than in human areas, where the factors and effects to be measured in population studies are confounded by uncontrollable, or only partially controllable, factors.

There have been two major objectives in preparing the Fourth Edition:

(1) to summarize, collate and integrate 647 new research reports and papers not previously reviewed in order to: (a) replace previous speculative evidence by more secure conclusions in several areas of equine husbandry, (b) indicate areas needing further investigation, and (c) inquire about issues not previously investigated by experiment.
(2) to integrate with this text the evidence, conclusions and recommendations published by the NRC in their excellent and comprehensive Sixth Revised Edition, "Nutrient Requirements of Horses" (NRC 2007) and, in particular, to compare their estimates for protein and energy with those of INRA (1984, 1990) at the feed level, where two dissimilar systems of measurement have been used.

David Frape

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I should like to thank my wife, Margery, for her support and Professor Sir Colin Spedding CBE, for his encouragement and for writing the Foreword.

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List of Abbreviations

acetyl-CoA	acetyl coenzyme A
ACTH	adrenocorticotropic hormone
ADAS	Agricultural Development and Advisory Service
ADF	acid detergent fibre
ADG	average daily liveweight gain
ADH	alcohol dehydrogenase
ADP	adenosine diphosphate
a.i.	active ingredient
ALP	alkaline phosphatase
ALT	alanine aminotransferase
AMP	adenosine monophosphate
ANP	atrial natriuretic peptide
AST	aspartate aminotransferase
ATP	adenosine triphosphate
BAL	bronchoalveolar lavage
BCAA	branched chain amino acids
BCS	body condition score (1 extremely emaciated – 9 extremely fat)
BE	base excess
BFGF	basic fibroblast growth factor
BHA	butylated hydroxyanisole
BHT	butylated hydroxytoluene
BMD	bone mineral density
BMR	basal metabolic rate
bpm	beats per minute
BSE	bovine spongiform encephalopathy
BSP	bromsulphalein™ (sulphobromophthalein)
BV	biological value
BW	body weight
CAFO	Concentrated Animal Feeding Operations
CCO	cytochrome c oxidase
CHO-F_R	rapidly fermentable carbohydrate
CF	crude fibre
CFU	colony-forming unit
CK	creatine kinase
COPD	chronic obstructive pulmonary disease
COX-2	cyclo-oxygenase-2
CP	crude protein
CRH	corticotropin-releasing hormone
CT	computed tomography
CTX-1	Type I collagen carboxy-terminal telopeptide
DCAB	dietary cation–anion balance
DCAD	dietary cation–anion difference
DCP	digestible crude protein
DDS	distiller's dark grains
DE	digestible energy
DHA	docosahexanoic acid
DM	dry matter
DMG	*N,N*-dimethylglycine
$DMSO_2$	dimethylsulphone
DNA	deoxyribonucleic acid
DOD	developmental orthopaedic disease
ECF	extracellular fluid
ED	energy digestibility
EDM	equine degenerative myeloencephalopathy
EE	ether extract
EG	ethylene glycol
EIPH	exercise-induced pulmonary haemorrhage
ELF	epithelial lining fluid
ELISA	enzyme-linked immunosorbent assay
EMND	equine motor neuron disease
EPA	Environmental Protection Agency (US)
EPA	eicosapentaenoic acid
EPM	equine protozoal myeloencephalitis

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ER	exertional rhabdomyolysis
EU	European Union
EVH-1/4	equine herpesvirus
FAD	flavin adenine dinucleotide
FDA	Food and Drug Agency
FE	fractional electrolyte excretion
FFA	free fatty acid
FMN	flavin mononucleotide
FOS	fructo-oligosaccharide
FSH	follicle-stimulating hormone
FTH	fast twitch, high oxidative
FT	fast twitch, low oxidative
GAG	glycosaminoglycan
GE	gross energy
GGT	gamma-glutamyltransferase
GI	gastrointestinal
Gla	γ-carboxyglutamic acid
GLC	gas-liquid chromatography
GLUT	glucose transporter
GnRH	gonadotropin-releasing hormone
GRAS	generally recognized as safe
GSH-Px	glutathione peroxidase
GSH	glutathione
GSSG	oxidised glutathione
HA	hyaluronic acid
Hb	haemoglobin
HCl	hydrochloric acid
HCN	hydrocyanic acid
HDS	hay dust suspension
HI	heat increment
HP	heat production
HPA	hypothalamo–pituitary–adrenal
HPLC	high performance liquid chromatography
HPP	hyperkalaemic periodic paralysis
HR	heart rate
5-HT	5-hydroxytryptamine (serotonin)
ICF	intracellular fluid
ICTP	telopeptide of type I collagen
IGER	Institute of Grassland and Environmental Research
IGF-1	insulin-like growth factor 1
IL-1	interleukin-1
IL-6	interleukin-6
IMP	inosine monophosphate
INRA	Institut National de la Recherche Agronomique
IR	insulin resistance
iu	international unit
i.v.	intravenous(ly)
LEM	leukoencephalomalacia
LTB_5	leukotriene B_5
LBS	*Lactobacillus* selection
LCT	lower critical temperature
LDH	lactic dehydrogenase
LH	luteinizing hormone
LPL	lipoprotein lipase
LPO	lipid hydroperoxide
LPS	lipopolysaccharide
MAD	modified acid detergent fibre
MADC	matières azotées digestibles corrigées (*or* cheval)
MCV	mean cell volume
MDA	malonyldialdehyde
ME	metabolizable energy
MRT	mean retention time
MRSA	methicillin-resistant *Staphylococcus aureus*
MSG	monosodium glutamate
MSM	methyl sulphonyl methane
NAD	nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotide phosphate
NDF	neutral detergent fibre
NE	net energy
NEFA	nonesterified fatty acid
NFE	nitrogen-free extractive
NIAB	National Institute of Agricultural Botany, Cambridge
NIS	nutritionally improved straw
NO	nitric oxide
NPN	non-protein nitrogen
NRC	National Research Council
NSAID	non-steroidal anti-inflammatory drug
NSC	non-structural carbohydrate
NSP	non-starch polysaccharides
NSHP	nutritional secondary hyperparathyroidism
OC	osteochondrosis
OCD	osteochondritis dissecans
OM	organic matter
OMD	organic matter digestibility
P_{osm}	plasma osmolality
PABA	*p*-aminobenzoic acid
PAF	platelet-activating factor
PCV	packed cell volume
PCr	phosphocreatine
PDH	pyruvate dehydrogenase
PGE_2	prostaglandin E_2
PICP	propeptide of type I procollagen
PN	parenteral nutrition

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PSSM	polysaccharide storage myopathy
PTH	parathyroid hormone
PTH-rP	parathyroid hormone-related protein
PUFA	polyunsaturated fatty acid
PV	plasma volume
RAO	recurrent airway obstruction
RBC	red blood cell
RCHV	red cell hypervolaemia
RDR	relative dose response
RER	respiratory exchange ratio
RH	relative humidity
RNA	ribonucleic acid
ROS	reactive oxygen species
RQ	respiratory quotient
RVO	recovered vegetable oil
s.d.	standard deviation
SDH	sorbitol dehydrogenase
SE	standard error
SET	standardized exercise test
SG	specific gravity
SI	insulin sensitivity
SID	strong ion difference
SOD	superoxide dismutase
ST	slow twitch, high oxidative
STP	standard temperature and pressure
T_3	triiodothyronine
T_4	thyroxine
TAG	triacylglycerol
TB	Thoroughbred
TBA	thiobarbituric acid
TBAR	thiobarbituric acid reactive substance
TCA	tricarboxylic acid
TLV	threshold limiting value
TNF-α	tumour necrosis factor-α
TNZ	thermoneutral zone
TPN	total parenteral nutrition
TPP	thiamin pyrophosphate
TRH	thyrotropin-releasing hormone
TSH	thyroid-stimulating hormone (thyrotropin)
TXB_2	thromboxane
UFC	unité fourragère cheval (horse feed units)
UKASTA	UK Agricultural Supply Trade Association
VFA	volatile fatty acid
VLDL	very low density lipoprotein
WBC	white blood cell (leukocyte)

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1
The Digestive System

A horse which is kept to dry meat will often slaver at the mouth. If he champs his hay and corn, and puts it out again, it arises from some fault in the grinders … there will sometimes be great holes cut with his grinders in the weaks of his mouth. First file his grinders quite smooth with a file made for the purpose.

Francis Clater, 1786

Horses are ungulates and, according to J.Z. Young (1950), members of the order Perissodactyla. Other extant members include asses, zebras, rhinoceroses and tapirs. Distinctive characteristics of the order are the development of the teeth, the lower limb with the peculiar plan of the carpus and tarsus bones and the evolution of the hind gut into chambers for fermentation of ingesta. Each of these distinctive features will play significant roles in the discussions in this text.

The domesticated horse consumes a variety of feeds, ranging in physical form from forage with a high content of moisture to cereals with large amounts of starch, and from hay in the form of physically long fibrous stems to salt licks and water. In contrast, the wild horse has evolved and adapted to a grazing and browsing existence, in which it selects succulent forages containing relatively large amounts of water, soluble proteins, lipids, sugars and structural carbohydrates, but little starch. Short periods of feeding occur throughout most of the day and night, although generally these are of greater intensity in daylight. In domesticating the horse, man has generally restricted its feeding time and introduced unfamiliar materials, particularly starchy cereals, protein concentrates and dried forages. The art of feeding gained by long experience is to ensure that these materials meet the varied requirements of horses without causing digestive and metabolic upsets. Thus, an understanding of the form and function of the alimentary canal is fundamental to a discussion of feeding and nutrition of the horse.

THE MOUTH

Eating rates of horses, cattle and sheep

The lips, tongue and teeth of the horse are ideally suited for the prehension, ingestion and alteration of the physical form of feed to that suitable for propulsion through the gastrointestinal (GI) tract in a state that facilitates admixture with digestive juices. The upper lip is strong, mobile and sensitive and is used during grazing to place forage between the teeth; in the cow the tongue is used for this purpose. By contrast, the horse's tongue moves ingested material to the cheek teeth for grinding. The lips are also used as a funnel through which water is sucked.

As distinct from cattle, the horse has both upper and lower incisors enabling it to graze closely by shearing off forage. More intensive mastication by the horse means that the ingestion rate of long hay, per kg of metabolic body weight (BW), is three to four times as fast in cattle and sheep than it is in ponies and horses, although the number of chews per minute is similar, according to published observations (73–92 for horses and 73–115 for sheep) for long hays. The dry matter (DM) intake per kg of metabolic BW for each chew is then 2.5 mg in horses (I calculate it to be even less – author) and 5.6–6.9 mg in sheep. Consequently, the horse needs longer daily periods of grazing than do sheep. The lateral and vertical movements of the horse's jaw, accompanied by profuse salivation, enable the cheek teeth to comminute long hay to a large extent and the small particles coated with mucus are suitable for swallowing. Sound teeth generally reduce hay and grass particles to less than 1.6 mm in length. Two-thirds of hay particles in the horse's stomach are less than 1 mm

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across, according to work by Meyer and colleagues (Meyer *et al.* 1975b).

The number of chewing movements for roughage is considerably greater than that required for chewing concentrates. Horses make between 800 and 1200 chewing movements per 1 kg concentrates, whereas 1 kg long hay requires between 3000 and 3500 movements. In ponies, chewing is even more protracted – they require 5000–8000 chewing movements per 1 kg concentrates alone, and very many more for hay (Meyer *et al.* 1975b). Horses given a hay diet chewed 40,000 times/day compared with 10,000 times/day for those fed on pellets (Houpt *et al.* 2004). Hay chewing, cf. pellets, by both horses and ponies, is protracted, with a lower chewing-cycle frequency, as the mandibular displacement is greater, both vertically and horizontally with an effect on faecal particle dimensions (Brøkner *et al.* 2009). Clayton *et al*. (2003) concluded that the development of sharp enamel points is more likely with a high concentrate diet.

Mature and young horses have a maximal daily DM intake of 3.0–3.2% of BW, although the average is lower (NRC 2007). Ponies have a higher voluntary DM intake than horses; Pearson *et al.* (2001) found ponies ate 3.9 kg/100 kg BW alfalfa hay while Argo *et al.* (2002) recorded 5.1 kg fresh weight/100 kg BW of a meal of 60% hay and 40% concentrate pellets. Such high intakes might occur with high quality feed after a period of feed restriction, as particle retention time is greater for poor quality feed (Pearson *et al.* 2001). The addition of 35% short chaff (<2 cm) to sweet coarse mix slowed the rate of consumption and doubled the eating time, but increased the eating rate (Harris *et al.* 2005) and the addition of chopped straw, either 2.5 or 4 cm in length at rates of 10–30% of a pelleted diet mixed with chopped alfalfa, increased the time to eat 1 kg wet matter (Ellis *et al.* 2005). These observations are important for an understanding of healthy digestion.

Dentition

As indicated above, teeth are vital to the well-being of horses. Diseased teeth are an encumbrance. Primary disorders of the cheek teeth represented 87% of the dental disorders in 400 horses (Dixon *et al*. 2000a). The disorders included abnormalities of wear, traumatic damage, and fractures from which the response to treatment was good. Dental and head pain have specific behavioural indicators, including altered eating patterns, anorexia, feed refusal and quidding (Ashley *et al.* 2005) and cause digestive disturbances and colic. The prevalence of dental disorders amongst donkeys increases with age, and is especially prominent at 15–20 years of age. Dental disease is associated with poor body condition score (BCS), previous episodes of colic, diastemata (a gap between adjacent teeth) and wave-, smooth- and step-mouth (Du Toit *et al* 2009a,b).

Apparent fibre digestibility, the proportion of faecal short fibre particles and plasma free fatty acids (FFAs) were all increased after dental correction in mares. Consequently, diseased and badly worn teeth, as in the geriatric horse, can limit the horse's ability to handle roughage, that compromises general health. Infections of cheek teeth are not uncommon and Dixon *et al*. (2000b) found that nasal discharge was more frequent with infections of caudal than with rostral maxillary teeth. Hudson *et al* (2006) describe cases of dysphagia in horses caused by a buccal abscess, a lingual abscess, a retropharyngeal foreign body and an oesophageal obstruction. Windley *et al* (2009b) reported that both two- and three-dimensional computed tomography (CT) were valuable as clinical diagnostic tools in detection of dental lesions and in selection of appropriate treatment.

The apparent digestibility of the protein and fibre in hay and grain is reduced if the occlusal angle of premolar 307 is greater than 80° relative to the (flattened) vertical angle (Ralston *et al*. 2001). However, no adverse effects were noted by Carmalt & Allen (2008) where normal variation occurs in occlusial characteristics; they found no relationships between cheek tooth occlusal morphology, apparent feed digestibility, and the reduction in particle size of three different hay-based feeds.

The normal horse has two sets of teeth. The first to appear, the deciduous, or temporary milk, teeth erupt soon after birth and are replaced during growth by the permanent teeth. The permanent incisors and cheek teeth erupt continuously to compensate for wear, and their changing form provides a basis for assessing the age of a horse. In the gap along the jaw between the incisors and the cheek teeth the male horse normally has a set of small canine teeth. The gap, by happy chance, securely locates the bit. The dental formulae and configuration of both deciduous and permanent teeth are given in Figure 1.1. The lower cheek teeth are implanted in the mandible in two straight rows that diverge towards the back. The space between the rows of teeth in the lower jaw is less than that separating the upper teeth (Figure 1.1). This accommodates a sideways, or circular, movement of the jaw that effectively shears feed. The action leads to a distinctive pattern of wear of the biting surface of the exposed crown. This pattern results from the differences in hardness which characterize the three materials (cement, enamel and dentine) of which teeth are composed. The enamel, being

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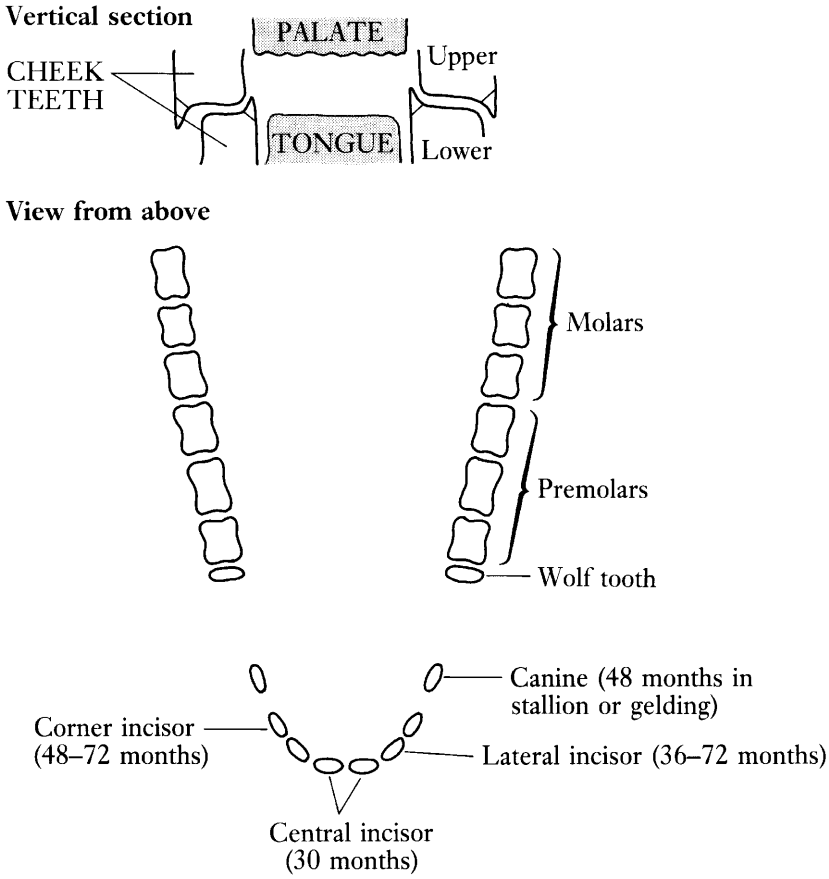


Figure 1.1 Configuration of permanent teeth in the upper or lower jaw (the molars and premolars in the lower jaw are slightly closer to the midline). The deciduous teeth on each side of each jaw consist of three incisors, one canine, and three molars. The deciduous canines are vestigial and do not erupt. The wolf teeth (present in the upper jaw of about 30% of fillies and about 65% of colts) are often extracted, as their sharp tips can injure cheeks when a snaffle bit is used. Months (in parentheses) are approximate ages at which permanent incisors and canines erupt, replacing the deciduous teeth.

the hardest, stands out in the form of sharp prominent ridges. It is estimated that the enamel ridges of an upper cheek tooth in a young adult horse, if straightened out, would form a line more than 30 cm (1 ft) long. This irregular surface provides a very efficient grinding organ.

Horses and ponies rely more on their teeth than we do. The human diet could be said to consist mainly of concentrates, which require much less chewing than does roughage. A dietary regime consisting mainly of concentrate feeds is associated with smaller mandibular excursions during chewing by the horse. This could imply that, during training, more frequent dental prophylactic treatment is needed to avoid development of dental irregularities (Bonin *et al*. 2007). Even among herbivores, horses and ponies depend to a far greater extent on their teeth than do the domesticated ruminants – cattle, sheep and goats.

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Ruminants, as discussed in 'Eating rates of horses, cattle and sheep' above, swallow grass and hay with minimal chewing and then depend on the activity of bacteria in the rumen to disrupt the fibre. The fibre is then much more readily fragmented during chewing the cud.

Saliva

The physical presence of feed material in the mouth stimulates the secretion of a copious amount of saliva. Some 10–12 L are secreted daily in a normally fed horse. This fluid seems to have no digestive enzyme activity, but its mucus content enables it to function as an efficient lubricant preventing 'choke'. Its bicarbonate content, amounting to some 50 mEq/L, provides it with a buffering capacity. The production of bicarbonate and sodium chloride in the saliva is directly proportional to the rate of secretion. The continuous secretion of saliva during eating seems to buffer the digesta in the proximal region of the stomach, permitting some microbial fermentation with the production of lactate. This has important implications for the well-being of the horse (see Chapter 11).

Obstruction of the oesophagus by impacted feed or foreign bodies is not uncommon, but attempts to pass a nasogastric tube are not justified, as most cases respond to conservative treatment. For cases of more than 48 h duration a cuffed nasogastric tube is advocated, although the value of oxytocin use is unclear (Duncanson 2006). To facilitate nutritional support during treatment of oesophageal perforation, a cervical oesophagotomy tube is placed and advanced into the stomach (Read *et al.* 2002). An enteral diet includes an electrolyte mixture (partly to compensate for salivary electrolyte losses through the oesophagotomy site), sucrose (1.2 kg/day), casein, canola rapeseed oil (1.1 L/day) and dehydrated alfalfa pellets. A nasogastric tube is subsequently introduced to allow repair of the oesophagotomy site.

THE STOMACH AND SMALL INTESTINE

The first quantitative aspects of digestion were demonstrated by Waldinger in 1808 with the passage of capsulated feedstuff through the intestines. Intensive studies concerning the physiology of digestion were started in Paris around 1850 by Colin, but they were continued predominately from 1880 in Dresden by Ellenberger and Hofmeister who investigated the mouth, stomach and small intestine. Scheunert continued with work on the large intestine in Dresden and Leipzig until the 1920s. Although the apparent digestibility of cellulose was appreciated by 1865 it took another 20 years for the discovery of the process of microbial digestion in the equine large intestine. Until 1950 most routine equine digestibility experiments were conducted in Germany, France and the USA (Klingeberg-Kraus 2001), while comparative studies were conducted by Phillipson, Elsden and colleagues at Cambridge in the 1940s.

Development of the gastrointestinal (GI) tract and associated organs

The GI tract tissue of the neonatal foal weighs only 35 g/kg BW, whereas the liver is large, nearly in the same proportion to BW, acting as a nutrient store for the early critical days. By six months of age the GI tract tissue has proportionately increased to 60 g/kg BW, whereas the liver has proportionately decreased to about 12–14 g/kg BW. By 12 months both these organs have stabilized at 45–50 g/kg BW for the GI tract and 10 g/kg BW for the liver. Organ size is also influenced by the activity of the horse. After a meal, the liver of mammals generally increases rapidly in weight, probably as a result of glycogen storage and blood flow. In the horse the consumption of hay has less impact on liver glycogen, so that following a meal of hay the liver weighs only three-quarters of that following mixed feed. Moreover, during and immediately after exercise the GI tract tissue weighs significantly less than in horses at rest, owing to the shunting of blood away from the mesenteric blood vessels to the muscles. At rest, about 30% of the cardiac output flows through the hepatic portal system. These aspects are discussed further in Chapter 9.

Surprisingly, the small intestine does not materially increase in length from 4 weeks of age, whereas the large intestine increases with age, the colon doing so until 20 years at least. The distal regions of the large intestine continue extension to a greater age than do the proximal regions. This development reflects the increasing reliance of the older animal on roughage. In an adult horse of 500 kg BW the small intestine is approximately 16 m in length, the caecum has a maximum length of about 0.8 m, the ascending colon 3 m and the descending colon 2.8 m.

Transit of digesta through the GI tract

The residence time for ingesta in each section of the GI tract allows for its adequate admixture with GI secretions, for hydrolysis by digestive enzymes, for absorption of the resulting products, for fermentation of resistant material by bacteria and for the absorption of the products of that fermentation. Transit time through the GI tract is normally considered in three phases, owing to their entirely different characteristics. These phases are:

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(1) expulsion rate from the stomach into the duodenum after a meal;
(2) rate of passage through the small intestine to the ileocaecal orifice;
(3) retention time in the large intestine.

The first of these will be considered below in relation to gastric disorders. Rate of passage of digesta through the small intestine varies with feed type. On pasture this rate is accelerated, although a previous feed of hay causes a decrease in the rate of the succeeding meal, with implications for exercise (see Chapter 9). Roughage is held in the large intestine for a considerable period that allows microbial fermentation time to break down structural carbohydrates. However, equine GI transit time of the residue of high fibre diets is less than that of low fibre diets of the same particle size, in common with the relationship found in other monogastric animals.

Digestive function of the stomach

The stomach of the adult horse is a small organ, its volume comprising about 10% of the GI tract (Figure 1.2, Plate 1.1; Meyer *et al.* 1993a). In the suckling foal, however, the stomach capacity represents a larger proportion of the total alimentary tract. Most digesta are held in the stomach for a comparatively short time, but this organ is rarely completely empty and a significant portion of the digesta remain in it for 2 to 6 h. Some digesta pass into the duodenum shortly after eating starts, when fresh ingesta enter the stomach. Expulsion into the duodenum is arrested as soon as feeding stops. When a horse drinks, a high proportion of the water passes along the curvature of the stomach wall so that mixing with digesta and dilution of the digestive juices it contains are avoided. This process is particularly noticeable when digesta fill the stomach.

The entrance to the stomach is guarded by a powerful muscular valve called the cardiac sphincter. Although a horse might feel nauseated, it rarely vomits, partly because of the way this valve functions. This too has important consequences. Despite extreme abdominal pressure the cardiac sphincter is reluctant to relax in order to permit the regurgitation of feed or gas. On the rare occasions when vomiting does occur, ingesta usually rush out through the nostrils, owing to the existence of a long soft palate. Such an event could indicate a ruptured stomach.

Gastric anatomy differentiates the equine stomach from that of other monogastrics. Apart from the considerable strengths of the cardiac and pyloric sphincters, almost half the mucosal surface is lined with squamous, instead of glandular, epithelium. The glandular mucosa is divided into fundic and pyloric regions (Figure 1.2). The fundic mucosa contains both parietal cells that secrete hydrochloric acid (HCl) and zymogen cells which secrete pepsin, while the polypeptide hormone gastrin is secreted into the blood by the pyloric region. The hormone's secretion is triggered by food, and equine studies in Sweden have shown that the gastric phase of release is triggered by distension of the stomach wall, rather than the sight of feed. The greatest and most prolonged gastrin secretion occurs when horses eat hay freely (A. Sandin, personal communication). In the horse, gastrin does not act as a stress hormone. The hormone strongly stimulates secretion of gastric acid and the daily secretion and release of gastric juice into the stomach amounts to some 10–30 L. Secretion of gastric juice continues even during fasting, although the rate seems to vary from hour to hour (see Chapter 11, Laminitis).

HCl secretion continues, but declines gradually at a variable rate when the stomach is nearly empty and hence at that time the pH is around 1.5–2.0. The pH rises rapidly during a subsequent meal, especially that of grain only, partly as a consequence of a delay in gastrin secretion, compared with the more rapid gastrin response to hay. The act of eating stimulates the flow of saliva – a source of sodium, potassium, bicarbonate and chloride ions. Saliva's buffering power retards the rate at which the pH of the stomach contents decreases. This action, combined with a stratification of the ingesta, brings about marked differences in the pH of different regions (about 5.4 in the fundic region and 2.6 in the pyloric region).

Fermentation, primarily yielding lactic acid, occurs in the oesophageal and fundic regions of the stomach, but particularly in the part known as the *saccus caecus*, which is lined by squamous cells. As digesta approach the pylorus at the distal end of the stomach, the gastric pH falls, owing to the secretion of HCl, which potentiates the proteolytic activity of pepsin and arrests that of fermentation. The activity of pepsin in the pyloric region is some 15–20 times greater than in the fundic region. Because of the stomach's small size and the consequentially relatively short dwelling time, the degree of protein digestion is low.

Gastric malfunction

Meyer and his colleagues in Hanover (Meyer *et al.* 1975a) have made detailed investigations of the flow of ingesta and digesta through the GI tract of horses. Their thesis is that abnormal gastric fermentation occurs when the postprandial dry matter content of the stomach is particularly high and a low pH is not achieved. There is, nevertheless,

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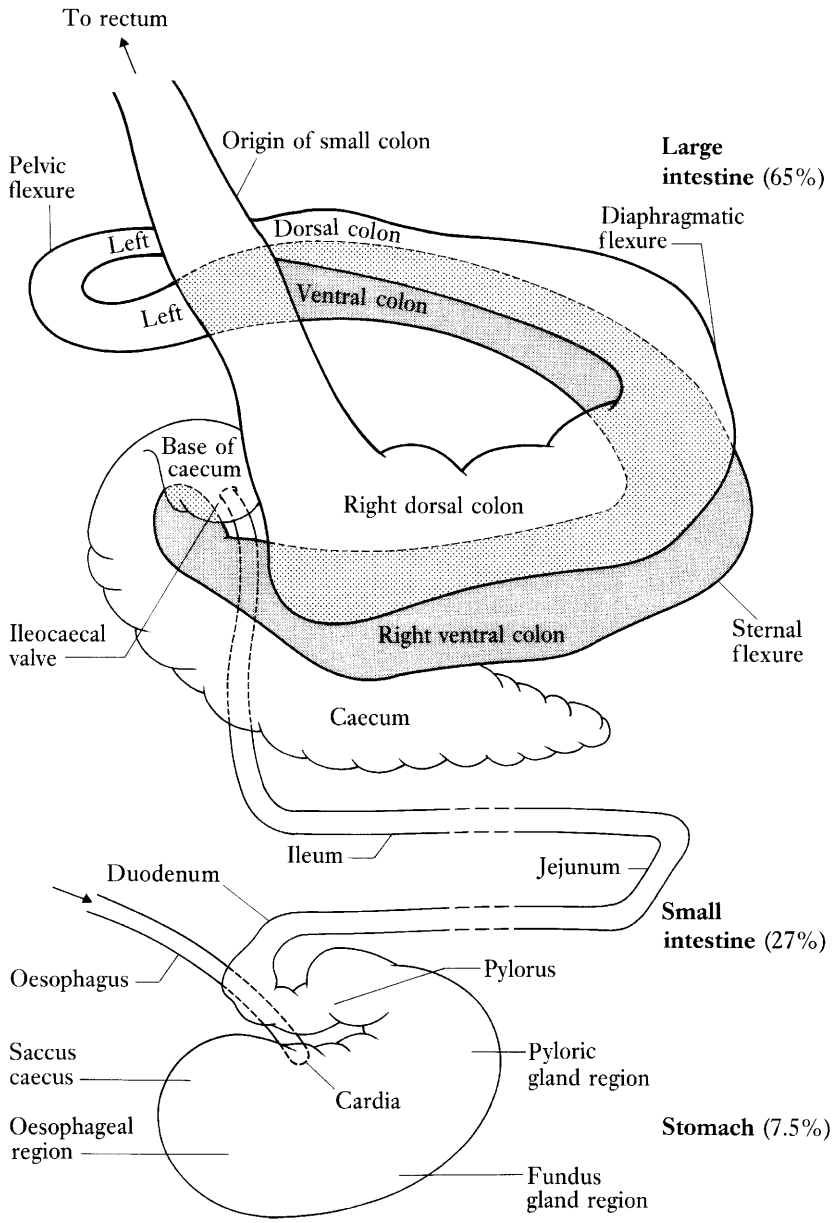


Figure 1.2 GI tract of adult horse (relative volumes are given in parentheses).

considerable layering and a differentiation in pH between the *saccus caecus* and pyloric region. Fermentation is therefore a normal characteristic of regions with a higher pH, in which the larger roughage particles tend to float. However, the dry-matter content is lower following a meal of roughage than it is following one of cereals. After meals of 1 kg loose hay and 1 kg pelleted cereals the resulting gastric dry matters were 211 and 291 g/kg contents, respectively.

The Hanover group compared long roughage with that which was chopped, ground or pelleted and observed that, as particle size of roughage decreased, the gastric dry matter contents decreased from 186 to 132 g/kg contents and the rate of passage of ingesta through the stomach

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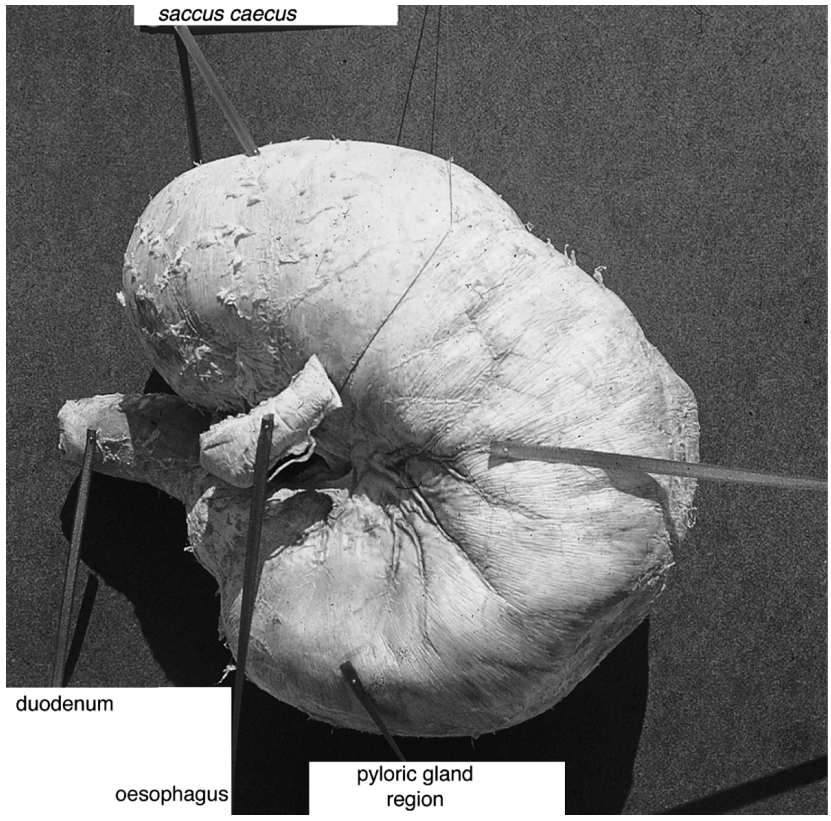


Plate 1.1 Stomach of a 550 kg TB mare, capacity 8.4 l, measuring about 20 × 30 × 15 cm. Acid fermentation of stomach contents takes place in the *saccus caecus* (*top*).

increased. The reason for this is probably that it is the finely divided material in a gastric slurry which passes first to the intestines. The slurry is forced into the duodenum by contractions termed *antral systole* at the rate of about 3 g/min. Nevertheless, particle size is generally small as a result of comminution by the molars. With larger meals of pelleted cereal, up to 2.5 kg/meal, the gastric dry matter content increased to 400 g/kg, and the pH to 5.6–5.8, for as long as 2–3 h after consumption. The dry matter accumulated faster than it was ejected into the duodenum and, as cereals could be consumed more rapidly than hay with a lower secretion of saliva, the dry matter of the stomach was higher following large meals of cereals. As much as 10–20% of a relatively small meal of concentrates (given at the rate of 0.4% BW) remains in the stomach 6 h after feeding ponies. A high dry-matter content acts as a potent buffer of the HCl in gastric juice and the glutinous nature of cereal ingesta inhibits the penetration of cereal ingesta by gastric juices.

Together with the delay in gastrin release during a cereal meal, these factors could account for the failure of the postprandial pH to fall to levels that inhibit further microbial growth and fermentation. Lactic-acid producing bacteria (*Lactobacilli* and *Streptococci*) thrive (see Chapter 5, Probiotics). Whereas *Streptococci* do not produce gas, some *Lactobacillus* species produce carbon dioxide, thrive at a pH of 5.5–6.0 and even grow in the pH range 4.0–6.8, with some strains growing in conditions as acid as pH 3.5. During the first hour after a starchy concentrate meal there was a linear increase in total gastric anaerobes, the concentrations of *Lactobacilli*, *Streptococci* and lactate-utilizing bacteria were, respectively, 5.52, 4.82 and 6.95 $\log_{10}$ colony-forming units (cfu)/mL. Lactate (mostly L-lactate) and volatile fatty acid (VFA) concentrations increased linearly over a period of up to 210 min (Varloud *et al.* 2007). The pH of the gastric contents will even increase to levels that permit non-lactic-acid-producing, gas-producing bacteria to survive, producing large amounts

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of VFAs. Gas production at a rate greater than that at which it can be absorbed into the bloodstream causes gastric tympany, and even gastric rupture, and hence it is desirable that the postprandial gastric pH falls sufficiently to arrest most bacterial growth and to kill potential pathogens.

Gastric ulceration

Ulceration and erosion occur in the gastric stratified squamous mucosa, particularly that adjacent to the *margo plicatus*, as the squamous epithelial mucosa lacks the protective processes, especially the mucus–bicarbonate barrier, possessed by the glandular mucosa (see Chapter 11 for a discussion of aetiology). This squamous mucosa exists in a potentially acidic environment and is susceptible to damage by HCl and pepsin. Bile, which is found in significant amounts in the stomach during long fasts, increases the risk of damage (Berschneider *et al.* 1999). Routine post-mortem examination of 195 Thoroughbreds (TBs) in Hong Kong (Hammond *et al.* 1986) revealed that 66% had suffered gastric ulceration. In TBs in training, the frequency was 80%, whereas it was only 52% among those that had been retired for a month or more. The lesions seem to be progressive during training, but to regress during retirement. Husted *et al.* (2008) gave horses coastal Bermuda hay *ad libitum* and commercial coarse mix under two housing schemes: a box stall bedded with wood shavings and a grassed paddock. The proximal and ventral gastric pH values were similar in both groups. Ventral pH was uniform throughout the study, while the proximal pH demonstrated a 24 h circadian pattern in both treatments. The authors concluded that housing alone during training does not explain the increased risk of squamous ulcer development. There was a low prevalence of gastric lesions in Caspian horses that had received anthelmintic drugs in Iran (Moghaddam *et al.* 2008). The prevalence was found to increase with increasing exercise and to occur in the glandular region in those having long-term treatment with nonsteroidal anti-inflammatory drugs (NSAIDs; NSAIDs reduce prostaglandin secretion, thus inhibiting mucus production. author). Observations by the research group in Hanover showed that clinical signs of periprandial colic and bruxism (grinding of teeth) were more pronounced in horses with more severe gastric lesions from diffuse ulcerative gastritis.

Concretions

Soluble tannins present in the persimmon fruit bind proteins, such as digestive enzymes. In the presence of gastric HCl the tannic acid polymerises to form a coagulum, described as a phytobezoar, that includes persimmon seeds and dietary fibre. This concretion can cause mechanical damage and abrasion to the mucosal lining of the stomach (Hurtado *et al.* 2007, Johnson & Kellam, 2007)

Foals

Lesions are not restricted to adult horses. Neonatal foals of 2 days of age are able to produce highly acidic gastric secretions; the mean pH of the glandular mucosal surface and fluid contents of 18 foals at 20 days of age were 2.1 and 1.8, respectively (Murray & Mahaffey 1993). Lesions in suckling foals include neonatal gastro-duodenal ulceration, gastric glandular ulceration and squamous mucosal ulceration (Lester 2004). Gastric mucosal inflammation and ulceration of foals post-weaning has been associated with cribbing, treated and reduced by antacids, and resolves on ulcer healing (Nicol *et al.* 2002). The intragastric pH was raised from a mean of 3.19 ± 1.5 to 6.20 ± 0.93 in eight clinically ill, full term, neonatal foals by one dose of omeprazole (4 mg/kg BW orally; Javsicas & Sanchez 2008).

Although treatment with omeprazole (Franklin *et al.* 2008), cimetidine or ranitidine, is effective, *Helicobacter* infection plays no part in the equine syndrome (as *H. pylori* frequently does in man, where the organisms shrewdly protect themselves from acid by urease secretion with an acid pH optimum). *H. equorum* is able to colonize the equine lower bowel without apparent GI disease or other pathology (Moyaert *et al.* 2007). Despite the greater risk to concentrate-fed horses, both periprandial microbial activity and the pH of gastric contents are higher in concentrate-fed than in hay-fed animals. Moreover, the pH is lowest during a fast. Horses are continuous secretors of gastric acid and feed deprivation for repeated 24-h periods, alternating with 24-h periods of *ad libitum* access to hay, induces gastric squamous tissue ulceration, associated with an intragastric pH of 1.6 during deprivation, cf. 3.1 during hay feeding (Murray & Schusser 1993).

Digestion in the small intestine

The 450 kg horse has a relatively short small intestine, 21–25 m in length, through which transit of digesta is quite rapid, some appearing in the caecum within 45 min after a meal. Much of the digesta moves through the small intestine at a rate of nearly 30 cm/min. and its motility is under both neural and hormonal control. When a liquid marker was instilled into the stomach of a pony, 50% reached the distal ileum in 1 h, and by 1.5 h after instillation 25% was present in the caecum (Merritt 1992, personal. communi-

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cation); transit of feed from stomach to caecum is much more rapid following a fast.

To estimate transit time monofilament polyester bags with a pore size of 41 µm containing 200 or 130 mg feed can be introduced into the stomach via a nasogastric tube and recovered in the faeces after transit times of 10–154 h. Transit times and digestibility in the small intestine are estimated following capture of the bags from near the ileocaecal valve with a magnet (Hyslop *et al*. 1998d). Caution should, however, be exercised in the interpretation of precaecal N-digestibility values, which can be considerably higher from the mobile bag cf. the ileal-fistula technique (Macheboeuf *et al*. 2003).

In consequence of the rapid transit of ingesta through the small intestine, it is surprising how much digestion and absorption occur there. Mechanisms of absorption have been studied by Cehak *et al.* (2009). Although differences in the composition of digesta entering the large intestine can be detected with a change in diet, it is a considerably more uniform material than that entering the rumen of the cow. This fact has notable practical and physiological significance in the nutrition and well-being of the horse. The material leaving the small intestine consists of fibrous feed residues, undigested feed starch and protein, microorganisms, intestinal secretions and cell debris.

Digestive secretions

Large quantities of pancreatic juice are secreted as a result of the presence of food in the stomach in response to stimuli mediated by vagal nerve fibres, and by gastric HCl in the duodenum stimulating the release of the polypeptide hormone secretin into the blood. In fact, although secretion is continuous, the rate of pancreatic juice secretion increases four- to five-fold when feed is first given. This secretion, which enters the duodenum, has a low order of enzymatic activity, although some active trypsin is present, but provides large quantities of fluid and sodium, potassium, chloride and bicarbonate ions. There is conflicting evidence for the presence of lipase in pancreatic secretions, and bile, secreted by the liver, probably has a greater, although different, influence on fat digestion. The stimulation of pancreatic juice secretion does not increase its bicarbonate content, as in other species. The bicarbonate content of digesta increases in the ileum, where it is secreted in exchange for chloride, so providing a buffer to large intestinal VFAs (see 'Products of fermentation', this chapter).

The horse lacks a gall bladder, but stimulation of bile is also caused by the presence of gastric HCl in the duodenum. Secretion of pancreatic juice and bile ceases after a fast of 48 h. Bile is both an excretion and a digestive secretion. As a reservoir of alkali it helps preserve an optimal reaction in the intestine for the functioning of digestive enzymes secreted there. In the horse, the pH of the digesta leaving the stomach rapidly rises to slightly over 7.0.

Carbohydrates

The ability of the horse to digest soluble carbohydrates and the efficiency of the mucosal monosaccharide transport systems of the small intestine have been established in a series of oral disaccharide and monosaccharide tolerance tests (Roberts 1975b). This ability is important to an understanding of certain digestive upsets to which the horse is subject.

A high proportion of the energy sources consumed by the working horse contain cereal starches. These have relatively long branched chains, the links of which are α-D-glucose molecules joined as shown in Figure 1.3. Absorption into the bloodstream depends on the disruption of the bonds linking the glucose molecules. This is contingent entirely upon enzymes secreted in the small intestine. These are held on the brush border of the villi in the form of α-amylase (secreted by the pancreas) and as α-glucosidases (secreted by the intestinal mucosa) (see Table 1.1).

The secretions of the pancreatic juice release sufficient oligosaccharides for further hydrolysis by the brush border enzymes at the intestinal cell surface (Roberts 1975a). Active carrier-mediated mechanisms then transport the final hexose products across the intestinal cell for uptake in the hepatic portal system. The digestive system can, however, be overloaded. Ponies weighing 266 kg BW were given 4 kg feed/day, as oat hulls : naked oats 2 : 1 (i.e. 1.33 kg naked oats). This led to changes in intracaecal fermentation, indicating that oat starch was reaching that organ, although the intracaecal pH did not decrease below 6.5. A comparison of unmolassed sugar beet pulp, hay cubes, soya hulls, or a 2 : 1 mixture of oat hulls : naked oats, indicate that beet pulp is subject to greater hind-gut fermentation than the other feeds (Moore-Colyer *et al*. 1997, Table 1.2). Starch fermentation in the hind-gut and its consequences are discussed below and in Chapters 2 and 11.

The concentration of α-amylase in the pancreatic juice of the horse is only 5–6% of that in the pig, whereas the concentration of α-glucosidase is comparable with that in many other domestic mammals. Thus, the addition of supplemental amylase from bacterial sources increases the digestion of ground maize (Meyer *et al.* 1993b) and

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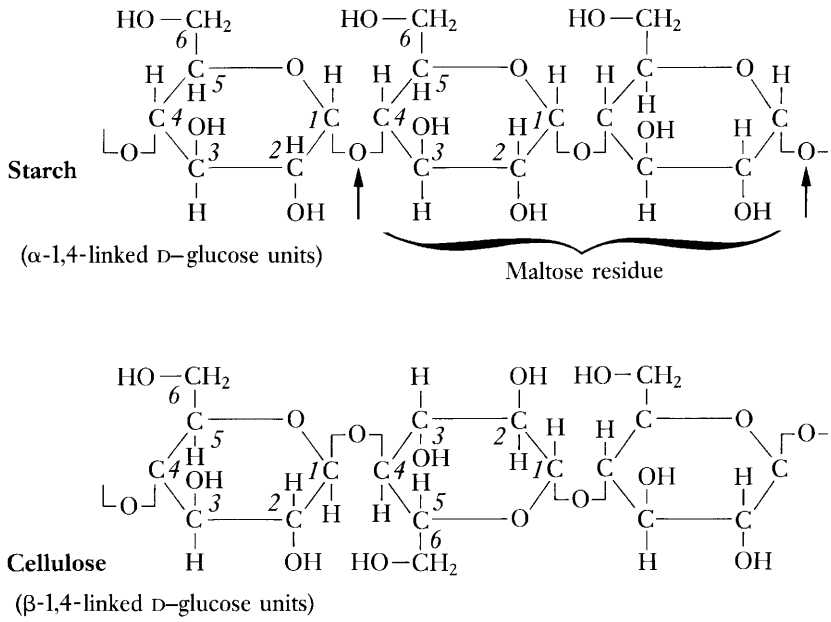


Figure 1.3 Diagrammatic representation of three glucose units in two carbohydrate chains (the starch granule also contains amylopectin, which has both 1–4 linkages and 1–6 linkages). *Arrows* indicate site of intermediate digestion.

Table 1.1 Carbohydrate digestion in the small intestine.

Substrate	Enzyme	Product
Starch	α-Amylase	Limiting dextrins (about 34 glucose units)
Limit dextrins	α-Glucosidases (glucoamylase, maltase and isomaltase)	Glucose
Sucrose	Sucrase	Fructose and glucose
Lactose	Neutral-β-galactosidase (lactase)	Glucose and galactose

Table 1.2 *In sacco* organic matter and crude protein (CP) disappearance from the DM or from the CP, in polyester bags during passage from the stomach to the caecum of Welsh cross ponies (Moore-Colyer *et al.* 1997).

	Component disappearance from the small intestine		
	Organic matter g/kg (DM)	Crude protein g/kg (CP)	Digestible crude protein g/kg (DM)
Sugar beet pulp	185	296	30
Hay cubes	294	521	52
Soya hulls	239	597	60
Oat hulls : naked oats, 2 : 1	337	771	54

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elevates the glycaemic response to a triticale diet (Richards *et al.* 2004).

The α-glucosidases (disaccharidases) include sucrase, a disaccharidase present in concentrations five times that of glucoamylase and capable of digesting sucrose. Sucrase activity is highest in the proximal small intestine where its activity is similar to that reported for other non-ruminant species; by contrast, maltase activity is extremely high in comparison with that reported for other species. Maltase activity is expressed similarly in proximal, mid- and distal regions. D-glucose and D-galactose are transported across the equine intestinal brush border membrane by a high-affinity, low-capacity, Na^+/glucose cotransporter type 1 isoform, with rates of transport in the order: duodenum > jejunum > ileum (Dyer *et al*. 2002).

Another important disaccharidase in the intestinal juice is the β-glucosidase, neutral β-galactosidase (neutral or brush-border lactase), which is necessary for the digestion of milk sugar in the foal. This enzyme has a pH optimum of around 6.0. Whereas functional lactase is expressed all along the small intestine of the adult horse, the activity is less than that in the immature horse (Dyer *et al*. 2002), thus large quantities of dietary lactose may cause digestive upsets and adult horses are relatively lactose intolerant. Healthy horses of all ages can absorb a glucose : galactose mixture without any change in the faeces. The relative intolerance is due to reduced lactose hydrolysis and does not normally involve the monosaccharide transport systems or malabsorption. If a suckling foal, or one given cow's milk, lacks an active form of the enzyme, it suffers from diarrhoea. An oral lactose tolerance test (1 g/kg BW as a 20% solution) may be of clinical value to determine small intestinal mucosal damage in diarrhoeic foals, when the continued ingestion of lactose might be detrimental. The deficient digestion or malabsorption of carbohydrate, whether primary or secondary, can almost always be localized to a defect in the enzymic, or transport, capacity of the small intestinal surface cell (see Chapter 11).

Lindemann *et al*. (1983) gave adult horses lactose or maize starch at 2 g/kg BW daily before a feed of wheat straw, or mixed with a diet of concentrate. Apparent precaecal digestibility of lactose was 38% and 71% in the straw and concentrate periods, respectively, while the digestibility of starch in the same periods was 88% and 93%, respectively. For straw about 1.2 g and for concentrate 0.6 g lactose per kg BW flowed into the caecum daily, leading to higher caecal VFA concentrations and a lower caecal pH with lactose than with starch in the straw period. Ileocaecal water flow reached 16.5 and 8.2 kg/kg feed DM with lactose in the straw and concentrate periods, respectively, compared with 15.2 and 7.0 kg/kg with starch. The 38% and 71% apparent precaecal digestibility of lactose is partly a reflection of microbial fermentation in the ileum. Faecal looseness with the feeding of lactose is therefore explicable.

Proteins

The amount of protein hydrolyzed in the small intestine is about three times that in the stomach. Proteins are in the form of long folded chains, linked by amino acid residues. For proteins to be digested and utilized by the horse these amino acids must usually be free, although the gut mucosal cells can absorb dipeptides. The enzymes responsible are amino- and carboxy-peptidases secreted by the wall of the small intestine.

Fats

The horse differs from the ruminant in that the composition of its body fat is influenced by the composition of dietary fat. This suggests that fats are digested and absorbed from the small intestine before they can be altered by the bacteria of the large intestine. The small intestine is the primary site for the absorption of dietary fat and long-chain fatty acids. Bile, continuously draining from the liver, facilitates this by promoting emulsification of fat, chiefly through the agency of bile salts. Emulsification increases the fat–water interface so that the enzyme lipase more readily hydrolyses neutral fats to fatty acids and glycerol, which are readily absorbed, although a proportion of dietary fat is absorbed into the lymphatic system as finely emulsified particles of neutral fat – triacylglycerols (TAGs) – and transported as lipoprotein in chylomicrons. The horse digests fat quite efficiently and the addition of edible fat to the diet has merit, particularly when endurance work is concerned (see Chapters 5 and 9).

Medium-chain TAGs (carbon chain length of 6–12) are readily absorbed as such by horses, followed by portal transport to the liver, where they are metabolized to ketones (Jackson *et al.* 2001).

Feed modification to improve digestion

The extent of precaecal breakdown of cereal starch from pelleted diets is in the sequence: oats > barley > maize (Meyer *et al*. 1995; de Fombelle *et al*. 2003a,b). Varloud *et al*. (2003) and de Fombelle *et al*. (2003b) found that, although much starch disappeared (but was not absorbed) in the stomach, the amount escaping precaecal digestion increased with starch intake: by 20% from barley and 30% from maize when horses received 281 g starch/100 kg BW in a meal. Thus, in order to increase digestibility and

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Table 1.3 Precaecal digestion of various sources of starch and digestion in the total GI tract of horses (Kienzle *et al.* 1992), (Rosenfeld & Austbø 2009)* and ponies (Potter *et al.* 1992a) (digested, g/kg intake).

	Starch intake, g/100 kg BW	Precaecal maize	Precaecal oats, barley*	Total oats	Total barley*	Precaecal sorghum	Total sorghum, maize*	Reference
Whole	200	289	835	—	—	—	—	Kienzle *et al.* 1992[1]
Rolled	200	299	852	—	—	—	—	Kienzle *et al.* 1992
Ground	200	706	980	—	—	—	—	Kienzle *et al.* 1992
""	—	663	949 705*	990	960*	—	910*	Rosenfeld & Austbø 2009
Crimped[2]	264^{CO} 295^{CS}	—	480	944	—	360	940	Potter *et al.* 1992a
Micronized	237^{MO} 283^{MS}	—	623	938	—	590	945	Potter *et al.* 1992a

[1]Maize and oat digestibilities measured by these workers refer to preileal measurements.

[2]Dry rolled with corrugated rollers to crack the kernels. ^{CO}, crimped oats; ^{CS}, crimped sorghum; ^{MO}, micronized oats; ^{MS}, micronized sorghum.

*Refer to barley and maize data in their respective columns.

avoid fermentation of starch in the equine large gut, commercial cooking of cereals is of economic interest. The processes used include infrared micronization of cereals and expansion or extrusion of products – the resulting starch gelatinization enhances small intestinal digestion at moderate, or high, rates of intake. The extent of cooking by the extrusion process varies considerably amongst the cookers used. Nevertheless, while small-intestinal digestibility is influenced, even in adult horses, *total* digestibility is not improved. The digestibility of raw and cooked cereals is similar when the comparison is between carbohydrate consumed and loss in faeces. Thus, the extent of precaecal and pre-ileal digestion influences the proportions of carbohydrate absorbed as glucose, VFAs, and lactic acid. Oat starch generally has greater small-intestinal digestibility than starch from either maize or barley (Kienzle *et al.* 1992; Meyer *et al.* 1993b; de Fombelle *et al.* 2004; Rosenfeld & Austbø 2009; Table 1.3).

The proportion of starch digested is influenced not only by cereal processing, but also by the amount fed. When starch intake per meal is only 2 g/kg BW the pre-ileal starch digestibility of ground oats is over 95%, whereas at the other extreme, that of whole or broken maize is less than 30%. The grinding of cereals increases pre-ileal digestibility compared with whole, rolled or cracked grain, although the keeping quality, or shelf-life, of ground grain is relatively short (Meyer *et al.* 1995). Workers in Hanover found that there is a much greater increase in the postprandial concentration of organic acids, including lactate, and in acidity, in jejunal chyme when oats are fed rather than maize. Whether this is related to the putative 'heating' effect of oats, compared with other cereals, has not been established. Lactate and other organic acid production is increased, and the pH is decreased in the ileum and caecum when undigested starch reaches those regions. In order to avoid starch 'overload', and therefore excessive starch fermentation, especially in the large intestine, starch intake in horses given two to three meals daily, should be limited to 4 g/kg BW per meal (Potter *et al.* 1992a; see also Chapter 11, Laminitis). This limit is too liberal where there is risk of laminitis. The Texas group (Gibbs *et al.* 1996) have found that when N intake is less than 125 mg/kg BW, 75–80% of the truly digestible protein of soya-bean meal is digested precaecally, 20% is digested in the large intestine, and 10% is indigestible. Physical state also influences protein digestibility. Extruded and micronized oats, barley and maize have a higher total tract protein digestibility than ground and pelleted forms, while pelleted and micron-

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Table 1.4 Effect of diet on the pH, production of VFAs and lactate and on microbial growth in the caecum and ventral colon of the horse 7 h after a meal.

Diet	pH	FA (mmol/L)				Total bacteria per ($mL \times 10^{-7}$)
		Acetate	Propionate	Butyrate	Lactate	
Hay	6.90	43	10	3	1	500
Concentrate plus minimal hay	6.25	54	15	5	21	800
Fasted	7.15	10	1	0.5	0.1	5

Note: Values given are typical, but all except the pH show large variations.

ized forms were shown to have the highest precaecal protein digestibility (Rosenfeld & Austbø 2009).

Nitrogen utilization

At high rates of protein intake more non-protein N (NPN) enters the GI tract in the form of urea. The N entering the caecum from the ileum is 25–40% NPN, varying with the feed type. Meyer (1983b) calculated that, in a 500 kg horse, 6–12 g urea N pass daily through the ileocaecal valve. The amount of N passing into the large intestine also varies with protein digestibility. At high intake rates of protein of low digestibility more N in total will flow into the large intestine, where it will be degraded to NH_3. From Meyer's evidence, about 10–20% of this total is urea N, as the daily range of total N flowing into the caecum is:

$$0.3–0.9 \text{ g N/kg BW}^{0.75}$$

N also enters the large intestine by local secretion, although the amount seems to be less than that entering through the ileocaecal valve and net absorption nearly always takes place. Nevertheless, net secretion can occur with low-protein, high-fibre diets.

Utilization of the derived NH_3 by gut bacteria is between 80% and 100%. Excessive protein intake must increase the burden of unusable N, either in the form of inorganic N, or as relatively unusable bacterial protein. This burden is influenced by feeding sequence. The provision of a concentrate feed 2 h after roughage, compared with simultaneous feeding, caused higher levels of free, particularly essential, amino acids in plasma 6–9 h later (Cabrera *et al.* 1992; Frape 1994). Plasma urea did not rise with dissociated, or separate, feeding, but rose continuously for 9 h after simultaneous feeding of roughage and concentrate. This indicates that mixed feeding led to a large flow of digesta N to the caecum, with much poorer dietary protein economy; interestingly, the separate feeding was in the reverse order to the standard practice of giving concentrates before roughage. More recently, Vervuert *et al.* (2009a,c) concluded that adding a fibre source to a cereal meal, or giving it before or after the cereal, prolonged precaecal digestion of starch, but did not influence precaecal starch digestibility or glucose and insulin responses.

THE LARGE INTESTINE

Grazing herbivores have a wide variety of mechanisms and anatomical arrangements for making use of the chemical energy locked up in the structural carbohydrates of plants. A characteristic of all grazing and browsing animals is the enlargement of some part of the GI tract to accommodate fermentation of digesta by microorganisms, producing steam-volatile FAs and lactate (Table 1.4).

More than half the dry weight of faeces is bacteria and the bacterial cells in the digestive tract of the horse number more than 10 times all the tissue cells in the body. No domestic mammal secretes enzymes capable of breaking down the complex molecules of cellulose, hemicellulose, pectin, fructo- and galacto oligosaccharides and lignin into their component parts, suitable for absorption, but, with the exception of lignin, intestinal bacteria do achieve this. The process is relatively slow in comparison with the digestion of starch and protein. This means that the flow of digesta has to be arrested for sufficient time to enable the process to reach a satisfactory conclusion from the point of view of the energy economy of the host animal. Physiochemical changes in the caecal contents over 24 h were reported by Tisserand *et al.* (1977a, b).

During the weaning and postweaning periods of the foal and yearling, the large intestine grows faster than the remainder of the alimentary canal to accommodate a more fibrous and bulky diet, hence energy digestibility of a mixed concentrate and forage diet increases at 5–8 months of age (Turcott *et al.* 2003).

At the distal end of the ileum, the large blind sack known as the caecum is about 1 m long in the adult horse and has a capacity of 25–35 L. At one end there are two

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muscular valves in relatively close proximity, one through which digesta enter from the ileum and the other through which it passes from the caecum to the right ventral colon. The right and left segments of the ventral colon and the left and right segments of the dorsal colon constitute the great colon, which is some 3–4 m long in the adult horse, having a capacity more than double that of the caecum. The four parts of the great colon are connected by bends known as flexures. In sequence, these are the sternal, pelvic and diaphragmatic flexures (Figure 1.2). Their significance probably lies in changes in function and microbial population from region to region and they probably act as foci of intestinal impactions.

Digestion in the caecum and ventral colon depends almost entirely on the activity of their constituent bacteria and ciliate protozoa. In contrast to the small intestine, the walls of the large intestine contain only mucus-secreting glands, that is, they provide no digestive enzymes. However, high levels of alkaline phosphatase activity, known to be associated with high digestive and absorptive action, are found in the large intestine of the horse, in contrast to the large intestinal environment in the cat, dog and man.

The diameter of the great colon varies considerably from region to region but reaches a maximum in the right dorsal colon where it forms a large sacculation with a diameter of up to 500 mm. This structure is succeeded by a funnel-shaped part below the left kidney where the bore narrows to 70–100 mm as the digesta enter the small colon. The latter continues dorsally in the abdominal cavity for 3 m before the rectum, which is some 300 mm long, terminates in the anus (Figure 1.2).

Contractions of the small and large intestine

The walls of the small and large intestine contain longitudinal and circular muscle fibres which are essential:

- for the contractions necessary to move the digesta, by the process of peristalsis, in the direction of the anus;
- for allowing thorough admixture with digestive juices; and
- for bathing the absorptive surfaces of the walls with the products of digestion.

During abdominal pain these movements stop so that the gases of fermentation accumulate.

Passage of digesta through the large intestine

Many digestive upsets are focused in the large intestine and therefore its function deserves discussion. The extent of intestinal contractions increases during feeding – large contractions of the caecum expel digesta into the ventral colon, but separate contractions expel gas, which is hurried through much of the colon. The reflux of digesta back into the caecum is largely prevented by the sigmoid configuration of the junction. Passage of digesta through the large intestine depends on gut motility, but is mainly a function of movement from one of the compartments to the next through a separating barrier. Considerable mixing occurs within each compartment, but there seems to be little retrograde flow between them. The barriers are:

- the ileocaecal valve already referred to;
- the caecoventral colonic valve;
- the ventrodorsal colonic flexure (pelvic flexure), which separates the ventral from the dorsal colon; and
- the dorsal small colonic junction at which the digesta enter the small colon.

Resistance to flow tends to increase in the same order, that is, the last of these barriers provides the greatest resistance (see Chapter 11). This resistance is much greater for large food particles than for small particles. In fact, the delay in passage for particles of 2 cm length can be more than a week. Normally the time taken for waste material to be voided after a meal is such that, in ponies receiving a grain diet, 10% is voided after 24 h, 50% after 36 h and 95% after 65 h. Mean retention time (MRT) in 18-month-old horses given a hay and concentrate diet was shown to be 42.7 and 33.8 h, respectively, for the solid and liquid phases of digesta (Chiara *et al*. 2003), while for a hay-based diet in mature heavy horses it was 21–40 h, decreasing within this range as intake increased (Miraglia *et al*. 2003). Within moderate variations of intake the digestibility of the diet was constant. A large decrease in MRT was associated with a lower digestibility coefficient.

Most digesta reach the caecum and ventral colon within 3 h of a meal, so that it is in the large intestine that unabsorbed material spends the greater proportion of time. The rate of passage in domestic ruminants is somewhat slower, and this partly explains their greater efficiency in digesting fibre. Nevertheless, the horse utilizes the energy of soluble carbohydrates more efficiently by absorbing a greater proportion of sugars in the small intestine.

In the horse, passage time is influenced by the physical form of the diet; for example, pelleted diets have a faster rate of passage than chopped or long hay, and fresh grass moves more rapidly than hay. Work at Edinburgh (Cuddeford *et al*. 1992) showed that fibre was digested more completely by the donkey than by the pony, which in turn digested it more effectively than the TB. These differences are probably due, in large measure, to the rela-

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tive sizes of the hind-gut and therefore, to the holding time of digesta. Donkeys working for 5 h daily with no access to food, subsequently ate as much poor quality hay and digested it as well as those not working and with continuous access (Nengomasha *et al*. 1999a). Holding time in the large gut seems to be uninfluenced by meal size, whereas rate of passage through the small intestine is greater with less frequent large meals. For mixed meals, the faecal excretion curves are similar for hay and oats, as mixing of the two components appears to occur in the caecum and right ventral and dorsal colons (Rosenfeld *et al.* 2006). Nevertheless, at normal intakes, most of the oat endosperm will have been digested in the small intestine.

Pattern of large intestinal contractions

The caecum contracts in a ring some 12–15 cm from the caecocolic junction, trapping ingesta in the caecal base and forcing some through the junction, which in the meantime has relaxed. With relaxation of the caecal muscles some reflux occurs, although there is a net movement of digesta into the ventral colon. The passage rate of digesta through the caecum is approximately 20%/h (Hintz 1990), compared with a typical rate for the rumen of 2–8%/h. However, disappearance rates of feed in monofilament polyester bags held in the pony caecum were greater during hay feeding than between meals (Hyslop *et al*. 1999). Feeding seems to cause an increase in the motility and volume of the caecum, allowing a more thorough mixing of its digesta with the bacteria.

Contractions of the colon are complex. There are bursts of contractile activity that propagate in an aboral (away from the mouth) direction, but some contractions propagate orally and some are isolated and do not propagate in either direction. Thus there is nonrhythmic haustral kneading as well as stronger rhythmic propulsive and retropulsive contractions. These contractions have the function of mixing the constituents, and promoting fermentation and absorption, as well as moving residues towards the rectum. The strong rhythmic contractions of the great colon begin at the pelvic flexure, where a variable site 'electrical pacemaker' exists. A major site of impactions is the left ventral colon, just orad (toward the mouth) to this pelvic flexure (Chapter 11). More detailed knowledge of this activity should ultimately help in the control of common causes of large gut malfunction and colic.

Microbial digestion (fermentation)

There are three main distinctions between microbial fermentation of feed and digestion brought about by the horse's own secretions:

(1) The β-1,4-linked polymers of cellulose (Figure 1.3) are degraded by intestinal microflora but not by the horse's own secretions. The cell walls of plants contain several carbohydrates (including hemicellulose) that form up to half the fibre of the cell walls of grasses and a quarter of those of clover. These carbohydrates are also digested by microorganisms, but the extent depends on the structure and degree of encrustation with lignin, which is indigestible to both gut bacteria and horse secretions (see 'Flora', this chapter).
(2) During their growth the microorganisms synthesize dietary indispensable (essential) amino acids.
(3) The bacteria are net producers of water-soluble vitamins of the B group and of vitamin K_2.

Microbial numbers

In the relatively small fundic region of the stomach, where the pH is about 5.4, there are normally 10^8–10^9 bacteria/g. The species present are those that can withstand moderate acidity, common types being lactobacilli, streptococci and *Veillonella gazogenes*. De Fombelle *et al*. (2003b) found that lactobacilli, streptococci and lactate-utilizing bacteria colonized the entire GI tract. The stomach and small intestine contain, per mL, the greatest number of these bacteria, so influencing the digestion of readily fermentable carbohydrates. De Fombelle (personal communication) also determined that the highest concentration of total anaerobic bacteria in the GI tract occurred in the stomach (see Gastric ulcers, Chapter 11). The jejunum and ileum support a flourishing population in which obligate anaerobic Gram-positive bacteria predominate (10^8–10^9/g). In this region of the small intestine a cereal diet can influence the proportion of the population producing lactic acid, compared with that producing VFAs as an end product, although the numbers of lactobacilli per gram of contents tend to be higher in the large intestine, where the pH is generally lower.

The flora of the caecum and colon are mainly bacteria which, in fed animals, number about 0.5×10^9 to 5×10^9/g contents. A characteristic difference between equine hind-gut fermentation and that in the rumen is the lower starch content of the hind-gut, which implies a generally lower rate of fermentation, yet the starch content of the caecum is variable, causing a variable suppression of cellulolytic and related bacteria. As the proportion of rolled barley to chopped meadow hay (given after the barley) was increased from nil to half, the digestibility of organic matter (OM) increased, whereas that of neutral detergent fibre (NDF) and acid detergent fibre (ADF) decreased, despite the slower flow rate of digesta with higher proportions of barley (C. Drogoul, personal communication).

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There is still a scarcity of knowledge concerning the activity of equine bacteria that digest the various entities of fibre. In one pony study (Moore & Dehority 1993), cellulolytic bacteria constituted 2–4% of the total. In addition, there were 2×10^2 to 25×10^2 fungal units/g, most of which were cellulolytic (see Probiotics, Chapter 5). In the horse, both caecal bacteria (which, with fungi, constitute the flora) and protozoa (fauna) participate in the decomposition of pectins and hemicellulose at an optimum pH of 5–6 (Bonhomme-Florentin 1988).

Fauna

Protozoa in the equine large intestine number about 10^{-4}, that is, 0.5×10^5 to 1.5×10^5/mL contents. Although protozoa are individually very much larger than bacteria and thus contribute a similar total mass to the large intestinal contents, their contribution to metabolism is less, as this is roughly proportional to the surface area. The species of fauna differ somewhat from those in the rumen. Some 72 species of protozoa, primarily ciliates, have been described as normal inhabitants of the equine large intestine, with some tendency to species differences between compartments. Moore & Dehority (1993) found in ponies that the protozoa were from the following genera: *Buetschlia*, *Cycloposthium*, *Blepharocorys* and a few *Paraisotricha*. Removal of the protozoa (defaunation) caused only a slight decrease in DM digestibility, with no effect on numbers of bacteria, or on cellulose digestibility.

Flora

In the large intestine the bacterial populations are highest in the caecum and ventral colon. Here, the concentration of cellulose-digesting bacteria is six to seven times higher than in the terminal colon. About 20% of the bacteria in the large intestine can degrade protein.

Numbers of specific microorganisms change by more than 100-fold over 24 h in domesticated horses being given, say, two discrete meals per day. These fluctuations reflect changes in the availability of nutrients (in particular, starch and protein) and consequentially changes in the pH of the medium. Thus, a change in the dietary ratio of cereal to hay will not only have large effects on the numbers of microorganisms, but will also considerably influence the species distribution in the hindgut. Although frequency of feeding has little impact on digestibility *per se*, it can have a large influence on the incidence of digestive disorders and metabolic upsets. Large concentrate meals lead to elevated glycaemic responses that can precipitate behavioural abnormalities, whereas fibrous feed lowers this response. Harris (2007) concluded that giving cereal meals once a day was unsuitable and that a high forage diet spread throughout the day was a safer choice. Recently Willing *et al.* (2009) showed that a forage diet, cf. a concentrate diet produced a more stable faecal microflora with lower counts of both lactic acid bacteria and members of the *Clostridiaceae*. Fibre stimulates peristalsis and is cationic, decreasing the risk of metabolic acidosis (Moore-Colyer 1998). Some of the consequences of digestive disorders and metabolic upsets result directly from the effects of diet and digesta upon the microbial populations (bacteria and protozoa).

Caecal bacteria from horses adapted to a grain diet are less efficient at digesting hay than are the microbes from hay-adapted horses. An analogous situation exists for hay-adapted caecal microbes when subjected to a grain substrate. If such a dietary change is made abruptly in the horse, impactions can occur in the first of these situations and colic, laminitis or puffy swollen legs can result in the second (see Chapter 11).

The caecal microorganisms in a pony or horse tend to be less efficient at digesting hay than the ruminal microbes in cows. The digestibilities of organic matter and crude fibre in horses fed a diet containing more than 15% crude fibre (a normal diet of concentrates and hay) are about 85% and 70–75%, respectively, of ruminant values. This has been attributed to the combined effects of a more rapid rate of passage of residues in horses and differences in cellulolytic microbial species. In fact, Hayes *et al*. (2003) concluded that a greater intestinal retention time in mares cf. foals of one month of age, accounted for their greater ability to digest fibre. The faecal population of microbes from the foals had a capacity similar to that of the mares to ferment fescue hay NDF. Differences also occur amongst regions of the GI tract in the time required for microbial enzyme adaptation to fibre fermentation. This influences the extent of fermentation in a limited time. Inocula from the stomach, duodenum and ileum expressed a lag time of 1–2 h, cf. 0.1–0.5 h for hind-gut inocula, in roughage fermentation, so limiting foregut fermentation (Moore-Colyer *et al*. 2003a). The degradation of ADF and crude protein of sugar beet pulp, hay cubes, soya hulls and a 2 : 1 mixture of oat hulls : naked oats was no poorer in the pony caecum than in the rumen of the steer over incubation periods of 12–48 h (Hyslop *et al*. 1997). During incubation for 12 h the degradation of beet pulp and hay was marginally greater in the caecum. Thus, the equine hind-gut microflora are not inherently less efficient than rumen microflora at feed degradation. Lower equine feed digestibility results largely from a more rapid rate of passage through the hind-gut than through the rumen.

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Estimation of fibre degradability

Moore-Colyer (1998) measured apparent digestibility and fibre degradation, as indicated by analysis of non-starch polysaccharides (NSPs) and NDF, of sugar beet pulp, soya hulls, hay cubes, and oat hulls : naked oats (2 : 1). NSP molecules are composed of several constituent monomers that are present in different proportions in various sources, and these monomers are normal components of cell walls. The principal monomers are: arabinose, galactose and uronic acids, glucose and xylose. The most *microbially* degradable monomers in the above four feeds were arabinose, galactose and uronic acids. sugar beet pulp had the highest concentrations of arabinose and uronic acids and was degraded at the fastest rate, whereas the rates for hay cubes and particularly for oat hulls were much slower. Hay cubes and oat hulls would have a lower apparent digestibility than sugar beet pulp, and soya hulls would be intermediate in value. NSP and NDF are simpler to measure, but are poorer guides to degradability than the monomer composition of the NSP of feeds. The subject of fibre analysis has been reviewed in several papers, notably by McCleary (2003). The presence of starch in the large intestine affects fibre degradability. The fermentation of ADF and NDF from a high starch diet in the hind gut was lower than for a high fibre diet (Varloud *et al.* 2004). This probably indicates that resistant starch (Englyst & Englyst 2005) had interfered with microbial action on fibre.

Analytical methods

Lattimer *et al.* (2007) compared data derived from an *in vitro* nylon bag/faecal inoculum technique with *in vivo* data to measure fermentability of feed NDF, ADF and OM, and concluded that their *in vitro* method produced valid data. (N.B. The inoculum must be appropriate to the diet tested!)

Products of fermentation

The microbial fermentation of dietary fibre, starch and protein yields large quantities of short-chain VFAs as by-products, principally acetic, propionic and butyric acids (Table 1.5, Figure 1.4). This fermentation and VFA absorption are promoted by:

- the buffering effect of bicarbonate and Na^+ derived from the ileum;
- an anaerobic environment; and
- normal motility to ensure adequate fermentation time and mixing.

Acetate and butyrate are major products of fibre digestion, whereas the proportion of propionate (and lactate, see Chapter 11) increases with increasing proportions of starch left undigested in the small intestine. In the pony, limited evidence indicates that 7% of total glucose production is derived from propionate produced in the caecum.

Table 1.5 Proportion of VFAs in digesta to body weight (BW) in four herbivores (Elsden *et al.* 1946).

	g VFA/kg BW
Ox	1.5
Sheep	1.5
Horse	1.0
Rabbit	0.5

VFA, fluid and electrolyte absorption in the large intestine

Without absorption, the VFAs produced during fermentation in the large intestine would soon pollute the medium, rapidly producing an environment unsuitable for continued microbial growth; however, an equable medium is maintained by the absorption of these acids into the bloodstream. In addition, there is vital absorption of large amounts of water and electrolytes (sodium, potassium, chloride and phosphate).

The largest proportion of water that moves through the ileocaecal junction is absorbed from the lumen of the caecum and the next largest from the ventral colon. Fluid is also absorbed from the contents of the small colon, to the benefit of the water economy of the horse and with the formation of faecal balls. This aboral decline in water absorption is accompanied by a parallel decrease in sodium absorption. In the pony, 96% of the sodium and chloride and 75% of the soluble potassium and phosphate entering the large bowel from the ileum are absorbed into the bloodstream. Although phosphate is efficiently absorbed from both the small and large gut, calcium and magnesium are absorbed mainly from the small intestine (Figure 1.5). This phenomenon has been proffered as a reason why excess dietary calcium does not depress phosphate absorption, but excess phosphate can depress calcium absorption, although not necessarily calcium balance, in the horse (see Chapter 3).

The water content of the small intestinal digesta amounts to some 87–93%, but the faeces of healthy horses contain only 58–62% water. The type of diet has a smaller effect on this than might be imagined. For instance, oats produce fairly dry faeces, but bran produces moist faeces, although bran contains only some 2 or 3 percentage units more moisture.

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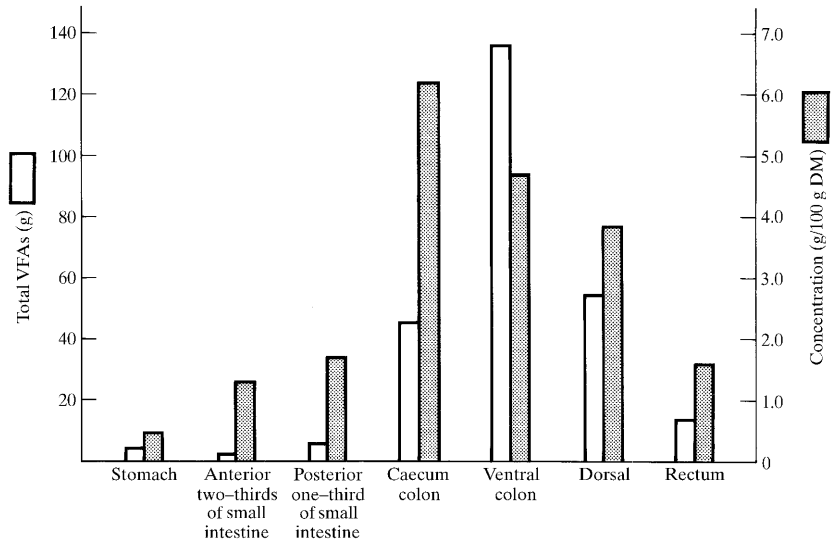


Figure 1.4 VFAs (□) calculated as the total weight (g) of acid (as acetic acid) in the organ or as the concentration (g/100 g DM) (⊠) in the lumen (after Elsden *et al.* 1946).

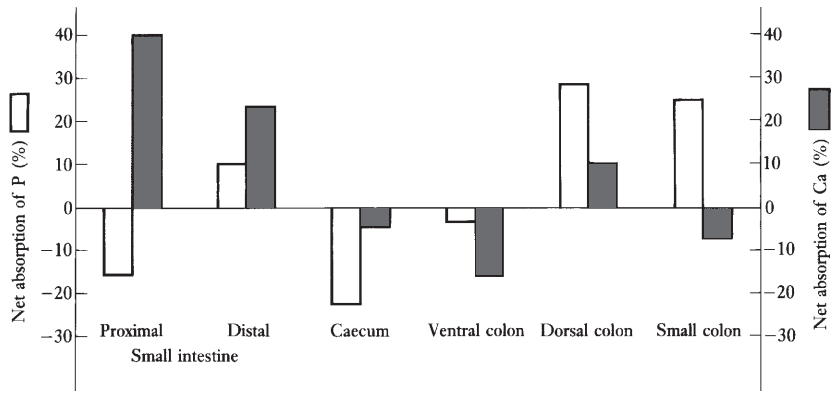


Figure 1.5 Net fractional absorption of P (□) and Ca (■) from various regions of the small and large intestine (after Schryver *et al.* 1974a).

Microbial degradation occurs at a far faster rate in the caecum and ventral colon than in the dorsal colon (Figure 1.4) and the rate is also faster when starches are degraded rather than structural carbohydrates. A change in the ratio of starch to fibre in the diet leads to a change in the proportions of the various acids yielded (Table 1.4) and in the organs in which the change occurs. Proportionately more propionate is produced with the consumption of starch, especially in the caecum and ventral colon affecting colic risk. Many bacteria have the capacity to degrade dietary protein, so yielding another blend of VFAs.

The optimum pH for microbial activity that promotes VFA absorption is 6.5. VFAs are absorbed in the unionized form. As the pH moves closer to the pK of a particular VFA, more is absorbed. The H^+ ions required for this are probably derived from mucosal cells in exchange for Na^+. HCO_3^- buffer is secreted into the lumen in exchange for Cl^-. Thus, absorption of VFAs is accompanied by a net

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absorption of NaCl. This in turn is a major determinant of water absorption. The ingestion of a large meal can cause a 15% reduction in plasma volume, ultimately resulting in renin–angiotensin, and then aldosterone, release. The increase in plasma aldosterone level causes increased Na^+ absorption (see Chapter 9). Whereas most ruminal butyrate is metabolized in the mucosa before entering the bloodstream, in horses all VFAs pass readily into the blood. Lactic acid produced in the stomach is apparently not well absorbed from the small intestine. On reaching the large intestine some is absorbed, along with that produced locally, but much is metabolized by bacteria to propionate.

Microbial activity inevitably produces gases – principally carbon dioxide, methane and small amounts of hydrogen – which are absorbed, ejected from the anus, or participate in further metabolism. The gases can, however, be a severe burden, with critical consequences when production rate exceeds that of disposal.

Protein degradation in the large intestine and amino acid absorption

Microbial growth, and therefore the breakdown of dietary fibre, also depends on a readily available source of nitrogen. This is supplied as dietary protein and as urea secreted into the lumen from the blood. Despite the proteolytic activity of microorganisms in the hind-gut, protein breakdown per litre is about 40-fold greater in the ileum than in the caecum or colon, through the activity of the horse's own digestive secretions in the small intestine.

The death and breakdown of microorganisms within the large intestine release proteins and amino acids. The extent to which nitrogen is absorbed from the large intestine in the form of amino acids and peptides useful to the host is slight. Isotope studies indicate that microbial amino acid synthesis within the hind-gut does not play a significant role in the host's amino acid economy. Quantitative estimates depend on the diet and the animal's requirements, but 1–12% of plasma amino acids are of hind-gut microbial origin. Absorption studies have shown that, while ammonia is readily absorbed by the proximal colon, significant basic amino acid absorption does not occur, and S-containing amino acid absorption occurs to only a small extent. Consequently, small-intestinal digestibility of protein is important, and this digestibility of sugar-beet pulp is poorer than that of hay cubes and much poorer than that of soya hulls (Moore-Colyer 1998). The latter, therefore, possess the highest amino acid value of the three.

Horses differ from ruminants in absorbing a higher proportion of dietary nitrogen in the form of the amino acids in dietary proteins, proportionately less being converted to microbial protein. As only a small proportion of the amino acids present in microbial protein is made available for direct utilization by the horse, young growing horses in particular respond to supplementation of poor-quality dietary protein with lysine and threonine, the principal limiting indispensable amino acids (Figure 1.6).

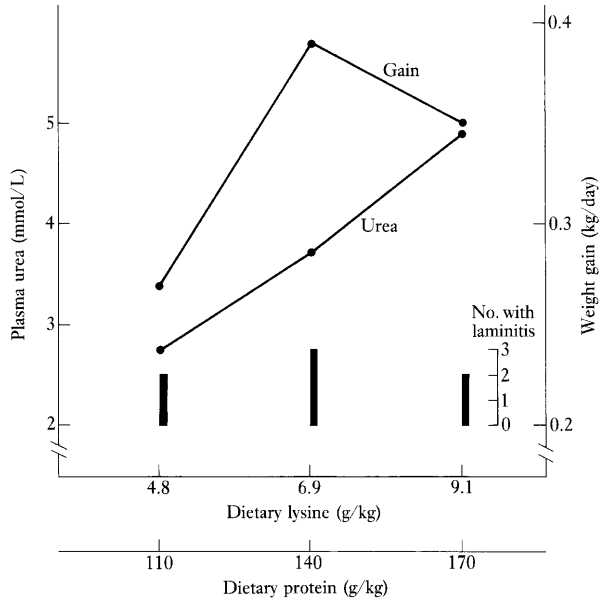


Figure 1.6 Response of 41 six-month-old ponies over a three-month period of diets containing different amounts of protein and lysine (initial weight 127 kg). In this experiment, increased protein intake led to elevated protein catabolism and urea production without an increase in incidence of laminitis (Yoakam *et al.* 1978).

Urea production

Urea is the principal end-product of protein catabolism in mammals and much of it is excreted through the kidneys. It is a highly soluble, relatively innocuous compound and a reasonably high proportion of the urea produced in the liver is secreted into the ileum and conveyed to the large intestine. Table 1.6 shows the flow of total N (12–24 mg/kg BW of which is urea N) from the ileum to the caecum; where most of it is degraded to ammonia by bacterial urease (which is not found in mammalian cells). Most of this ammonia is re-utilized by the bacteria in protein synthesis. Some, however, diffuses into the blood, where levels are normally kept very low by a healthy liver.

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Table 1.6 Effect of diet on the flow of nitrogen from the ileum to the caecum (Schmidt *et al.* 1982).

Diet	Nitrogen flow daily (mg N/kg BW)
Concentrate, 3.75 kg daily (1%)*	62
Concentrate, 7.5 kg daily (2%)*	113
Hay	68
Straw	37

*Weight of concentrate given as percentage of BW.

If ammonia production greatly exceeds the capacity of the bacteria, and liver, to utilize it, ammonia toxicity can arise. The fate of any urea added to the diet is similar.

In summary, digestion and fermentation in, and absorption from, the large intestine, account, in net terms, for 30% of dietary protein, 15–30% of dietary soluble carbohydrate and 75–85% of dietary structural carbohydrate. The salient causes of variation in values for each of the principal components of the horse's diet are:

- the degree of adaptation of the animal;
- the processing to which the feed is subjected; and
- the differences in digestibility among alternative feedstuffs.

For a discussion of commercial enzyme and microorganism products, see Chapter 5.

STUDY QUESTIONS

(1) What are the advantages and disadvantages of a digestive system with a major microbial fermentation site in the hind-gut only, compared to the fermentation system of the ruminant?
(2) The stomach of the horse is relatively smaller than that of the rat or human. What consequences do you draw from this?

FURTHER READING

Andrews, F.M., Buchanan, B.R., Elliott, S.B., Al Jassim, R.A.M., McGowan, C.M. & Saxton, A.M. (2008) *In vitro* effects of hydrochloric and lactic acids on bioelectric properties of equine gastric squamous mucosa. *Equine Veterinary Journal*, **40**, 301–5.

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2
Utilization of the Products of Dietary Energy and Protein

A horse whose work consists of travelling a stage of twenty miles three times a week, or twelve every day, should have one peck of good oats, and never more than eight pounds of good hay in twenty-four hours. The hay, as well as the corn, should, if possible, be divided into four portions.

J. White 1823

CARBOHYDRATE, FAT AND PROTEIN AS SOURCES OF ENERGY, AND THE HORMONAL REGULATION OF ENERGY

Glucose, VFA and TAG clearance

Horse diets rarely contain more than 5% fat and 7–12% protein so that these represent relatively minor sources of energy in comparison to carbohydrate, which constitutes two-thirds of the diet by weight. Although protein is required primarily in the building and replacement of tissues and is an expensive source of energy, both dietary protein and fat can contribute to the substrates used by the horse to meet its energy demands for work. Protein does so by the conversion of the carbon chain of amino acids to intermediary acids and of some of the carbon chains to glucose. Neutral fat does so following its hydrolysis to glycerol and fatty acids. The glycerol can be converted to glucose and the fatty acid chain broken down by a stepwise process called β-oxidation, which occurs in the mitochondria, yielding ATP and acetate, or, more strictly, acetyl coenzyme A (acetyl-CoA), and requiring tissue oxygen (see Figure 9.2).

Carbohydrate digestion and fermentation yield predominantly glucose and acetic, propionic and butyric VFAs. These nutrients are collected by the portal venous system draining the intestine and are partly removed from the blood as it passes through the liver. Both glucose and propionate contribute to liver starch (glycogen) reserves, while acetate and butyrate bolster the fat pool (Figure 2.1) and also constitute primary energy sources for many tissues.

Sequence of feeding and amount fed

Studies in France and Germany have shown that the sequence in which feeds are given can influence the metabolic outcome. When concentrate was given to ponies two hours after roughage, plasma urea concentrations were significantly lower, and plasma free amino acids higher, in the postprandial period, than when the concentrates and roughage were given simultaneously (Cabrera *et al.* 1992). This indicates that the conventional procedure of feeding concentrates with, or before, hay is likely to depress the potential net protein value of the diet. Concentrates appear to be better utilized if given after roughage consumption, probably as a result of their slower passage through the small intestine.

Larger feeds are passed more quickly through the GI tract, e.g., in horses commencing periods of greater work. Rate of passage is the prime factor influencing the extent to which fibrous materials are fermented by the bacteria in grazing animals. This process is relatively slow and hence the larger the volume of the fermentation 'vat', the more complete the fermentation process. The ratio of the volume of the 'vat' to body weight in most ponies seems to be less than it is in donkeys. As increased rates of passage slightly reduce roughage digestibility which accounts for somewhat poorer fibre utilization in ponies than in donkeys (Pearson & Merritt 1991). van Weyenberg *et al.* (2007) gave a complete ration either once (at 08:00 h) or three times (at 08:00, 13:00 and 18:00 h) daily without any apparent effect on digestibility or on plasma metabolites; potential effects were masked by the sparing of feed for ingestion throughout the day in the once-fed group.

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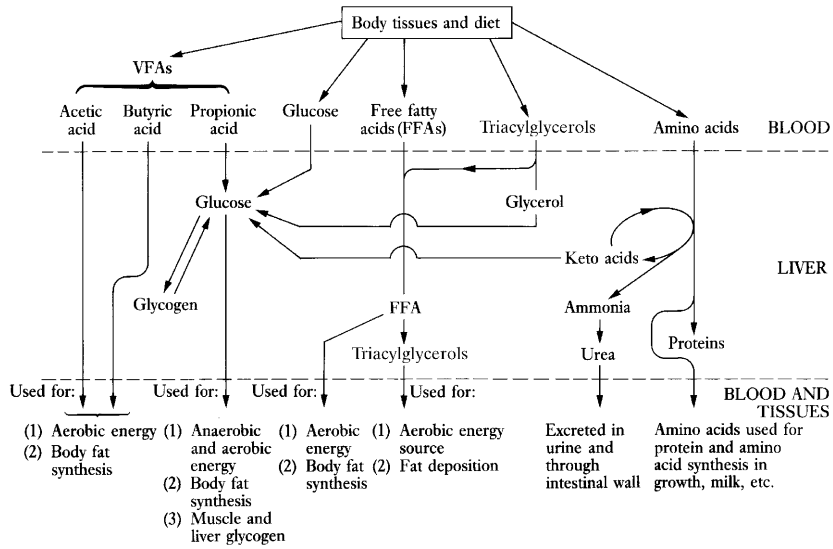


Figure 2.1 Sources, metabolism and fate of major energy-yielding nutrients derived from body tissues and diet (after McDonald *et al.* 1981).

Timing of feeds and appetite

Feeding in the horse or pony causes mesenteric hyperaemia, that is, a diversion of blood to, and engorgement of, the blood vessels serving the GI tract. In a similar manner, exercise causes increased blood flow to muscles. Exercise within a few hours of feeding therefore increases the demands on the heart to supply blood for both activities. Even moderately strenuous exercise (75% of heart rate maximum) under these conditions leads to increased heart rate, cardiac output, stroke volume and arterial pressure, in comparison to the effects of exercise on fasted animals (Duren *et al.* 1992). The optimum timing of meals in relation to exercise is discussed in Chapter 9. (See also 'Appetite', this chapter.)

Blood glucose and hormonal responses to feeding

In healthy horses and ponies, blood plasma glucose concentrations are maintained within defined limits. This is necessary as glucose is the preferred source of energy for most tissues. In ponies, normal healthy resting levels range between 2.8 and 3.3 mmol/L, but horse breeds generally have higher resting levels, with TBs in the region of 4.4–4.7 mmol/L. The concentration in horses rises dramatically from the commencement of a meal to ≥6.5 mmol/L after 2 h (Figure 2.2). A return to fasting concentrations is much slower than in the human and slower still in ponies, while the scale of the plasma glucose response to a meal is greatly influenced by the intensity of any previous exercise, with intense exercise greatly diminishing the response (Frape 1989). (See also Laminitis in Chapter 11.)

Insulin

If food intake is restricted for 12 h before giving a test starchy diet with no further roughage intake during blood sampling (Vervuert *et al.* 2009e), plasma glucose and insulin responses can be measured as the areas under the (response) curves (AUCs). Feed sources of carbohydrate are compared to an equivalent amount of glucose administered by nasogastric tube (Jose-Cunilleras *et al.* 2004), or to the feeding of oats or maize (Rodiek & Stull 2005; Vervuert *et al.* 2004, 2005a). Jose-Cunilleras *et al.* (2004) found that the glycaemic indices of oat groats, maize and barley were similar, with a value of approximately 60 compared with glucose. The glycaemic indices for common feeds measured in Quarter Horse mares ranged from 7 to 129 based on a value of 100 for oats (Rodiek & Stull 2007). Sweet feed, corn, jockey oats, and oats had the highest values, and beet pulp, alfalfa, rice bran, and soy hulls had the lowest. Intermediate glycaemic indices (55–69) were found for barley and wheat bran. It is apparent that including structural carbohydrate in isoenergetic feeds lowers the glycaemic index and insulin response slightly

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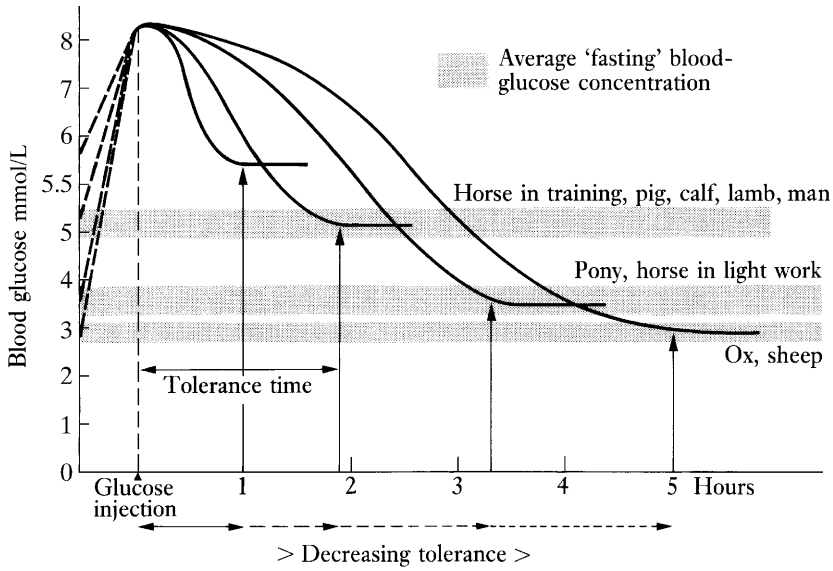


Figure 2.2 Approximate glucose tolerance times (arrows) and normal 'fasting' blood-glucose concentration (shaded). Glucose is injected i.v. to allow comparisons between species with different digestive anatomy and mechanisms. By providing glucose in the form of a starch meal, the peak is delayed 2–4 h in the horse. When oats are given as feed the maximum blood-glucose concentration in TBs occurs about 2 h following the start of feeding. The determination of 'tolerance time' has been largely superseded by the determination of the AUC following a glucose dose, as this is determined with greater precision. Generally, the greater the AUC, the longer the tolerance time in an individual horse.

(Gordon *et al.* 2007), whereas adding structural carbohydrate to an existing starchy feed might not (Vervuert *et al.* 2009a). By comparison, increasing the NSC content and feeding at a lower rate can lower the insulin response considerably (Gordon *et al.* 2007). Vervuert *et al.* (2009c) observed that adding alfalfa chaff to a meal of oats prolonged the precaecal digestion of starch (and presumably eating time), without affecting precaecal starch digestibility. In summary, insulin is an anabolic hormone, the function of which is primarily to switch on enzymes directed towards storage of blood glucose and fat. The effects of dietary fibre depend on the physical characteristics of the natural fibre source, but its addition to a starchy feed can extend the period over which small intestinal digestion occurs, lowering peak glucose and insulin responses without substantially altering the total glucose absorption, i.e. the AUC. A more pronounced reduction in insulin response is achieved by giving smaller quantities of cereals in each of more daily meals. Therefore, large rations of concentrates should be divided amongst several daily meals. The more rapidly the plasma glucose is cleared, that is, the greater the tolerance, the smaller the AUC (Figures 2.2 and 2.3). This clearance from the blood results from uptake, particularly into liver and muscle cells, where it is converted to glycogen and also to fat, although the net conversion to fat in a fit athletic animal is small. Fat clearance occurs through activation of adipose tissue lipoprotein lipase (LPL). (The oral glucose tolerance test suffers from the influence of factors other than insulin, for instance, impaired gut function; intravenous loading therefore measures insulin sensitivity more reliably; see Chapter 12.) Circulating glucose is used directly to meet immediate energy demands. Concomitant with the rise in plasma glucose concentration, plasma NEFA level decreases, owing to a reduction in the mobilization of fat caused by insulin repressing the activity of intracellular, hormone-sensitive lipase in adipocytes (see also Chapter 9). Plasma TAG concentrations in ponies subjected to an extended fast are much higher than those in horses. This results from raised plasma NEFA that are converted by the liver to TAG, where this is mobilized and transported as very low density lipoproteins (VLDLs). Raised plasma TAG

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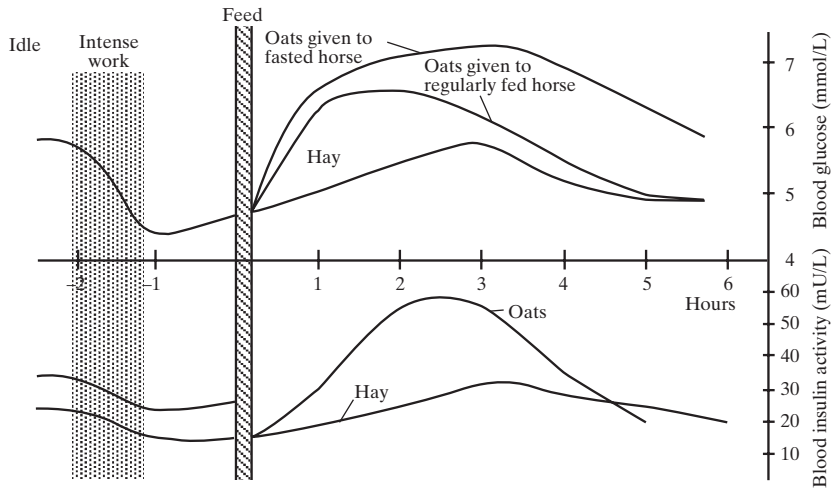


Figure 2.3 Responses of blood glucose and insulin to feed.

promotes insulin resistance, so it is not coincidental that hyperlipidaemia occurs more frequently in ponies than in horses. In humans, raised postprandial plasma fat causes a rise in plasma nonesterified fatty acids (NEFAs), which in turn promotes insulin resistance (Frape *et al.* 1997a,b). Generally, plasma glucose and insulin concentrations are lower and plasma NEFA and urea concentrations higher in horses on restricted rations (Ralston 1992). Sticker *et al.* (1996) found that these plasma changes occurred promptly in light horses, whereas plasma glucagon (see Glossary) responds with a slightly slower rate of decrease and thyroxine with a much slower increase in concentration upon the imposition of the restriction.

In order to avoid hypoglycaemia the effects of insulin are counterbalanced by those of other hormones (glucagon, the glucocorticoids, and the catecholamines epinephrine and norepinephrine). The system is thereby maintained in a state of dynamic equilibrium. The anabolic nature of insulin conflicts with the catabolic requirements of exercise, and although catecholamines, secreted during intense exercise, suppress further insulin secretion, elevated circulating postprandial insulin is undesirable if exercise is initiated. Both from this viewpoint, and from that of blood redistribution, exercise is to be discouraged during the postprandial period. Hypoglycaemic seizures have been reported in horses, during which plasma glucose can fall to less than 2 mmol/L; the cause is adenoma of pancreatic islet cell origin with hyperplasia, predominately of β-cells, leading to hyperinsulinaemia.

Blood insulin peaks shortly after blood glucose and concentrations reach 4–8-fold fasting values one to two hours following a feed. Blood insulin remains above fasting concentrations throughout the day in TBs after early morning training, again unlike the human response to a meal (Frape 1989). Horses and ponies have a lower glucose tolerance than man or pigs, but a slightly greater one than ruminants. TB and other hot-blooded horses generally have a higher tolerance than ponies; i.e. the tissues of ponies, particularly Shetland ponies, are less sensitive to insulin, although there is considerable adaptation to diet. This explains the lesser decrease in plasma NEFA concentrations in ponies following a glucose load. Fasted animals are more intolerant than fed, but ponies can withstand fasting better than TBs, which become more excitable after feeding. Adult ponies given a pelleted high-fibre diet are more glucose tolerant than either foals, or adults given long hay (Murphy *et al.* 1997). The substitution of a high-grain diet by a high fat–high fibre diet decreases the glycaemic and insulinaemic responses to a meal (Williams *et al.* 2001b), but for this reason it also decreases tissue sensitivity (Hoffman *et al.* 2003b), so that both the glycaemic and insulin responses to soluble carbohydrates are increased. Vervuert *et al.* (2009d) suggested that insulin responses may be more appropriate than glycaemic responses in defining the effect of feeding different amounts of starch to horses. A starch intake of <1.1 g/kg BW/meal produced only moderate glucose and insulin responses, so intake should be restricted to that amount (insulin resistance in the adult horse is not caused by

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glucose-yielding carbohydrates at 'normal' daily rates of intake, Author); but insulin sensitivity is lower in weanlings on a diet rich in starch and sugar than in those on a diet based on fibre and fat (Treiber *et al.* 2005a).

Leptin, ghrelin, and adiponectin

Insulin-dependent diabetes is very rare in horses. Whether or not the non-insulin-dependent form might occasionally involve a deficiency of dietary trivalent chromium in horses is unknown (see Chapter 3). Insulin resistance is associated with an inadequate response of tissue receptors to the hormone; a higher than normal local concentration of the hormone is required to elicit a normal tissue response. The postprandial rise in plasma insulin, not the **cortisol rhythm**, drives the associated increase in plasma leptin concentration (Cartmill *et al.* 2005), although according to Gordon & McKeever (2005) a response of both plasma leptin and ghrelin occurs following the afternoon, but not the morning feed, when the insulin response to feeding is much greater (as in the human subject; Frape *et al.* 1997b). Plasma levels are higher in fed than in fasted individuals and in fed animals plasma concentrations are higher from 12.00 to 24.00 than from 24.00 to 12.00 (Buff *et al.* 2001). The direction of these responses depends on the extent of the insulin excursion as Gordon & McKeever (2006) reported an initial decrease in plasma ghrelin before it increased and decreases in plasma leptin and cortisol following a large increase in plasma insulin, whereas when plasma insulin rise was half this, leptin rose. A large plasma insulin response is associated with a low insulin sensitivity (SI) and obesity in the horse. Poor SI and obesity are also associated with elevation of the plasma inflammatory cytokines interleukin 1 (IL-1) and tumour necrosis factor-α (TNFα), although an association with TNFα was only noted in a horse >20 years old, according to Vick *et al.* (2007).

Quinn *et al.* (2008) measured SI in mature TB geldings with a similar BCS (4.3) and SI values given either a high starch and sugar diet, or one high in fat and fibre. Following dietary acclimatization, SI was decreased in horses on the high starch diet compared with those on the high fat one. However, after 32 weeks of controlled weight gain (90.8 kg; final BCS 7.0) their SIs did not differ significantly from the starting values. Exercise was then restricted for 2 weeks, resulting in a reduction in SI in horses on the high fat diet, but no change in those on the high starch diet. This suggests that dietary energy source could have more influence than weight gain on SI in the mature TB gelding between BCS 4 and 7, while physical activity seems to be required for a better SI in horses on a high fat diet**.** Factors affecting SI have been reviewed by Firshman & Valberg (2007). The principal dietary and metabolic involvement in the causation of resistance is excessive feed energy, obesity, aging and inadequate exercise.

Leptin is a satiety-inducing hormone, its serum concentration is greater in horses with higher condition scores (Steelman *et al.* 2006). After exercise, leptin and TAG concentrations were positively correlated (Kedzierski & Kapica 2008). Both mean and peak plasma glucose concentrations were greatest in horses fed twice daily than those fed the same total amount in three or four divided feeds. This response was associated with an increase from baseline in serum leptin concentration only in those fed twice daily, indicating that a departure from baseline of serum leptin has the potential to affect appetite (Steelman *et al.* 2006). Plasma leptin concentration increased initially during overfeeding of Shetland ponies, but returned to previous values after 7 day and decreased during underfeeding. Underfeeding was associated with a higher AUC for plasma glucose and plasma insulin, and decreasing glucose tolerance (Van Weyenberg *et al.* 2008), due probably to decreased feed intake increasing plasma cortisol. The authors concluded that changes in energy balance, without altering bodyweight, can influence plasma leptin concentrations in ponies. Plasma leptin and ghrelin concentrations increased after exercise to a greater extent in horses of 1.5 years old than 2.5 years of age and the leptin concentration was higher in fillies than in colts (Kedzierski & Kapica 2008).

Plasma concentrations of leptin, adiponectin or ghrelin are associated with appetite. Leptin levels are lower and ghrelin higher, post-exercise. Ghrelin is generally lower after meals (Gordon *et al.* 2007b). Plasma leptin tends to have a lower concentration in fit than in unfit horses, and to be positively correlated with BCS, whereas ghrelin and adiponectin are at a higher concentration in fit horses and adiponectin is negatively correlated with BCS (Gordon *et al.* 2007a).

Equine Cushing's disease

Pathologically elevated fasting plasma glucose concentrations occur in hyperadrenocorticism (hypersecretion of cortisol), or Cushing's syndrome. This is associated with luxus gluconeogenesis, severe muscle wasting and protein breakdown. The resulting excess production of glucose in the horse is due most frequently to the presence of a pituitary adenoma, although primary adrenocortical neoplasms have been reported (van der Kolk *et al.* 2001). It is a chronic progressive disease with hypertrophy of the *pars intermedia* of the pituitary gland in older horses,

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resulting in excess production of releasing hormones, including ACTH. The insidious clinical sign is hirsutism in horses aged >15 years (Pilsworth & Knottenbelt 2006). Hyperglycaemia and hyperinsulinaemia are common findings and it is therefore prudent to provide energy from dietary fibre and edible oil that do not contribute to blood glucose (see also 'Amino acids', this chapter).

ENERGY METABOLISM

Hard muscular work requires that energy is available for muscular contraction at a rate some 40 times that needed for normal resting activity. Thus, rapid changes in the supply of blood glucose may result unless the animal's system responds quickly. Many changes are made to accommodate these altered circumstances, but our discussion at this point will relate to the supply of nutrients to the tissues.

During a gallop, pulmonary ventilation increases rapidly to ensure that more oxygen (O_2) is available for transport by the blood to the skeletal and cardiac muscles for the oxidative release of energy. However, this process cannot keep pace with the demand for energy, and glucose is therefore broken down to lactic acid, rapidly releasing energy in the absence of O_2. The fall in blood glucose stimulates the glucocorticoids and the other hormones that enhance glycogen breakdown so that blood glucose can rise during moderate exercise.

Repeated hard work (see also Training, Chapter 9) brings about several useful physiological adaptations to meet the energy demands of muscular work. First, the pulmonary volume, and therefore the tidal volume, of O_2 increases and the diffusion capacity for gases increases, so that carbon dioxide (CO_2) is disposed of more efficiently from the blood and O_2 is absorbed at a faster rate. This process is greatly assisted by changes in both numbers of red cells (erythrocytes) and the amount of haemoglobin in the blood. There is therefore a greater capacity for the oxidation of lactic acid and fatty acids to CO_2. However, training is also associated with a decrease in insulin secretion, possibly a higher glucocorticoid secretion, larger amounts of muscle glycogen and blood glucose and, because of the greater work capacity, higher concentrations of blood lactate. The glucocorticoids, and possibly epinephrine in the trained (fit) animal, stimulate more efficient breakdown (lipolysis) and oxidation of body fat as a source of energy, so conserving glycogen and yielding higher concentrations of NEFAs in the blood. Kędzierski & Kowalik (2009) working with exercising trotters, concluded that the lipolysis rate in geldings is higher than in stallions or mares. The glycerol released during fat breakdown tends to accumulate during hard exercise, possibly because of the raised concentration of blood lactate, and only on completion of hard muscular work is it utilized for the regeneration of glucose (Figure 2.1).

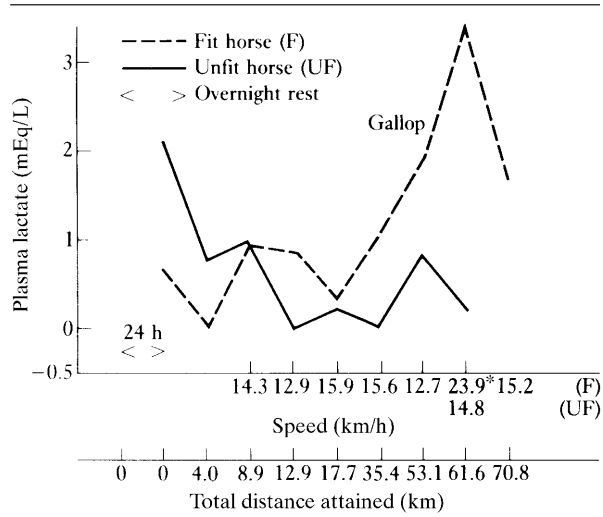


Figure 2.4 Effect of speed and distance achieved on the concentration of plasma lactate in two horses. The horses separated at 53.1 km and only the fit horse galloped between 53.1 and 61.6 km from start. The unfit horse retired after 61.6 km (Frape *et al.* 1979).

The energy requirements of extended work can be accommodated entirely by the aerobic breakdown of glucose and by the oxidation of body fat. Thus, no continued accumulation of lactate was observed in two horses subjected to an endurance ride (Figure 2.4), and although body fat represents the primary source of energy, its relatively slower breakdown means that there is a gradual exhaustion of muscle and liver glycogen associated with a continuous decline in blood glucose (Figure 2.5), despite elevated concentrations of NEFAs in the blood. Exhaustion occurs when blood glucose reaches its lower tolerable limit.

In a more general sense, hypoglycaemia contributes to a decrease in exercise tolerance. Therefore, horses and ponies conditioned to gluconeogenesis through adaptation and training (Figure 2.1) withstand extended work more readily. Hypoglycaemia may occur when extra-hard exercise coincides with a peak in insulin secretion, suggesting that horses and ponies conditioned to gluconeogenesis through high-roughage diets, cope more readily with sustained anorexia (persistent lack of appetite, usually through

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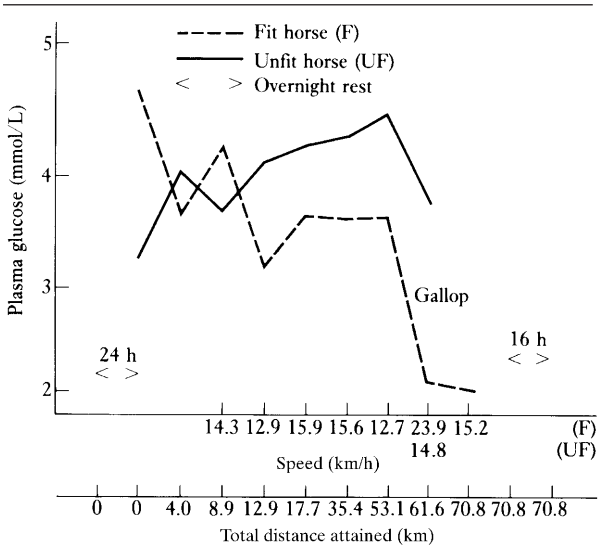


Figure 2.5 Effect of speed and distance achieved on the concentration of plasma glucose in two horses (Frape *et al.* 1979).

feed scarcity). Glucose represents a much larger energy substrate in individuals given a high-grain diet, whereas VFAs will do so in those subsisting on roughage. Horses and ponies accustomed to a diet rich in cereals will have, in a rhythmical fashion, greater peaks and lower troughs of blood glucose than those individuals maintained on a roughage diet, owing to differences in insulin secretion and rates of consumption of the two types of diet. The grain-fed horse at peak blood glucose is more spirited, and less so in the trough. The practical corollary of this is that individual horses and ponies accustomed to a diet rich in concentrates should be fed regularly and frequently in relatively small quantities, not only to prevent the occurrence of colic, but also to smooth out the cyclic changes in blood glucose (preparation for exercise is a different matter and is discussed in Chapter 9). The energy transfers of the young adult working horse are summarized in Figure 2.6.

Appetite

The endocrine system plays a prominent role in controlling appetite, but what triggers that system? It seems that leptin

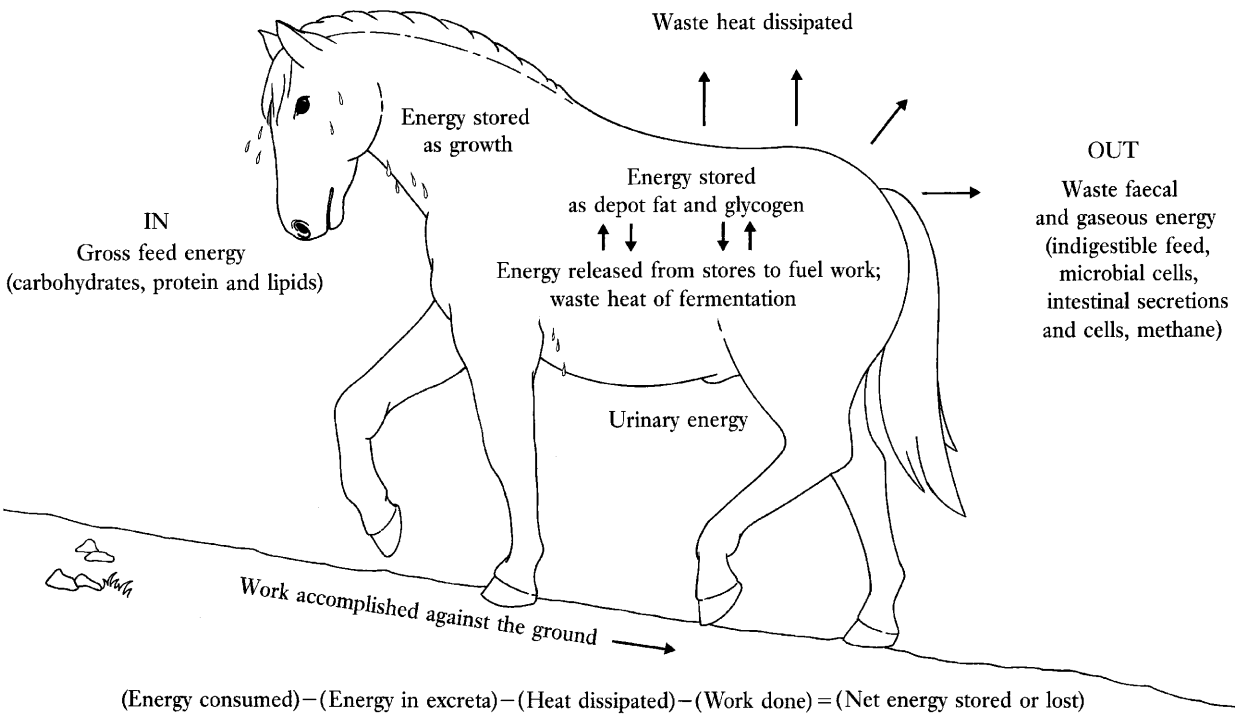


Figure 2.6 Energy transfers in the young adult working horse.

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may induce satiety in the presence of elevated plasma insulin where the sensitivity of tissues to both insulin and leptin is adequate. Satiety is not directly associated with an elevation in blood glucose, although individuals with low concentrations of blood glucose tend to eat more and faster. Blood-glucose concentration in ponies influences the interval between voluntary feeds, without affecting the amount consumed. Supplementary corn oil seems to extend the interval before the next meal and reduce total feed intake 3–18 h after administration. The degree of fill in the stomach and the blood-glucose concentration, have an indirect influence on eating. Taste, visual contact between horses, energy density of feed, rate of eating and time of day all seem to influence feed intake. The practical interpretations of this are considerable and are discussed in Chapter 6.

DIETARY PROTEIN

When alternative feeds are available the digestibility of dietary protein is an important attribute to consider. For example, leather is a rich source of protein, but valueless to the horse because of its low digestibility. Most proteins have an apparent digestibility coefficient of 0.6–0.8, but this indicates only the amount of N digested. Protein that reaches the large intestine does not provide absorbable amino acids in significant amounts. Precaecal apparent digestibility of most amino acids is 0.3–0.6 (Almeida *et al*. 1999; see also Chapter 5), implying that small intestinal digestibility of amino acids is the critical criterion for comparing dietary sources. A feature related to digestibility is availability of amino acids, particularly lysine. In practice, a reduction in lysine availability is encountered when skimmed milk, fish-meal and meat meals are overheated during processing, leading to a reaction between lysine and both unsaturated fats and reducing sugar, e.g. lactose. These reactions lead to products from which the animal's digestive system cannot recover the lysine. In summary, the protein value of a diet is the product of the amount, quality and digestibility of its constituent amino acids in the small intestine, but most dietary proteins have total tract digestibilities that fall within a narrow range, so that protein requirements are frequently given as total dietary protein.

PROTEIN REQUIREMENTS FOR MAINTENANCE

Tissue proteins are broken down into amino acids and resynthesized during the normal maintenance of adult or growing animals. This process is not fully efficient and, together with losses of protein in the sloughing of epithelial tissues and in various secretions, there is a continual need for dietary protein to make good the loss. However, these losses are relatively small in comparison with the protein synthesized during normal growth, or milk production, and proportionately less lysine is required. It follows that less protein, or protein of poorer quality, is needed for maintenance than is necessary for growth or milk secretion. Nevertheless, the protein needs of the adult horse for maintenance are less when good-quality protein is provided than when poor-quality protein is given. For example, adult TB mares were shown to remain in nitrogen balance when given 97 g fish protein/day, but they required 112 g when the protein source was maize gluten.

The protein needed by the horse for body maintenance can be defined as the amount of protein required by an individual making no net gain or loss in body nitrogen and excluding any protein that is secreted in milk. In these circumstances the animal must replace shed epithelial cells and hair, it must provide for various secretions and keep all cellular tissues in a state of dynamic equilibrium. The losses are a function of the lean mass of body tissues, depicted as a direct proportion of metabolic body size. For most purposes, the latter is considered to be BW raised to the power 0.75. However, the NRC (2007) adopts a broken straight line approach, relating the requirement to BW (in kg) and draws a distinction between resting horses and working horses with elevated needs, to calculate daily requirements:

Minimum work: BW (kg) × 1.08 g CP
Average work: BW (kg) × 1.26 g CP
Hard work: BW (kg) × 1.44 g CP

These amounts assume that the average requirement is met by CP containing 4.3% lysine, as the average lysine requirement for maintenance is: BW (kg) × 0.054 g (see Tables 2.2 and 6.6 for the NRC estimates of energy and protein requirements). This is discussed in more detail in Chapter 6.

Muhonen *et al.* (2008) abruptly changed the CP content of grass silage-only diets of adult geldings, given at the maintenance level of energy and similar DM intakes, from 873 g to 615 g CP/day. During the first 24 h after a change in either direction, the concentrations of total anaerobic bacteria and lactobacilli in the colon were greater for the higher than for the lower protein (7.1 *vs* 6.7 $\log_{10}$ cfu/mL and 6.0 *vs* 5.5 $\log_{10}$ cfu/mL, respectively), but VFA concentrations did not differ. From day 7 to 22 over both periods, total VFA concentration was greater on the higher protein diet than on the lower (51.8 *vs* 45.1 mmol/L), and

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colon pH was lower on the higher protein diet than on the lower (6.9 *vs* 7.2). The results suggest that in horses fed in the upper range of the NRC (2007) maintenance needs, a CP change between silages can alter the colon bacterial counts within the first 24 h. Muhonen *et al.* (2009) later found higher colon counts of lactobacilli and streptococci and lower colon and faecal DM contents in horses receiving a silage diet than a haylage diet, but concluded that an abrupt feed change from grass hay to grass silage or haylage does not induce any major alterations in the colonic ecosystem.

AMINO ACIDS

Proteins consist of long chains of amino acids, each link constituting one amino acid residue. In all natural proteins that have been examined, there are about 20 different kinds of amino acid. The horse does not have the metabolic capacity to synthesize the amino group contained in 10 or 11 of them sufficiently fast, or at all, to meet its requirements for protein in tissue growth, milk secretion, maintenance, etc., although it can produce the remainder by transferring the amino group from one carbon skeleton to another in a process known as transamination. Plants and many microorganisms can synthesize all 25 amino acids. Thus, the horse and other animals must have plant material in their diet, or animal products originally derived from plant food, in order to meet all their needs for amino acids. A small proportion of the amino acids derived from proteins synthesized by microorganisms, chiefly in the horse's large intestine, can be utilized directly by the horse. Microbial proteins might make some contribution, in the ileum, but by far the major part is voided as intact bacterial protein in the faeces.

During the digestion of dietary protein, the constituent amino acids are released and absorbed into the portal blood system. The amount of protein consumed by the horse may be in excess of immediate requirements and, although there is some capacity for storage in the form of blood albumin, most excess amino acids, or those provided in excess of the energy available to utilize them in protein synthesis, are deaminated in the liver with the formation of urea. The concentration of urea rises in the horse's blood (Figure 1.6), although some of the amino-nitrogen can be utilized in the liver for the synthesis of dispensable amino acids (Figure 2.7). An increase in the blood concentration of urea in endurance horses simply reflects rapid

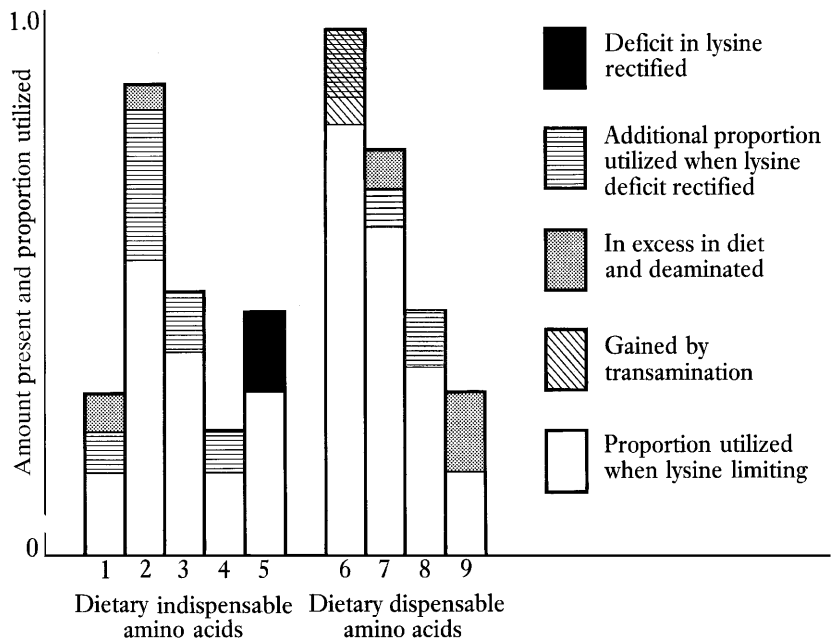


Figure 2.7 Effect of lysine supplementation on the utilization of a dietary protein that is grossly limiting in its lysine content (5). Only nine of a possible 25 amino acids are shown. Amino acids 3 and 4 limit further utilization of a lysine-supplemented diet. Addition of supplementary 3 and 4 would decrease deamination of 1, 2, 7 and 9.

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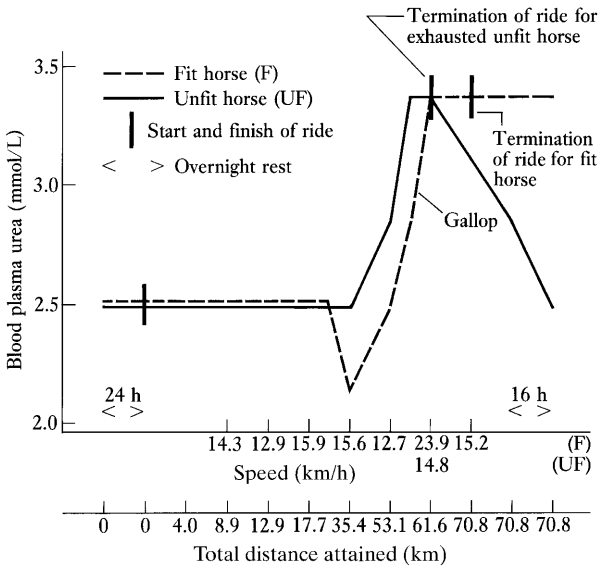


Figure 2.8 Effect of speed and distance on the concentration of blood urea in two horses. The rise in plasma urea of the unfit horse resulted from stress other than that caused by galloping (Frape *et al.* 1979).

tissue protein catabolism for gluconeogenesis (Figure 2.8; see also Chapter 9 on N metabolism). The carbon skeleton of deaminated dietary glucogenic (glycine, alanine, glutamic acid, proline, and methionine) and ketogenic (leucine and in part isoleucine, phenylalanine and tyrosine) amino acids is used as an energy source.

The extent to which dietary protein meets the present requirements of the horse depends on its quality as well as its quantity. The more closely the proportions of each of the different indispensable amino acids in the dietary protein conform with the proportions in the mixture required by the tissues, the higher is said to be the quality of the protein. It has been shown that those proportions in the common sources of feed proteins are such that lysine is most likely to limit utilization of the protein and threonine is the second most likely. If a protein, such as maize gluten, containing a low proportion of lysine is digested, the amount of it which can be used in protein synthesis will be in proportion to its lysine content. As the lysine is limiting, little of it will be wasted, but, conversely, the other amino acids, both dispensable and indispensable, will be present in excessive quantities and so will be deaminated to an alarming extent. If the relative deficiency of lysine in gluten is made good by supplementing the diet with either soya, or synthetic lysine, then the proportions of all the amino acids available in the blood plasma will more closely conform with the requirement, so that a higher proportion of all can be used for protein synthesis (Figure 2.7).

Amino acid and protein needs for growth and exercise

Growth

A young horse with a mature weight of 450 kg normally gains 100 kg between three and six months of age at the rate of 1 kg daily. Growth rate in kg per day then declines and the next 100 kg are gained between about 6 and 12 months of age and the following 75 kg between 12 and 18 months (Hintz 1980a). From a very young age the rate of gain per unit of body weight decreases continuously, while the daily maintenance requirement increases (see Chapter 8). As the weaned foal grows, an increasing proportion of that daily gain is composed of fat and a decreasing proportion is lean. It is thus apparent that the dietary requirement for protein and lysine decline with increasing age in the growing horse. For colts aged three months, a maximum rate of gain has been achieved with diets containing 140–150 g protein/kg and 7.5 g lysine/kg.

Diets differ in the amount of digestible energy (DE) they provide per kg. For obvious reasons it is more accurate to state the protein requirements as a proportion of the DE or net energy (NE). TB and Quarter Horse yearlings require 0.45 g lysine per MJ DE (see Chapters 6 and 8). A compound stud nut for young growing horses contains about 12–13 MJ DE/kg and oats about 11 MJ DE/kg. However, hard hay, containing 50–60 g protein/kg provides 7.5–8 MJ DE/kg. If the yearling consumes a mixture (approximately 50 : 50) of concentrates and hay, the diet provides on average 10 MJ DE/kg and the minimum lysine requirement is 4.5 g/kg total diet (i.e., 0.45 g/MJ DE). Hay containing 50–60 g/kg protein contains only 2 g/kg digestible lysine and therefore the concentrate should contain at least 7 g lysine/kg in order to meet minimum requirements. A yearling consuming 9 kg daily of total feed of this type would receive about 40 g lysine. Several studies have been undertaken with growing TB and Quarter Horses to measure their growth response to the addition of lysine-HCl to conventional 12% CP concentrates containing maize, oats and soya-bean meal, fed with hay. Graham *et al.* (1993) gave yearlings such a concentrate, to appetite, twice daily with coastal Bermuda grass hay (1 kg/100 kg BW) for 112 days (Table 2.1). The concentrate was supplemented with 2 g/kg diet lysine or with 2 g/kg lysine

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Table 2.1 Treatment mean response (with SEs) of TB and Quarter Horse yearlings to amino acid supplementation of a concentrate mix (Graham *et al.* 1993).

	Gain per feed (g/kg DM)	Daily weight gain (g/day)	Girth gain (cm)	Urea N (mg/g serum protein)
Basal	71.0	570 (20)	9.7 (0.49)	2.6 (0.20)
Basal + lysine	77.2	640 (20)	10.1 (0.46)	2.5 (0.19)
Basal + lysine + threonine	78.7	670 (20)	11.3 (0.47)	2.0 (0.19)

plus 1 g/kg threonine, or neither, both supplements raising the lysine content of the protein supplement to 7 g/kg. Amino acid supplementation increased the rate of body weight gain and the efficiency of the gain, while decreasing serum urea content. This reduction suggests that threonine improved the amino acid balance of the diet. Threonine increased the increment in muscle gain over the increment due to lysine, as indicated by the absence of an increase in rump fat with the amino acid supplements.

Much of the growth of horses takes place on pasture. Leafy grass protein of several species has been shown to contain 55–59 g lysine/kg. During the growing season the protein content in the DM of grass varies considerably (from 110 to 260 g/kg in the leaf), whereas the flowering stem contains only 35–45 g/kg. Thus, the lysine content of the grazed material as a fraction of air-dry weight can vary from 5 to about 13 g/kg, and, if a *leafy* grass diet is supplemented with a concentrate mixture, the lysine and protein requirements are met by cereals as a source of that protein. But as the quality of pasture varies greatly, the protein and lysine requirements are not always met with cereals alone. Grazing TB mares and foals were given either a 14% CP mix composed of maize, soya-bean meal and corn oil, or a 9% CP cereal supplemented with 6 g/kg L-lysine and 4 g/kg L-threonine (Staniar *et al.* 1999). Growth rates were similar over 12 months, although the latter group of foals grew faster in October, following weaning, when pasture quality was declining. Table 10.4 gives some analytical data for pasture in several months of the grazing season.

Exercise

Wickens *et al.* (2003) observed that N retention could be increased over a 14-day period by increasing the protein intake of exercised horses to 12.5% above the NRC (1989) recommendations. This period could be inadequate and measures of assessment such as urinary N increases, and other influences on N metabolism during running, described in Chapter 9, may be more relevant. Antilley *et al.* (2007) measured N retention to determine the value of synthetic amino acid supplementation and concluded that this was not an effective response criterion. N retained as a percentage of absorbed N may be acceptable if the dosage adopted achieves the same or a higher N retention value as that for an amino-acid-sufficient diet. In support of Wickens *et al.* (2003), Lawrence *et al.* (2003b) estimated N retention in exercising and sedentary horses to be 93 and 53 mg/kg BW, respectively, not accounting for losses in sweat. Lightly exercised adult horses supplemented with 2.5 g lysine and 2.0 g threonine per kg diet increased their muscle mass over a 14-week period (Graham-Thiers *et al.* 2003). Graham-Thiers *et al.* (2003), Graham-Thiers & Kronfeld (2005) gave aging, lightly exercised horses a hay/grain diet containing CP to meet recommendations (providing 6.9 g lysine plus 6.7 g threonine per 100 kg BW daily); but additional supplementary lysine (20 g/day) and threonine (15 g/day) resulted in increased muscle mass scores, weight gain, reduced fatness and lowered blood urea-N (decreased 3-methyl histidine, a by-product of the breakdown of actin and myosin proteins in muscle fibres, is a marker of the degree of muscle catabolism and increased plasma creatinine, is a marker of increased muscle mass). The authors concluded that supplementation with amino acids improved the maintenance of muscle mass regardless of age. Wickens *et al.* (2003) found that N retention was maximized in moderately exercised horses receiving 1016 g CP/d. These results led to a recommendation of 1.9 to 2.1 g dietary CP/kg BW/day for heavily exercised horses (NRC 2007, Table 2.2). Using 3-methylhistidine to determine minimum muscle protein turnover, Wickens *et al.* (2005) concluded that the requirement for maximum N retention in exercising horses is 954 g CP/day (1.9 g CP/kg BW daily, range 1.65–2.20 g CP/kg BW daily). Plasma concentrations of branched chain amino acids (BCAA) increase during exercise (Assenza *et al.* 2004) and decrease following exercise and

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Table 2.2 Crude protein, lysine and threonine requirements of growing, working and breeding horses, based upon NRC (2007) recommendations, (in g/kg BW per day, except for ADG). N.B. (1) These requirements assume positive linearity with BW. (2) CP requirements assume the protein sources meet the limiting amino acid needs. (3) NRC assumes that the efficiency, E, of dietary protein use is 0.5 at 4–6 months, 0.45 at 7–8 months, 0.4 at 9–10 months and 0.3 for horses >11 months old. This accounts for the constant and high requirement/kg ADG>6 months. The elevated maintenance requirements will also apply to fully active horses.

	Protein	Lysine	Threonine
Maintenance for growth, elevated*	1.44	0.0620	0.0445
Growth (g/kg ADG/day to 6 months)[A]	512.0	22.0	15.5
Growth (g/kg ADG/day >6 months)[A]	844.0	36.3	25.6
Maintenance, adults, minimum*	1.08	0.0464	0.0333
Maintenance, adults, average*	1.26	0.0542	0.0389
Maintenance, adults, maximum*	1.44	0.0620	0.0445
Maintenance and light exercise[A]	1.40	0.0602	0.0432
Maintenance and moderate exercise[A]	1.54	0.0660	0.0474
Maintenance and heavy exercise[A]	1.72	0.0742	0.0532
Maintenance and very heavy exercise[A]	2.01	0.0864	0.0620
Gestation, months 1–6[C]	1.26	0.0590	0.0423
Gestation, months 7–8[C]	1.45	0.0622	0.0446
Gestation, months 9–11[C]	1.56	0.0666	0.0478
Lactation, foaling to 3 months[BC]	3.06	0.168	0.121
Lactation, 4–5 months[BC]	2.73	0.147	0.105
Lactation, >5 months[BC]	2.53	0.134	0.096
Stallion, nonbreeding	1.44	0.0620	0.0445
Stallion, breeding	1.58	0.0678	0.0487

*Maintenance amount is adequate for N balance, i.e. Dig. N intake – (endogenous faecal + urinary + cutaneous N losses), with adequate dietary energy.

[A]Where appropriate, growth requirements should be added to this maintenance requirement, e.g. yearlings in training.

[B]Requirements assume milk yield is directly and linearly proportional to BW.

[C]Including maintenance of the non-pregnant horse.

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during recovery (Trottier *et al.* 2002). These amino acids are oxidized for energy during exercise in muscles; but supplementation with BCAA for exercise has produced inconclusive results (Casini *et al.* 2000).

Hormone and neurotransmitter secretion

Inadequate dietary protein causes a fall in the concentration of albumin, total protein and, according to Scandinavian evidence, the free essential amino acids isoleucine, leucine, lysine, phenylalanine, threonine and valine in plasma (see Chapter 8). These changes severely restrict the rate of protein synthesis and influence hormone secretion. Aspartic acid and glutamic acid are secretogogues for growth hormone and aspartic acid is also, to some extent, a secretogogue for gonadotropic hormones, whereas arginine and lysine are secretogogues for prolactin and insulin (Sticker *et al.* 1999). Tryptophan is a precursor of the neurotransmitter serotonin. Nevertheless, an oral dose of 50 g tryptophan in Standardbreds, two hours before exercise, was insufficient to induce a serotonin response, although maximum mean heart rate during exercise was decreased (Vervuert *et al.* 2003c).

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NON-PROTEIN NITROGEN

Urea is synthesized in the liver from amino acids present in excess of need so that a rise in dietary protein above requirements is associated with a rise in plasma urea (Figures 1.6 and 2.7). In ponies given diets containing 6–18% protein, between 200 and 574 mg urea N/kg metabolic BW ($W^{0.75}$) daily are recycled and degraded in the intestinal tract. In a pony weighing about 150 kg, this range is equivalent to 54–154 g CP/day.

While urea is within the tissues of a horse it cannot be degraded or otherwise utilized. However, when provided with an adequate source of dietary energy, microorganisms – chiefly in the large intestine – utilize it in protein synthesis, first degrading it to ammonia by the action of bacterial urease. In the absence of an adequate supply of energy, which is normally present as fibre, starch and protein, a proportion of the ammonia, at a relatively neutral pH, diffuses back into the blood and is not effectively utilized either by the horse (except as described below), or by its captive microorganisms. A fine balance is required, for in the absence of sufficient nitrogen, microbial growth cannot occur at a maximum rate and there will therefore be a submaximal rate of fibre breakdown and utilization will not prevail. Whereas circulating urea is nontoxic to the horse, except when very high concentrations affect osmolality, absorbed ammonia is highly toxic. A healthy liver copes adequately with low concentrations in the amination of keto acids, forming dispensable amino acids, and by urea synthesis. However, if liver failure occurs, and this is more frequent in older horses, ammonia intoxication can occur without any increase in blood urea (see Chapter 11).

The flow of urea and other nitrogen compounds into the large intestine from the ileum varies with the quantity of diet and its type (Table 1.6). These digesta are relatively impoverished of N in horses receiving a diet of straw. The provision of NPN or, for that matter, protein as a supplement to this diet, results in an increased flow of N and a stimulation of microbial growth in the large intestine. Urea, or more effectively biuret, added to low-protein diets in concentrations of 1.5–3%, has increased N retention in both adult and growing horses with functioning large intestines, and pregnant mares subsisting on poor pasture apparently benefit from the consumption of supplementary urea.

Nevertheless, in most other circumstances the response to a urea supplement is poor and difficult to justify. Martin *et al.* (1996a) found that no nutritional benefit could be derived by mature horses from urea supplementation of a low-protein diet, in that N balance was not increased. Where urea has been given to lactating mares the limiting factor has usually been energy intake. In this situation, feed intake and body weight have been depressed and plasma urea N has been increased without raising blood ammonia concentration.

The addition of urea or biuret to low-protein, poor-quality hay diets increases DM and fibre digestion. This response is small from a practical viewpoint. Detailed studies with adult geldings, conducted by Martin *et al.* (1996b), failed to find any improvement in barley straw digestion, measured as DM, OM, or NDF digestibility, from the addition of 20.3 g urea/kg dietary DM to a diet containing 4.4 g/kg N.

In summary, it would seem that horses and ponies with functioning large intestines given diets containing less than 7–8% CP make only minor use of supplementary NPN as an adjunct to that secreted back into the small intestine in digestive secretions or directly from the blood. The reason for this is that bacterially synthesized amino acids are absorbed from the large intestine in only small amounts. In ruminants, large amounts of soluble N entering the rumen lead to a rapid production of ammonia and therefore to ammonia toxicity. Limited evidence (Figure 1.6) does not support the widely held view that excessive protein consumption *per se* predisposes horses to laminitis.

Treatment of ammonia toxicity

Ammonia toxicity, expressed as hyperammonaemia (blood levels exceeding 150 µmol/L; note that careful sample handling is required, with rapid analysis), caused by excessive dietary NPN, or protein, is less likely in the healthy horse with normal hepatic function, chiefly because much of the nitrogen is absorbed into the bloodstream before it reaches the regions of major microbial activity in the large intestine. Hyperammonaemia has been produced experimentally with the ingestion of large amounts of urea, but in these cases blood urea is also elevated. Where serum urea levels are normal (6–8 mmol/L), liver dysfunction is frequently the cause of hyperammonaemia with encephalopathic signs (ammonia readily crosses the blood–brain barrier to compete with K). Peek *et al.* (1997) reported evidence of hyperammonaemia associated with normal blood urea and liver enzyme levels, but with hyperglycaemia and acidaemia. Clinical signs included head pressing, symmetric ataxia, tachycardia, diarrhoea, and behaviour suggesting sudden blindness and abdominal pain.

Ammonia interferes with the citric acid cycle, oxidative phosphorylation and aerobic metabolism, resulting in lactic acidosis and hyperglycaemia. Treatment should therefore include administration of fluids, excluding

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dextrose, but including strong ions to counteract acidosis, given slowly intravenously (i.v.). Where liver dysfunction has been eliminated as a cause, the origin of the ammonia is likely to be the large intestine. In this case, oral acidifying agents, such as lactulose, which decrease ammonia absorption by converting it to the ammonium ion, should also be given.

LAMINITIS AND ENERGY INTAKE

An increased risk of laminitis and colic have been associated with an abrupt overload of non-structural carbohydrate (NSC), attributed to hydrolysable carbohydrate (CHO-H) in grain concentrates as well as to rapidly fermentable carbohydrates (CHO-F_R) in pastures. Hoffman *et al.* (2001a) demonstrated that CHO-H accounted for 97% or more of the NSC in concentrates, but for only 33% in pasture and hay, the remainder being CHO-F_R. These workers found that pasture was surprisingly rich in CHO-F_R during the autumn and this fraction of NSC is a major contributor to laminitis risk. Horses should be adjusted gradually to lush pasture by slowly increasing their daily period of access to allow the microbial population of the large intestine to adapt. Similar advice applies to concentrate feeding (see also Chapter 11 for discussion of insulin resistance, obesity and laminitis).

De Fombelle *et al.* (1999) abruptly introduced 30% or 50% of the DM as barley into a hay diet, as part of two daily meals for ponies. At each meal the barley was consumed before the hay. The amount of starch did not exceed 2.3 g/kg BW per meal, but the colonic lactate concentration increased 10-fold and the *Lactobacillus* and *Streptococcus* populations were increased in the right ventral colon 5 h following the diet change, without a significant decrease in pH. After 14 days on 50% barley, the large intestinal pH had dropped from 6.74 to 6.26, in association with a decline in the population of cellulolytic bacteria. A consequential reduction in the digestion of fibre could create a favourable situation for large intestinal impaction (Reeves *et al.* 1996). (A method for *ad libitum* feeding of concentrates is outlined in Chapter 8.) As horses utilize dietary fat efficiently, the gradual introduction of a diet rich in fat and fibre should play a useful role in prevention of digestive and metabolic disorders (Williams *et al.* 2001b). Ponies which have previously suffered laminitis are much more glucose intolerant (Jeffcott *et al.* 1986) than those which have not (see Chapter 11). Obesity in humans delays the plasma clearance of fat from a meal, owing to low insulin sensitivity of receptors in adipocytes. It has been shown that 4 days of fasting can cause visibly lipaemic plasma in ponies but not in horses, and the same factors are associated with laminitis. Moderate regular exercise can prevent both laminitis and insulin resistance. The laminitic risks associated with body-fat accumulation in overweight animals should be appreciated. Feed intake should be reduced gradually to decrease fat deposition, consistent with the avoidance of hyperlipidaemia. For horses and ponies with both normal- and high-condition scores, the selection of feeds suitable for laminitis requires analytical evidence.

General requirements of analytical method

The analytical method should be capable of estimating the following in feed:

- total starch* plus indigestible, rapidly fermentable carbohydrate (CHO-F_R); and
- neutral detergent fibre (NDF). (Improved analytical methods for fibre have been discussed previously, see McCleary 2003, and associated papers.)

The method of Hoffman *et al.* (2001a) (Figure 2.9) allows these characteristics of feed to be estimated from determinations of the moisture, crude protein, fat, ash and NDF. The non-structural carbohydrate (NSC) is then calculated by summing these five determinants and subtracting from 100. Average NDF and ADF values of feeds are given in NRC (2007) and some will be found in Appendix C.

The NSC consists of hydrolysable carbohydrates (CHO-H) and rapidly-fermentable carbohydrate (CHO-F_R). The majority of CHO-F_R reaches the large intestine, whereas the proportion of CHO-H reaching the large gut depends on:

- its digestibility; and
- the quantity of feed given in a meal.

If the feed material contains a small quantity of mono- and di-saccharides (except lactose), which are normally digested in the small intestine, they are extracted with boiling water. The remaining CHO-H can then, if necessary, be determined following enzymatic hydrolysis of starch. The value of (CHO-F_R CHO-H) should not exceed 0.25% of BW per meal to provide a reasonably low risk of diet-related laminitis. NRC (2007) recommends that single-meal intakes of starch are less than 0.2–0.4%

*It is informative to have an estimate of the fraction of starch that is digestible, the remainder being 'resistant' starch (resistant to hydrolysis by digestive enzymes).

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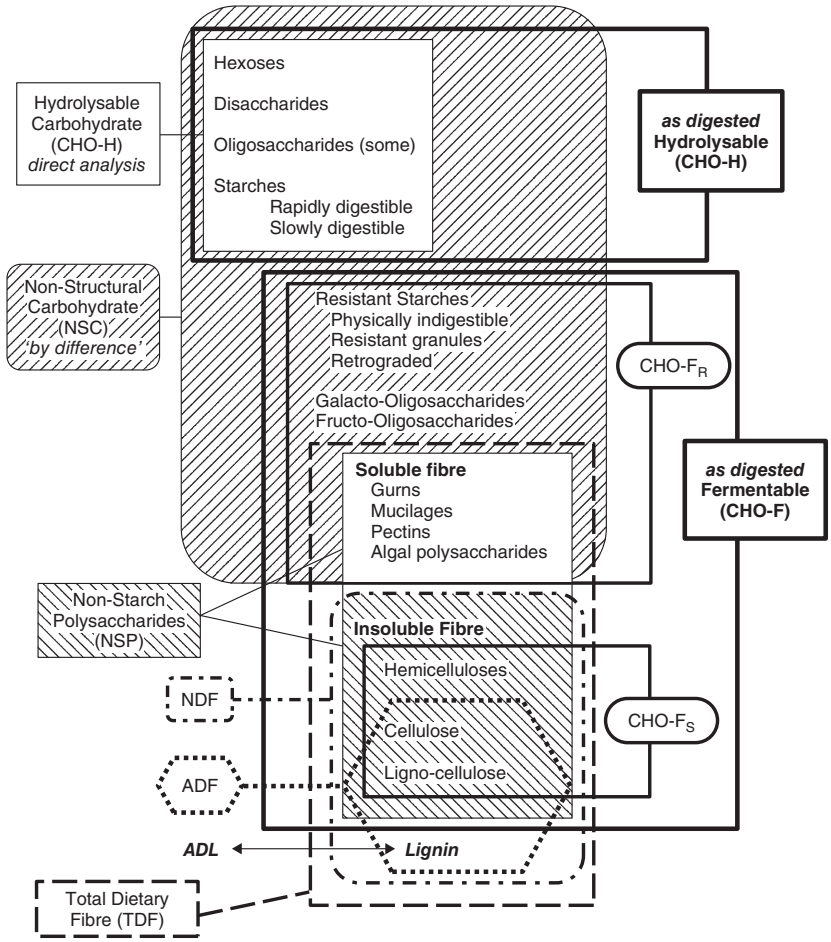


Figure 2.9 A scheme of dietary carbohydrate fractions for the horse. Proximate analysis fractions of the diet are noted on the left of the figure. NSC is estimated as a difference: NSC = 100 – (water + protein + fat + ash + NDF). The fraction, Total Dietary Fibre, is frequently mentioned in descriptions of foods for human consumption. The fractions, as used by the horse, digestible carbohydrate (CHO-H) and fermentable carbohydrate (CHO-F), are noted on the right of the figure. CHO-H is the carbohydrate fraction that is digested by the horse with the release mainly of glucose, unless excessive amounts of starch are given in a meal, whereas resistant starches will pass to the hind-gut and together with excess CHO-H will constitute a risk of digestive upset. CHO-F is further subdivided into a rapidly fermented fraction (CHO-F_R), contributing to the risk, and a slowly fermented fraction (CHO-F_S). Reproduced from Hoffman *et al.* (2001a), with permission from the American Society of Animal Science.

of BW. The level here is obviously dependent on the digestibility of the starch, as the limit is to minimize the amount entering GI regions of principal microbial fermentation.

It is recognized that pasture, as a feed source, presents major issues, both in definition of its chemical characteristics and quantity grazed per hour. In order to prevent excessive grazing it is necessary for the horse to eat an adequate amount of a safe feed before it is released onto pasture. This can be difficult to achieve. Evidence indicates that fructans (and possibly other indigestible oligosaccharides, all of which are components of CHO-F_R) present in pasture plants are a major cause of laminitis, discussed in detail in Chapter 11. It has been estimated that a 500 kg horse at pasture would consume, on average, 1.8 kg of readily fermentable CHO-F_R per day.

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With respect to laminitis risk, safe feeds include:

- good quality hay;
- nutritionally improved straw (NIS);
- cereal straw;
- oat feed;
- soya hulls; and
- vegetable oil.

Feeds acceptable in restricted quantities:

- dried lucerne (alfalfa) and grass pellets;
- molasses;
- wheat bran; and
- sugar beet pulp.

STUDY QUESTIONS

(1) The horse evolved as a browsing animal engaging in many small feeds each day. What impact has this had with respect to:
   (a) food selection and metabolic responses;
   (b) social habits; and
   (c) safety in the wild?

(2) What is meant by limiting dietary amino acids and what is the relation, if any, between this and (a) maintenance diets and (b) production diets? What is meant by nitrogen balance?

(3) What factors should be considered when a horse has lost its appetite for sufficient feed to maintain body weight?

FURTHER READING

Englyst, K.N. & Englyst, H.N. (2005) Carbohydrate availability. *British Journal of Nutrition*, **94**, 1–11.

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3
The Roles of Major Minerals and Trace Elements

Grass is the first nourishment of all colts after they are weaned. … Whereas when they are fed with corn and hay, but especially with the first, … it exposes them to unspeakable injuries.

W. Gibson 1726

MAJOR MINERALS

Calcium and phosphorus

Function

The functions of calcium (Ca) and phosphorus (P) are considered together because of their interdependent role as the main elements of the crystal apatite, which provides the strength and rigidity of the skeleton. Bone has a Ca : P ratio of 2 : 1, whereas in the whole body of the horse the ratio is approximately 1.7 : 1.0, because of the P distribution in soft tissue. Bone acts as a reservoir of both elements, which is tapped when diet does not meet requirements. The elements of bone are in a continual state of flux with Ca and P being removed and redeposited by a process that facilitates the reservoir role and enables growth and remodelling of the skeleton to proceed during growth and development. The acute role of Ca relates to its involvement in a soluble ionic form for nerve and muscle function. Consequently [Ca^{2+}] concentration in the blood plasma must be maintained within closely defined limits.

Control of plasma Ca ion concentration and Ca and P metabolism

The flux and distribution of Ca and P in the body are strictly regulated by two proteinaceous hormones, functioning antagonistically at the blood–bone interface, the intestinal mucosa and the renal tubules (see also under 'Vitamin D', Chapter 4). These are the potent parathyroid hormone (PTH), secreted by the parathyroid glands adjacent to the thyroid glands, and the less significant calcitonin, or thyrocalcitonin, secreted by the parafollicular C cells of the thyroid gland. A slight decrease in [Ca^{2+}] concentration in extracellular fluid of horses causes the immediate secretion of parathyroid hormone (Estepa *et al.* 1998) and the stress of pregnancy and lactation cause the enlargement of the parathyroids. Excessive [Ca^{2+}] concentration, such as occurs in vitamin D toxicity, leads to a decreased activity and size of the glands. Calcitonin, on the other hand, rapidly decreases blood [Ca^{2+}] concentration by decreasing osteoclastic, and increasing osteoblastic, activities.

The horse kidney seems to play a greater part in controlling concentrations of Ca in the blood than does the intestinal tract and this has practical significance for diet and renal disease. The mean values and ranges for serum total Ca and P are listed in Table 3.1. Normal resting plasma phosphate concentration decreases with increasing age and ionized plasma Ca concentration is approximately 1.7 mmol/L lower than the total values given in the table.

Serum phosphate values vary without untoward physiological effects. For example, strenuous exercise can depress blood phosphate to half the resting value for 2–2.5 h. Nutritional secondary hyperparathyroidism (NSHP) is a diet-related clinical disorder of horses in which increased blood PTH concentration, normal or slightly raise serum phosphate concentration and slightly depressed serum Ca values occur (Estepa *et al.* 2003; Figures 3.1 and 3.2). Hyperphosphataemia reduces blood Ca concentration, by the law of mass action and suppresses PTH's ability to stimulate renal activation of vitamin D, further decreasing digestive absorption efficiency (Bertone 1992). This contrasts with primary hyperparathyroidism where elevated PTH concentration is associated with hypercalcaemia (Frank *et al.* 1998), and where PTH leads

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Table 3.1 Mean values and ranges for serum total concentrations (mmol/L) of electrolytes in horses of different ages. (Modified from published tables of S.W. Ricketts, Beaufort Cottage Laboratories, Newmarket, Suffolk.)

		Birth to 36 h	3 Weeks	Yearlings	Horses in training	Mares at stud
Ca	Mean	3.2	3.2	3.3	3.4	3.4
	Range	2.7–3.6	2.5–4.0	2.7–4	2.6–3.9	2.9–3.9
P	Mean	2.5	2.5	1.8	1.3	1.1
	Range	1.2–3.8	1.6–3.4	1.4–2.3	1.1–1.5	0.5–1.6
Na	Mean	136	137.5	138.5	138.5	138.5
	Range	126–146	130–144	134–143	134–143	134–143
K	Mean	4.8	4.5	4.3	4.3	4.3
	Range	3.7–5.4	3.6–5.4	3.3–5.3	3.3–5.3	3.3–5.3
Mg	Mean	0.83	0.81	0.78	0.78	0.78
	Range	0.57–1.10	0.66–1.10	0.62–1.10	0.62–1.10	0.62–1.10
Cl	Mean	—	—	—	—	—
	Range	Normal range for all ages 99–109				

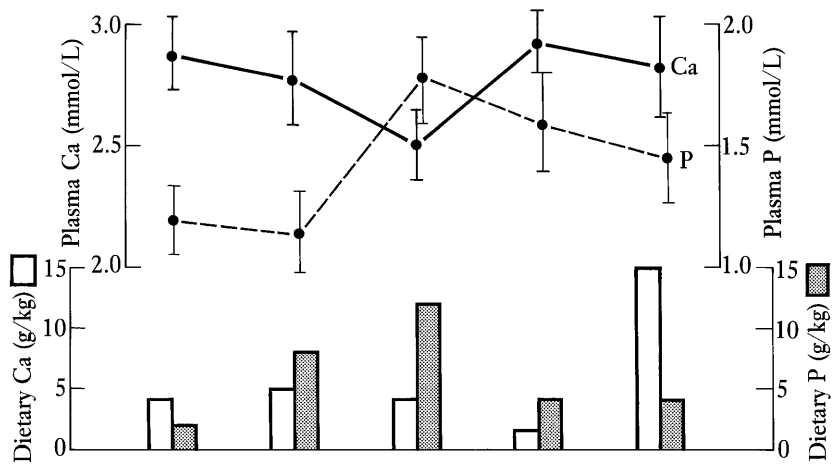


Figure 3.1 Effects of dietary Ca (□) and P (⚄) on mean concentrations in blood plasma, ±SE (after Schryver, Hintz & Lowe 1971b).

to increased fractional renal clearance of phosphate and elevated serum alkaline phosphatase (ALP), as a result of bone osteoclastic activity. However, By far the most common hypercalcaemic state in the horse occurs in renal disease. The other states are: hypervitaminosis D and pseudohyperparathyroidism. The latter condition is caused by an elevated concentration, found in mare's milk, but also in serum, of parathyroid hormone-related protein (PTH-rP), which is the major cause of malignancy-associated hypercalcaemia (van der Kolk 2007). There have been reports of calcinosis in horses from the consumption of *Cestrum diurum* (in Florida) and *Tricetum flavescens* (in Austria). These plants increase the release of $1,25(OH)_2D_3$, so increasing Ca absorption and resulting in hypercalcaemia (Mello 2003).

NSHP is well recognised, but is much less common today. Its typical presentation is fibrous osteodystrophia of the facial bones ('big-head'), present in a pony I once purchased from the Welsh hills. Nevertheless, NSHP occasionally occurs without the typical facial abnormality

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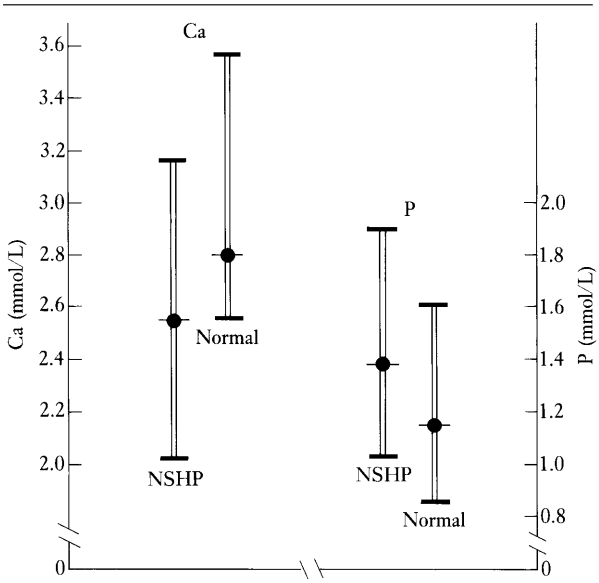


Figure 3.2 Mean and range of serum Ca and P concentrations in spontaneous NSHP (Krook 1968). Notice how in cases of NSHP Ca is depressed and P elevated.

(Little *et al.* 2000). Other clinical signs include a stiff gait, or shifting-limb lameness, abnormal mastication with oral dysphagia and upper airway stridor.

Ca and P in bone

Horses have no 'horse sense' when it comes to selecting a diet containing a balanced mixture of Ca and P – they prefer the palatability of a P-rich diet, whereas this is not available for selection in the natural grazing environment. Hence, a dietary Ca deficiency is not an infrequent occurrence among domestic horses.

Inadequate dietary Ca and P for growing foals causes a delay in the closure times of the epiphyseal plates of long bones and contributes to developmental orthopaedic disease (DOD). In adult working horses it causes lameness and bone fractures. Failure of the osteoid, or young bone, to mineralize is called rickets in young and osteomalacia in adult horses.

In extreme cases, when mineral is being reabsorbed from bone, the outcome is osteodystrophia fibrosa in which fibrous tissue is substituted for hard bone as occurs in 'big-head'. In the presence of vitamin D in each of these conditions the body, through the agency of PTH, is endeavouring to maintain homeostasis of blood Ca by accelerating the removal of Ca from the bones and increasing the tubular reabsorption of Ca. Diets based on wheat bran and cereals are rich in organic P and low in Ca, predisposing horses to these conditions.

The tendency to lower blood [Ca^{2+}] leads to increased bone resorption, increased renal excretion of phosphate, an increased rate of bone–mineral exchange and to a greater susceptibility of bones to fracture. Deposition of Ca salts in soft tissue, including the kidney (nephrocalcinosis), becomes apparent. A physiological example of rising serum PTH concentration occurs in periparturient mares during mammary secretion of Ca when there is a decrease in serum total and ionized Ca concentrations (Martin *et al.* 1996b), and an active fragment of PTH has been detected and measured in mare's milk (Care *et al.* 1997). The hormone calcitonin opposes the effects of PTH. When plasma Ca concentration is elevated, PTH secretion is decreased, reducing plasma Ca by decreasing the activity of osteoclasts and increasing that of osteoblasts.

Hypocalcaemia

Ca is principally an extracellular cation existing in ionized [Ca^{2+}], complexed and protein-bound forms. The ionized form is physiologically active and its concentration in blood plasma is precisely controlled, but the concentration is influenced by acid–base changes (see also Chapter 9). Normal serum total Ca concentration in the horse is 3.2 mmol/L, of this ionized [Ca^{2+}] concentration is about 1.5 mmol/L, although laboratory method influences the values.

Pronounced hypocalcaemia is unlikely to result from a dietary inadequacy of Ca, but rather from metabolic alkalosis. Thus, hypocalcaemia occurs in adult horses as a post-exertional stress. The clinical signs are: muscle fasciculation and tetanic spasms, incoordination, synchronous diaphragmatic flutter, decreased gut sounds and even inability to stand. Extended work and overheating lead to a rise in blood pH that depresses the concentration of [Ca^{2+}] in the blood. Moreover, elevated body temperature *per se* can bring about a loss of 350–500 mg Ca/h in sweat and this rate of loss can exceed the capacity of blood replenishment by bone mobilization.

Fast exercise causes a fall in blood pH and is expected to cause a slight rise in plasma [Ca^{2+}]; but, surprisingly, Vervuert *et al.* (2002, 2003b) detected a fall in plasma pH and [Ca^{2+}], but rises in lactate, P_i (inorganic P) and intact PTH. Low-speed exercise resulted in an increase in pH, whereas lactate, [Ca^{2+}], total Ca, P_i and PTH were unchanged. Nevertheless, in accordance with the mechanism for homeostatic control of plasma[Ca^{2+}], a close negative relationship between intact PTH and [Ca^{2+}] was

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maintained (Vervuert *et al.* 2002). Intact PTH is a mediator in counter-regulation of exercise-induced hypocalcaemia by reabsorption of renal Ca or by osteoclast-mediated bone resorption.

Problems have been associated with the determination of true mineral availability (Coenen 2005a; O'Connor & Nielsen 2006). Garcia-Lopez *et al.* (2001) and Lopez *et al.* (2006) reported that the total serum Ca of a healthy horse should have mean values of 48.5% of physiologically active [Ca^{2+}], 47.4% protein-bound Ca and 4.1% Ca complexed with weak acids, with a normal reference range for total serum Ca of 2.69–3.37 mmol/L (10.8–13.5 mg/dL). For [Ca^{2+}], the normal range of values for healthy horses is 1.45–1.75 mmol/L heparinized blood and for normal serum, 1.58–1.90 mmol/L (Van der Kolk *et al.* 2002), 1.61–1.68 mmol/L (6.44–6.74 mg/dL) (Garcia-Lopez *et al.* 2001), or for hypocalcaemic serum, ionized values of 1.50 mmol/L (<6 mg/dL) (Toribio *et al.* 2001). The Spanish group showed that when plasma [Ca^{2+}] fell to 1.25 mmol/L, PTH attained a maximum of >80 pg/mL, but, when plasma [Ca^{2+}] rose to 1.8 mmol/L, PTH dropped to a minimum of <12 pg/mL). Van der Kolk *et al.* (2002) found that the average EDTA plasma concentrations for intact PTH and for C-terminal PTH were 0.6 pmol/L and 96 pmol/L, respectively. The Spanish workers, on the other hand, found a plasma PTH concentration of 20–30 pg/mL, determined by radioimmunoassay, when using an antibody against intact human PTH (MW 9424), and 35–45 pg/mL using an antibody against the amino-terminal region of rat PTH. This fragmented region possesses biological activity. Van der Kolk's values for intact PTH are lower than the Spanish values (0.6 pmol/L, equivalent to 5.7 pg/mL, human PTH).

As [Ca^{2+}] concentration varies with blood pH, Van der Kolk computed a prediction equation for heparinized blood at pH 7.4:

$$[Ca^{2+}]_{pH7.4}\ \text{mmol/L} = -6.4570 + 0.8739(pH_{measured}) + 0.9944([Ca^{2+}]_{measured}\ \text{mmol/L})$$

The i.v. administration of a solution of 50–100 mmol $CaCl_2$, or Ca gluconate, in 1 L over 30 min should resolve the clinical signs of hypocalcaemia. The higher dose can cause cardiac arrhythmias and so administration should be accompanied by cardiac auscultation, decreasing the rate of dosing if necessary. Hypercalcaemia also induces cardiac arrhythmias (hypo- and hypercalcaemias, respectively, prolong and shorten the Q–T interval of the electrocardiogram).

Dietary levels of Ca and P in relation to requirement

Excessive amounts of dietary Ca do not initiate the kinds of dietary problems encountered in other domestic species. However, one experiment in which Shetland foals aged four months were given a diet containing 25 g Ca/kg, with a Ca : P ratio of 6 : 1, for four years (Jordan *et al.* 1975) resulted in a slight enlargement of the marrow region of the long bones and thinning of the cortical area, together with less bone mineral per unit of cortex. Lameness through loss of support for tendons and ligaments is frequently a characteristic of resorption of cortical bone. Excessive amounts of dietary Ca can make bones brittle, through abnormal bone storage of Ca. However, investigations in which diets containing 7–27 g Ca/kg were compared for up to two years, show that differences are small; bone density is increased by high-Ca diets and the cortex of the long bones is slightly thinner (Schryver *et al.* 1970a, 1971b, 1974a, 1974b). Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and assuming a growth rate of 1.0 kg/day on pasture, estimated the daily requirement for Ca and P to be 28 g and 21 g/day, respectively.

Rapid growth rate has questionable consequences. Weanling stock-type horses 187 days of age and 236 kg BW were fed to increase BW at 0.97 kg/day or 0.49 kg/day (Petersen *et al.* 2001) on diets that met, or exceeded, NRC (1989) mineral requirements. Growth at the faster rate caused a greater increase in bone mineral content, without compromising bone quality. Nevertheless, three of six of these horses developed clinical signs of **physitis**, accompanied by joint pain, demonstrated by knuckling over at the knee, from which they recovered by 302 days of age.

It has been shown that bone strength increases with increasing density and Ca content. Some precision has, therefore, been given to requirements by measurements of Ca retention. The increase of Ca intake to 115% of NRC (1989) recommendations (4.8 g Ca/kg diet, 11.7 MJ DE/kg) in growing horses increases Ca retention (Moffett *et al.* 2001) (Table 3.2). The bone density of juvenile Quarter Horses in training was increased by giving them moderately large amounts of Ca, P and Mg (respectively 151%, 130% and 159% of NRC 1989 recommendations) cf. lower amounts (respectively 136%, 98%, and 126% of NRC 1989) (Nolan *et al.* 2001).

Exercise is critical for bone strength and mineral content in growing horses (Henry *et al.* 2003). Exercise increases size and mass of the third metacarpal and increases serum carboxy-terminal propeptide of type I procollagen (PICP) concentration of weanling Quarter Horses (Williams *et al.*

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Table 3.2 The response of yearling Quarter Horses between 12 and 18 months of age to diets containing 115%, 100% and 85% of the NRC (1989) dietary requirements for calcium (Moffett *et al.* 2001).

	12 months	15 months	18 months
115%			
Intake (g Ca/day)	28.13	32.87	38.00
Apparent digestibility (%)	39.75	42.98	40.36
Retention (g/day)	8.07	12.86	11.64
100%			
Intake (g Ca/day)	25.35	29.14	39.40
Apparent digestibility (%)	38.75	38.89	55.85
Retention (g/day)	6.90	9.93	18.47
85%			
Intake (g Ca/day)	19.33	21.05	36.60
Apparent digestibility (%)	38.61	39.97	64.03
Retention (g/day)	2.74	7.49	18.87

2003a), markers of bone formation (Nielsen 2005). Short sprints stimulate bone formation in weanling horses, but slower speeds (Hiney *et al.* 2004), and draught load exercise at a low velocity (Vervuert *et al.* 2005b) have no, or little, effect, indicating the importance of sprint exercise in stimulating skeletal Ca deposition in young horses. It is probable that pasturing, as opposed to stalling (Bell *et al.* 1999), or holding in dry lots (Stephens *et al.* 2003) increases bone mineral content of young horses, partly for reasons of this kind. Nevertheless, despite higher Ca intakes than in those described by Moffett *et al.* (2001; Table 3.2), during the early stages of training, in long yearlings and two year olds, there is a period of demineralization of bone with a decrease in Ca and P retention, but not Mg (Stephens *et al.* 2001), so that the stress of hard exercise should be delayed. This demineralization is followed by a period of remineralization. The NRC (2007) Ca and P recommendations for resting and exercising horses are given in Table 3.3.

Bone turnover, including osteoblastic activity, is tracked with serum osteocalcin. Bone resorption, i.e. osteoclastic activity, is tracked by measuring the carboxy-terminal telopeptide of type I collagen (ICTP) and bone formation is monitored by measuring PICP. However, circadian changes in the serum concentrations of osteocalcin and IGF-I of two-year-old TB mares have been shown. The peak time for osteocalcin was 09.00 hours, while that for IGF-I, with less amplitude, was 17.30 hours (Jackson *et al.* 2003a). Amongst horses given 275% of NRC (1989) requirements for Ca and P, cf. those given 133% of requirements, the Ca concentration was higher amongst geldings of various ages (2 to >15 years old) given the greater quantity during both sedentary and exercise periods (Buchholz *et al.* 1999) and blood ICTP concentration was increased, indicating that bone remodelling was accelerated (Mansell *et al.* 1999). Juvenile racehorses given Ca, P and Mg intakes that were, respectively, 169%, 132% and 168% of NRC (1989) recommendations achieved higher rates of bone formation, with lower rates of demineralization, than did those given lower amounts (Michael *et al.* 2001). Apparent Ca digestibility declines with increasing dietary Ca concentration, yet increased Ca retention (positive balance) is likely above the requirement; although some of this excess will be excreted by the kidneys in the horse (Doorn *et al.* 2004b).The revised mineral requirements are given in Table 3.4 (cf. NRC 2007, Table 3.3).

The equine kidney plays a vital part in Ca homeostasis, and daily urinary Ca excretion shows a direct relationship with intake. In many species the urinary loss of Ca is raised by increased intakes of sodium. This relationship does not exist in the horse (see Sodium, this chapter). Diets rich in Ca yield urine containing a precipitate of Ca salts; the urinary loss of Ca in a 300 kg yearling given a diet containing 20 g Ca/kg was 20–30 g in 6–8 L of urine daily (that is, 0.36% Ca). The absence of calculus formation in the kidney demonstrates the horse's ability to deal with large amounts of Ca despite the low solubility of the

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Table 3.3 Major mineral requirements of growing, working and breeding horses, based upon NRC (2007) recommendations, (g/kg BW per day, except for ADG) N.B. These requirements assume positive linearity with BW.

	Calcium	Phosphorus	Sodium	Potassium	Magnesium	Chloride
Maintenance, foals*	0.072	0.040	0.020	0.050	0.015	0.080
Growth (g per kg ADG/d)[A]	32.0	18.0	1.00	3.0	1.25	0.013[D]
Maintenance, adults*	0.043	0.028	0.020	0.048	0.015	0.080
Maintenance for light exercise[A]	0.060	0.036	0.028	0.057	0.019	0.112
Maintenance for moderate exercise[A]	0.070	0.042	0.051[E]	0.064[F]	0.023	0.133[G]
Maintenance for heavy exercise[A]	0.080	0.058	0.066[E]	0.078[F]	0.030	0.155[G]
Maintenance for very heavy exercise[A]	0.080	0.058	0.082[E]	0.105[F]	0.030	0.186[G]
Gestation, months 1–6[B]	0.040	0.029	0.020	0.050	0.0150	0.080
Gestation, months 7–8[B]	0.056	0.041	0.020	0.050	0.0152	0.080
Gestation, months 9–11[B]	0.072	0.053	0.022	0.052	0.0154	0.082
Lactation, Foaling to 3 months[BC]	0.120	0.076	0.026	0.095	0.022	0.091
Lactation, 4–5 months[BC]	0.085	0.051	0.024	0.071	0.021	0.091
Lactation, >5 months[BC]	0.075	0.044	0.023	0.067	0.017	0.091
Stallion, nonbreeding	0.043	0.028	0.020	0.050	0.015	0.080
Stallion, breeding	0.060	0.036	0.028	0.057	0.019	0.080

*Maintenance is the dietary intake to achieve equilibrium, replacing endogenous (faecal, urinary and cutaneous) losses, and each requirement assumes an absorption efficiency of 0.5 for Ca and an efficiency of 0.35 or 0.45 for P.

[A]Where appropriate growth requirement should be added to maintenance requirement, e.g. yearlings in training.

[B]Including maintenance.

[C]Requirements assume milk yield is directly and linearly proportional to BW.

[D]Birth to 6 months of age, whereas 0.005 g/kg BW gain between 6 and 12 months of age and 0.0025 g/kg BW gain between 12 and 24 months of age.

[E]These figures assume that 3.1 g Na is required per kg BW **loss** as sweat during exercise and that 10 g to 20 g BW is lost per kg BW during exercise.

[F]These figures assume that 2.8 g K is required per kg BW **loss** as sweat during exercise and that 10 g to 20 g BW is lost per kg BW during exercise.

[G]These figures assume that 5.3 g Cl is required per kg BW **loss** as sweat during exercise and that 10 g to 20 g BW is lost per kg BW during exercise.

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element; conversely, a dietary deficiency of Ca yields urine almost devoid of the element.

Endogenous losses

In contrast, endogenous loss of Ca in faeces, representing the minimal obligatory loss that must be replenished from dietary sources, is largely unaffected by the dietary amounts. Endogenous faecal Ca and P have been estimated, respectively, to be 36 mg/kg BW and 18 mg/kg BW daily in growing horses (Cymbaluk *et al.* 1989b; NRC 2007) and 20 mg/kg BW and 10 mg/kg BW daily for adults not in training (NRC 2007). Urinary losses of Ca decrease by 50–75% in extended work (Schryver *et al.* 1975, 1978a), whereas sweat losses increase. During 20 min of hard work, ranges of 80–145 mg Ca and 11–17 mg P have been found in the total yield of sweat (Schryver *et al.* 1978c). Over a day's hard work this represents a considerable loss of Ca. On the other hand, horses and ponies idle for long periods retain less Ca than those worked. Following such an inactive period dietary Ca and P should be raised 20% above minimal requirement levels.

The proposed dietary requirements for Ca and P partly depend on the degree of bone density deemed necessary for adequate bone strength. The requirements given in

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Table 3.4 Daily requirements for Ca and P of growing horses (Schryver *et al.* 1974a; Grace *et al.* 1998b; Michael *et al.* 2001; Moffett *et al.* 2001; Nolan *et al.* 2001; Petersen *et al.* 2001). (Compare Table 3.3)

Age (months)	BW (kg)	Weight gain (kg/day)	Ca (g/day)	P (g/day)
3	100	1	55	31
6	200	0.8	55	29
12	300	0.6	50	28
18	375	0.3	50	28
Mature	450	0	46	27

Table 3.4 exceed those proposed by NRC (2007; Table 3.3), who concluded that the Ca intake of growing horses not in training should meet endogenous losses of 36 mg Ca/kg BW and growth needs of 16 g Ca/kg BW gain, all with a 50% absorption rate (and an absorption efficiency for P of 0.45 for foals and 0.35 for adults). The resultant Ca requirement is:

$$0.072\ \text{g/kg BW} + 32\ \text{g} \times \text{average daily liveweight gain (ADG), kg}$$

Stephens *et al.* (2004) reported that Ca retention was maximal when young horses in training were receiving 123 mg Ca/kg BW daily. NRC (2007) concludes that the Ca requirements for horses in training, typically under 2 years of age, would be met by the requirements for growth, given above. When the daily weight gain falls to 0.1 kg or less, NRC (2007) sets the requirements for heavy exercise at 0.08 g Ca/kg BW, and for light exercise 0.06 g Ca/kg BW (Table 3.3).

Intestinal absorption

The lack of impact of dietary Ca on the efficiency of P absorption in the horse is related to the fact that Ca and P are absorbed from different regions of the intestine (Figure 1.5), but excessive intakes of Ca increase the faecal loss of P and can depress the absorption of Mg, Mn and Fe. Around 50–80% of dietary Ca and 45–60% of Mg are absorbed in the small intestine (Meyer *et al.* 1982c), whereas there is a net secretion of these elements into the large intestine.

True Ca and P digestibilities are reported to decline from 71% to 42% and from 52% to 6%, respectively, between 6 and 24 months of age (Cymbaluk *et al.* 1989b). Aged horses may be even less efficient in the absorption of Ca. Meyer's group demonstrated that the site of P absorption varies with the composition of the diet. No P is absorbed from roughage in the upper small intestine, whereas some is absorbed in the distal small intestine, especially in those fed only concentrates. Large amounts of phosphate secreted into the caecum and ventral colon probably act as a buffer to VFAs produced there, and the dorsal and small colon are the major sites of absorption and reabsorption of phosphate (Figure 1.5).

Availability

Calcium carbonate, sulphate and oxide are common inorganic forms of calcium. The net availability of Ca in a variety of feeds lies between 45 and 70%, except where significant amounts of oxalates are present. Calcium–amino acid proteinate and calcium carbonate are absorbed to a similar extent (Highfill *et al.* 2005). Apparent Ca digestibility from alfalfa is greater than that from several grass hays (Cuddeford *et al.* 1990; Sturgeon *et al.* 2000). Schryver *et al.* (1987a) reported that true Ca digestibility increased from 51 to 69% and P absorption from 28 to 40% as the dietary content of NaCl was increased from 1 to 5%.

The dietary level of phosphate influences Ca absorption. When dietary P, as inorganic phosphate, was raised from 2 to 12 g/kg, Ca absorption was decreased by more than 50% in young ponies receiving a diet otherwise adequate in Ca (4 g/kg diet) (Schryver *et al.* 1971a). Dicalcium phosphate-P or bone flour-P is digested to 45–50%. Rock phosphate and metaphosphates are poor sources of P and Ca. Phosphorus in salts of phytic acid, the predominant source in cereal and legume seeds, is only 35% available, despite the presence of large numbers of intestinal bacteria secreting phytase. Phytate-P constitutes at least 75% of the total P in wheat grain and 54–82% of the P in beans. The addition of **phytase**, derived from *Aspergillus niger*, to pig diets increased phytate digestion. The response of adult horses to phytase has been disappointing, with diets containing 1.8–3.0 g P/kg from natural sources (Hainze *et al.* 2004; Morris-Stoker *et al.* 2001; Patterson *et al.* 2002), but it has improved apparent Ca digestibility from 26.4 to 42.4% (van Doorn *et al.* 2004a). Large amounts of dietary vitamin D can increase the utilization of phytin-P, but as these amounts border the toxic, they cannot be recommended. Phytin-P use can be improved by supplementation with yeast culture (see 'Probiotics', Chapter 5). Doorn *et al.* (2004b) showed that P absorption from a high-P diet (0.125 g P/kg BW) decreased from 25% in diets containing 0.148 Ca/kg BW daily to 11 and 13%

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with diets containing, respectively, 0.316 and 0.535 g Ca/kg BW daily.

Excessive dietary P, feeds rich in oxalates, or a low dietary Ca : P ratio, (Luthersson *et al.* 2005) can cause NSHP. Most forages have satisfactory ratios, although orchard grass (Cocksfoot, *Dactylis glomerata*) can have an inverted ratio. A fat- and fibre-based diet cf. a starch- and sugar-based diet produced a lower mineral content of the third metacarpus of growing foals (Hoffman *et al.* 1999a). This effect was attributed to a nutrient–endocrine interaction rather than fat and fibre causing reduced availability of Ca (Hoffman *et al.* 2001b).

Oxalates and other dietary factors affecting Ca absorption

The bioavailability, or true digestibility, of dietary Ca varies considerably. The principal factors controlling bioavailability are:

- amount of dietary Ca: true digestibility is 0.7 at requirement intake, cf. 0.46 at several times the requirement;
- amount of dietary P: 10 g P added/kg diet containing 4 g Ca/kg reduced true Ca digestibility from 0.68 to 0.43;
- vitamin D status: of less significance for absorption in the horse than in some other domestic species;
- dietary phytates and oxalates: phytates and oxalates bind Ca and so reduce Ca availability; Ca : oxalate <0.5 : 1 causes NSHP; implicated grasses rich in oxalates include: napier, guineagrass, buffel (*Cenchrus ciliaris)*, pangola (*Digitaria decumbens)*, green panic, paragrass, kikuyu (*Pennisetum clandestinum*), setaria (*Setaria sphacelata)* and probably some species of millet grass; lucerne (alfalfa) contains oxalic acid, see below; and
- age of animal: bioavailability declines slightly with age, but the relationship is not pronounced in horses.

Oxalic acid binds divalent cations, such as Ca, in an unavailable form. In contrast, rumen microorganisms degrade oxalic acid so that plasma Ca concentration is unaffected and renal function remains unimpaired in sheep and goats (Duncan *et al.* 1997). Many tropical and subtropical grasses are rich in oxalates and horses grazing pastures, or given hay, containing an abundance of these species become Ca-deficient with osteoporosis and lameness (see also Chapter 10). The bioavailability of Ca, nevertheless, differs among plant sources containing oxalates. Lucerne contains oxalates, but its Ca has a high feeding value, about three-quarters of the Ca is absorbed, although its Ca content relative to that of oxalic acid is much higher than that in the grasses listed above. Lucerne Ca is better utilized than that from timothy hay, not a significant source of oxalic acid. The feeding of Ca sources with a low bioavailability to laboratory animals compromises both the quantity and quality of bone, whereas inadequate quantities of highly available Ca reduce only the quantity of bone.

Summary of the Ca and P requirements for growth, work and reproduction in the mare (Tables 3.3 and 3.4)

The maintenance requirement for the major minerals Ca and P is that necessary to balance losses in the faeces and urine and unspecified 'dermal losses'. There is an additional need for growth and, in the breeding mare, for mineralization of the foetal skeleton and lactation. Each kg of lean body tissue in the horse contains about 20 g Ca and 10 g P.

Foals deposit approximately 8 g P/kg BW gain (Schryver *et al.* 1974b) with an absorption efficiency of 0.45. The amounts required in the diet to allow for maintenance and growth are shown in Tables 3.3 and 3.4. Stephens *et al.* (2004) reported that trained horses needed at least 66 mg P/kg BW daily a value in agreement with Nolan *et al.* (2001) and 32% more than the value given by NRC (1989). However, these reports related to young horses that were still growing and NRC (2007) has proposed a range of requirements depending on work load (Table 3.3).

The Ca : P ratio of mare's milk ranges between 1.8 and 2.5 : 1, but contains on average 900 mg Ca and 350 mg P/kg (see Figure 7.3). A 500 kg mare can produce a total of 2000 kg milk in a lactation extending over 5 to 6 months – a total lactation deficit of 1.8 kg Ca and 0.7 kg P derived from skeletal reserves and feed. (For milk synthesis, dietary Ca and P daily requirements, with average availabilities of 50% and 35%, are respectively, 10 g Ca and 5.5 g P, to balance that secreted daily). Limestone flour and dicalcium phosphate are reliable sources of Ca and the latter also of P. A requirement for 10 g Ca is met by 28 g limestone or 40 g dicalcium phosphate, which also meets the P needs entirely.

NRC (2007) concluded that the Ca requirement for late gestation of the pregnant mare should allow for endogenous losses of 20 mg Ca/kg BW and a foetal deposition rate of 15.9 mg Ca/kg BW of mare, both with an absorption rate of 50%. Using these estimates the Ca requirement during months 9, 10 and 11 of pregnancy was calculated as 0.04 g Ca/kg BW + 0.032 g Ca/kg BW. During months 7 and 8 of gestation, foetal deposition is equivalent to 8 mg Ca/kg BW (NRC 2007). Likewise, NRC (2007) estimates the Ca requirements of the lactating mare to be:

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(1) Foaling to 3 months = 0.04 g Ca/kg BW + 2.4 g Ca × 0.032 × BW, kg, where the Ca content of milk is 1.2 g/kg, with a 50% absorption efficiency and milk production from foaling to 3 months is 0.032 kg/kg BW;
(2) 4–5 months = 0.04 g Ca/kg BW + 1.6 g Ca × 0.026 × BW, kg, where the Ca content of milk is 0.8 g/kg and milk production is 0.026 kg/kg BW after 4 months and 0.020 kg/kg BW after 5 months;
(3) after 5 months = 0.04 g Ca/kg BW + 1.6 g Ca × 0.020 × BW, kg.

The equivalent P requirements for the products of conception during months 9, 10 and 11 of pregnancy have been determined by Drepper *et al.* (1982) to be 7, 12 and 6.7 mg/kg BW, respectively, of the mare daily (mean 8.6 mg/kg BW). Added to this are the daily endogenous losses of 10 mg/kg BW. The equivalent values for months 7 and 8 are 4.3 mg P/kg BW daily for products of conception plus 10 mg/kg BW to meet endogenous losses. An assumed absorption efficiency of 0.35 was adopted. During months 7 and 8 the daily needs would therefore be ((10 mg/0.35) + (4.3 mg/0.35)) per kg BW compared with ((10 mg/0.35) + (8.6 mg/0.35)) per kg BW for months 9, 10 and 11 (NRC 2007; Table 3.3).

The P concentration of mare's milk ranges from 0.75 g/kg of fluid milk during early lactation to 0.50 g/kg in late lactation. An absorption efficiency of 0.45 is assumed for the higher quality feed of lactation, with an endogenous loss of 10 mg/kg BW and daily milk production as given above. The daily needs of the lactating mare in months 0–3 would therefore be: (10 mg/0.45 per kg BW) + (0.032 g/kg BW) (0.75 g/0.45). In months 4–5 of lactation with a milk P content of 0.50 g P/kg the daily need would be 0.051 g P/kg BW. After 5 months, with milk containing 0.5 g P/kg, the daily needs will be (0.022 g/kg BW) + (0.020 g/kg BW)(1.11 g) (NRC 2007, Table 3.3).

Magnesium (Mg)

Mg constitutes approximately 0.05% of the body mass, 60% of which is in the skeleton and 30% in muscle (Grace 1999b). Bone ash contains 8 g Mg/kg in addition to 360 g Ca/kg and 170 g P/kg. Mg is an important ion in the blood, forming an essential element of intercellular and intracellular fluids. It is a cofactor of many enzyme systems and also participates in muscle contractions. Berlin *et al.* (2009) established reference intervals for ionized and total Mg and Ca concentrations in whole blood, plasma and serum of adult, neonatal foals, and pregnant and lactating mares. A rarely observed frank dietary deficiency of Mg leads to hypomagnesaemia associated with loss of appetite, nervousness, sweating, muscular tremors, ataxia, with the potential for collapse and death, rapid breathing (hyperpnoea), convulsions, heart and skeletal muscle degeneration and, in chronic cases, mineralization of the pulmonary artery and aorta, caused by focal deposition of Ca and P salts. Normal blood serum values are given in Table 3.1.

There is a small net absorption of Mg from the large intestine, but the majority of net absorption occurs from the lower half of the small intestine. The endogenous (obligatory loss) of Mg secreted into the intestinal tract amounts to about 1.8 mg/kg BW daily; a further obligatory loss of about 2.8 mg/kg BW occurs in the urine, although Pagan (1994) reported endogenous Mg excretion of 2.2 mg/kg BW daily. The maintenance requirement to offset these losses is 10 mg/kg BW daily, or 0.5–0.6 g/kg diet.

Homeostasis is achieved by a balance between gut absorption and renal excretion. Adrenal, thyroid and parathyroid hormones influence status, although plasma [Mg^{2+}] has a less potent effect on PTH than does [Ca^{2+}]. PTH causes an increase in plasma Mg by increasing absorption from intestines and renal tubules and resorption from bone (PTH also requires Mg ions for activation of adenylate cyclase in bone and kidney). Aldosterone secretion (see Sodium, this chapter) causes a lowering of plasma Mg and an increase in urinary Mg excretion. Some diets do not meet the horse's Mg need without supplementation.

The common inorganic supplementary sources are magnesium oxide (calcined magnesite), magnesium sulphate and magnesium carbonate – all up to 70% absorbed in the horse, although oxides from different countries of origin have differing availabilities. Generally, therefore, Mg carbonate is a more reliable source. Mg absorption does not seem to be influenced by the presence of oxalate (McKenzie *et al.* 1981) and neither supplemental phytase, nor the restricted range of dietary Ca (0.148–0.535 g Ca/kg BW daily) affects absorption efficiency of Mg (van Doorn *et al.* 2004a, 2004b). Excess dietary P decreases Mg absorption (Kapusniak *et al.* 1988), but apparent Mg digestibility did not seem to be affected at a normal rate of P intake above requirements (van Doorn *et al.* 2004b). Mg of vegetable origin, naturally present in feed, is available in proportions ranging from 45% to 60%, the more digestible sources being milk and lucerne. Absorption of Mg from high-temperature-dried lucerne is greater than it is from timothy hay, according to evidence from Edinburgh (see also 'Oxalates and other dietary factors affecting Ca absorption' this chapter). Sugar beet pulp and

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Table 3.5 Proposed daily requirements of Mg (g). (Compare Table 3.3)

	Mature weight (kg)	
	400	500
Adult		
Rest	5.6	7.0
Medium	6.5	8.0
Last 90 days of gestation	6.5	8.0
Peak lactation	6.6	8.1
Juvenile: age (months)		
3	4.2–5.5	4.8–6.8
6	4.0–5.2	4.4–6.3
12	4.2–5.3	4.5–6.5
18	4.5–5.6	5.0–7.0

beet molasses are reasonably good sources of digestible Mg containing about 2.8 g Mg/kg DM).

van Doorn *et al.* (2004b) indicated retention values in mature horses of 4.4 mg Mg/kg BW daily (2.2 g Mg/day per 500kg BW). Schryver *et al.* (1974b) reported that the requirements for growth ranged from 0.85 to 1.25 g Mg/day per kg BW gained. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and, assuming a growth rate of 1.00 kg/day on pasture, estimated a daily requirement for Mg, including endogenous losses, of 4.4 g/day. Table 3.5 gives the daily requirements of Mg for horses weighing 400 and 500 kg, and Table 3.3 gives the NRC (2007) estimates.

Using an absorption efficiency of 0.4 and an endogenous loss of 6 mg/kg BW daily NRC (2007) calculates that the horse's maintenance needs are 15 mg/kg BW daily. Applying the same efficiency of absorption and values determined by van Doorn *et al.* (2004b), the NRC (2007) calculates factorially the Mg requirement for endogenous losses and growth: (0.015 g/kg BW) + (1.25 g/kg ADG), in agreement with the estimate of Grace *et al.* (1998b) above.

For young racehorses Stephens *et al.* (2004) reported that an intake of 36 mg Mg/kg BW daily resulted in maximal retention of Mg in training.

NRC (2007) estimated the dietary Mg requirements for work, assuming endogenous losses of 6 mg/kg BW daily and absorption efficiency of 0.4. The additional needs for light, moderate and heavy work are given as, respectively, 1.6, 3.2 and 6.0 mg Mg/kg BW. Hence, the dietary requirements for light work are (0.015 g/kg BW) + (0.004 g/kg BW) and a similar procedure gives values for moderate and heavy work (Table 3.3).

The NRC (2007) estimates for the pregnant mare during months 7 and 8 of gestation are 6 mg Mg/kg BW to meet endogenous losses plus 0.08 mg Mg/kg BW for early foetal growth. Thus, with an absorption efficiency of 0.4, the Mg requirements for months 7 and 8 of gestation are: (0.015 g/kg BW) + (0.0002 g/kg BW), and for months 9, 10 and 11 when the foetal deposition rate is approximately 0.12 mg Mg/kg BW daily the needs can be similarly calculated (Table 3.3).

Grace *et al.* (1999a) reported the average Mg concentration of mare's milk between days 55–150 to be 47 mg/L, whereas Schryver *et al.* (1986) and NRC (1989) concluded that the Mg concentration of milk in early lactation was double that later (90 µg/g). Assuming a 0.4 efficiency of absorption and milk production at the rate of 32 g/kg BW daily, the dietary Mg requirement for milk production would be: (0.015 g/kg BW) + (0.032 g/kg BW)(0.09/0.4) from foaling to 3 months. From 4 to 5 months with milk production at the rate of 26 g/kg BW daily, and containing 0.09 g Mg/kg (NRC assumes the higher value) equivalent estimates are achieved. After 5 months a milk production rate of 20 g/kg BW daily, containing 0.045 g Mg/kg is used to yield a lower daily dietary requirement (NRC 2007, Table 3.3).

Potassium (K)

Potassium is involved in maintenance of the acid–base balance and osmotic pressure and is the most quantitatively important ion for neuromuscular excitability (Kronfeld 2001). A 500 kg horse contains in its body approximately 28,000 mEq K (Rose 1990), 75% of which is in the skeletal muscles (Meyer 1987, Johnson 1995); less than 1.5% of the total is present in the extracellular fluid (Rose 1990), 5% is in the skeleton, 5% in the blood and skin and 4.5% is present in the ingesta (Meyer 1987).

Potassium is primarily subject to precaecal absorption. Pagan (1994) estimated a true K digestibility of around 75%. Normally K intake greatly exceeds requirements and the kidneys are the primary route for the variable excretion of excess (Coenen 2005a).

NRC (2007) estimates an endogenous loss of 40 mg K/kg BW daily with an absorption efficiency of 0.8, yielding a daily dietary maintenance requirement of 0.05 g K/kg BW. For growth Schryver *et al.* (1974) estimated that 1.5 g K were deposited per kg BW gain and with a conservative absorption efficiency of 0.5, the needs for K are: (1.5 g/0.5 kg gain) + (0.04/0.8 kg BW) daily (Table 3.3).

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Table 3.6 Serum total concentrations (mmol/L) of electrolytes in TB foals (Sato *et al.* 1978).

Days from birth	Ca	P	Na	K	Cl	Mg
0	2.64	1.51	131.6	4.68	103.6	0.88
10	2.62	1.48	142.0	4.42	97.8	0.97
20	2.83	1.79	138.6	4.08	98.4	0.90
50	2.34	2.11	137.2	4.28	96.6	1.32*
90	2.50	1.74	140.0	3.96	99.8	—
120	2.23	1.63	136.4	4.12	101.4	—

*Thirty days of age.

Drepper *et al.* (1982) estimated that the products of conception require on average 1.36 mg K/kg BW of the mare during months 9–11 of gestation and assuming an absorption efficiency of 0.8 and endogenous losses of 40 mg K/kg BW the daily K requirements for months 9–11 of gestation are (0.04/0.8 kg BW) + (0.00136/0.8 kg BW), (NRC 2007, Table 3.3).

For lactation, NRC (2007) estimates a K requirement from foaling to 3 months with endogenous losses of 40 mg/kg BW, an absorption efficiency of 0.8 and milk production of 0.032 kg/kg BW containing 0.7 g K/kg milk with an absorption efficiency of 0.5: (0.04/0.8 kg BW) + (0.032 × 0.7 g/0.5 kg BW). During months 4 and 5 of lactation with an assumed milk production rate of 0.026 kg milk/kg BW, containing 0.4 g K/kg milk, the requirements are similarly calculated**.** After 5 months milk production is assumed to have decreased to 0.020 kg milk/kg BW containing 0.4 g K/kg (NRC 2007, Table 3.3)

Exercise

Diets normally contain excessive amounts of K. This could have accounted for the observation of Hess *et al.* (2005) that supplementation of an electrolyte mixture excluding potassium resulted in a similar completion rate in an 80-km endurance ride to that found when horses received potassium in the mixture. Sweat losses account for about 90% of the body weight loss of horses during exercise (Meyer 1990a). Coenen (2005a) estimated sweat to contain 1.4 g K/L so, assuming the lower absorption efficiency of 0.5, the K recommendation for exercise can be expressed as: maintenance (0.04 g/0.8 kg BW) + (1.4 g/0.5 kg BW lost during exercise). (NRC 2007 assumes sweat loss through light, moderate, heavy and very heavy exercise to be: 0.25, 0.5, 1.0 and 2.0% BW daily, respectively) ignoring the small amount of weight lost through defecation or urination (Hinchcliff 2005; NRC 2007, Table 3.3). Spontaneous changes in plasma [K^+] can therefore result from strenuous exercise; this will be discussed in Chapter 9.

Foals require perhaps as much as 150–200 mg K/kg BW, that is, 7 g K/kg diet. Young foals become deficient in K as a result of persistent diarrhoea and this in turn tends to precipitate acidosis. The normal range of serum, or plasma, K concentration is 2.4 to 5.6 mEq/L (or mmol/L) (Puls 1994; Table 3.6). The plasma concentration increases during episodes of acidosis, as intracellular red cell K (normal concentration 83–100 mmol/L; Muylle *et al.* 1984b) exchanges for H^+ ions, when cardiac arrhythmias can occur. Red cell K concentrations <81 mmol/L are accompanied by signs of skeletal muscle weakness (Frape 1984b). The means of assessing K status and the major causes of K depletion and their therapy are discussed in Chapter 11.

Deficiency

A dietary deficiency of K reduces appetite, depresses growth rate and decreases plasma K (**hypokalaemia**). In extreme deficiency, clinical muscular dystrophy and stiffness of the joints occurs. Hypokalaemia can occasionally result from persistent diarrhoea, or from excessive sodium bicarbonate administration. In K-depleted ponies, K losses in urine did not fall below 20 mmol/L (Meyer *et al.* 1986) and the K content of sweat remained invariable at about 27 mmol/L (cf. sodium). Blood plasma K decreased from 3.5 to 2.3 mmol/L, whereas red cell K changed little. However, the K depletion of the skeleton was as much as 60%, whereas losses in muscles, vital organs and GI contents were only 9%, 15% and 7%, respectively. Food and water intake decreased, the ponies were more excitable and exercise exhaustion occurred sooner.

Excess

The maximum tolerable concentration of dietary K has not been determined where there is free access to water, as

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excess is excreted readily. Hyperkalaemia induced by parenteral administration would be expected to cause cardiac arrest, although this does not occur when hyperkalaemia is induced during exercise.

Hyperkalaemic periodic paralysis (see also Chapter 11)

A syndrome of episodic weakness in horses, accompanied by elevated serum K concentrations – hyperkalaemic periodic paralysis (HPP) – has been described in horses (Naylor *et al.* 1993). It originated in descendants of the American Quarter Horse, Impressive. The genetic cause has been identified and progress is being made to eliminate the disease (Meyer *et al.* 1999). HPP is not caused by a dietary K deficiency or excess. It is accompanied by myotonia, facial muscle spasm and fasciculations and recumbency. Tracheotomy is required if there is severe dyspnoea. Most episodes resolve spontaneously in 15–120 min, but may require parenteral administration of Ca gluconate, Na bicarbonate, dextrose, insulin and subsequently dietary management. This situation reflects the exchange of intracellular K with H in acidosis mentioned above.

Sources

Lush pastures contain large amounts of K in the DM. Cereals are relatively poor sources of K, but hay contains 15–25 g K/kg. Most diets should contain ample K if at least one-third is in the form of good-quality roughage. Animals in heavy work generally consume more cereals, lowering dietary K when losses in sweat would normally be increasing.

Sodium (Na)

Sodium (Na; see also electrolyte supplementation, Chapter 9) is the principal determinant of the osmolarity of extracellular fluid and consequently of the volume of that fluid. Chloride concentration in the extracellular fluid is directly related to that of Na. Na is critical for the normal function of the central nervous system, generation of action potentials in excitable tissues and transport of many substances, such as glucose, across cell membranes. It is the major extracellular cation (138–140 mmol/L) and the major electrolyte for the maintenance of acid–base balance and osmotic regulation of body fluids (Rose 1990). The skeleton contains 51.1% of the Na in the body, ingesta 12.4%, blood and muscle 10.8%, the skin 8.5% and organs 2.1% (Meyer 1987). Chronic Na depletion results in decreased cutaneous turgor, a tendency to lick sweaty objects, decreased water intake and a slow rate of eating that eventually ceases, a craving for large amounts of sodium chloride, and muscular and nervous dysfunction (muscular tremor, gait and chewing incoordination) (Meyer *et al.* 1984).

Na excretion via the kidney is controlled by the renin–angiotensin–aldosterone system. Hyponatraemia can occur from reduced intake of Na and it causes aldosterone secretion; however, Na deficits can also occur through excessive losses from the GI or urinary tract relative to water loss. These can indicate intestinal obstruction, enterocolitis or renal failure. Excessive sweating normally causes hypernatraemia, with haemoconcentration. In some other species, increased plasma levels of aldosterone result in increased urinary Mg excretion, although what occurs in the horse is unclear.

Usually, about 95% Na is reabsorbed by the large intestine, but in Na deficiency, reabsorption reaches 99% and renal Na losses are reduced, thus conserving tissue content (Meyer *et al.* 1982b). The Na content of the sweat of working horses is, however, only slightly decreased, being partly replaced by K. In advanced Na depletion, plasma Na and chloride concentrations fall to 120 and 70 mmol/L, respectively, with an increase in plasma K of up to 5.5 mmol/L and a decrease in total body water, largely through increased DM of the GI tract contents.

Although pasture grass contains as much as 18 times more K than Na, supplementary Na in the form of common salt for grazing stock is normally unnecessary. Forages are a richer source of Na than cereals and within normal ranges the one element tends to inhibit the loss of the other in the urine by the action of aldosterone on the kidney tubules, thus conserving the body's resources of Na. Diets providing 2–4 g Na/kg should adequately meet the requirement for Na, except during periods of excessive sweating in very hot weather, or as a result of diarrhoea, but repeated oral administration of an electrolyte solution is associated with exacerbation of gastric ulcers (Holbrook *et al.* 2005).

The Na concentration of natural feedstuffs for horses is often lower than 0.1%, but the horse can adapt to a low dietary Na concentration. Meyer *et al.* (1984) reported that a negative Na balance could occur initially, and transiently, in non-exercised horses and ponies after a restriction in Na intake to 5 mg/kg BW daily; ultimately they could be in a positive Na balance while receiving only 1.6 mg Na/kg BW daily. Ingesta in the large intestine are a reservoir for water, sodium, chloride and potassium when exercising (Meyer 1996a,b), probably reducing the risk of hyponatraemia, despite sweat losses; but sweat contains 2.8 g Na/L (Coenen 2005b) and Na losses in sweat during endurance exercise can amount to 2000–2500 mmol (34.5–46 g) (Butudom *et al.* 2002). About 90%

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of Na is absorbed and the dietary need is 3.1 g Na/kg BW lost during exercise (8 g sodium chloride) to maintain Na in balance. Endogenous losses in the adult are 15–20 mg/kg BW daily (assume 18 mg/kg) (Meyer *et al.* 1984; Schryver *et al.* 1987b). The maintenance requirement is then 0.02 g Na/kg BW daily (NRC 2007) and the dietary requirement of exercising horses is: (0.02 g Na/kg BW) + (3.1 g/kg BW loss) (Table 3.3). However, Na excretion is reduced by as much as 73% during the early stages of training (McKeever *et al.* 2002). Optimal dietary Na concentration for moderate to heavy work is 3.6 g/kg DM (Jarridge & Martin-Rosset 1981).

Grace *et al.* (1999b) determined from whole body analysis that foals deposited 0.85 g Na/day with an absorption efficiency of 0.8. This indicates a daily Na requirement for a growing horse of (0.02 g/kg BW) + (1.0 g/kg gain/day). Based upon the evidence of Drepper *et al.* (1982) the Na requirement for pregnant mares during months 9–11 of gestation has been estimated by NRC (2007) to meet endogenous losses plus foetal requirements as: (0.02 g/kg BW) + (0.002 g/kg BW). Thus, the Na requirement for maintenance and growth and for late gestation are 1.6–1.8 g/kg dietary DM. Based on the evidence of Schryver *et al.* (1986b) and Grace *et al.* (1999b) the NRC (2007) estimates milk to contain 126 mg Na/kg during the first 3 months of lactation. Assuming an absorption efficiency of 90%, the NRC (2007) estimates the dietary requirement per kg milk for the first 3 months of lactation to be 0.17 g Na, compared with 0.14 g Na for the 4th month to the end. Thus, from foaling to 3 months a mare producing 32 g milk per kg BW has a dietary requirement of: (0.02 g/kg BW) + (0.032 × 0.17 kg BW) to meet endogenous losses. From 4 to 5 months of lactation when milk production is at the rate of 26 g/kg BW daily, the dietary Na requirement is: (0.02 g/kg BW) + (0.026 × 0.14 kg BW). After 5 months the Na requirement is: (0.02 g/kg BW) + (0.020 × 0.14 kg BW) (Table 3.3).

Chloride (Cl)

Where the requirements for Na are met as common salt (NaCl), it is unlikely that a deficiency of chloride will occur. Chloride is an important extracellular anion involved in acid:base balance and osmotic regulation. It is an essential component of bile and of hydrochloric acid in gastric secretions. The chloride concentrations in common equine feedstuffs range from approximately 0.05% in cereal grains to 3% in cane molasses. Schryver *et al.* (1987) reported that 100% of chloride was absorbed, regardless of the dietary concentration of NaCl. Cl deficiency is unlikely unless horses are administered large amounts of sodium bicarbonate, when metabolic alkalosis occurs with a compensatory increase in plasma bicarbonate (Tasker 1980; Coenen 1991). Clinical signs might include those reported in ruminants, including decreased food intake, weight loss, muscle weakness, decreased milk production, dehydration, constipation and depraved appetite. The major source of loss, particularly in hot weather, is sweat, where even at moderate rates of work horses lose 60 g Cl/day.

Coenen (1999) proposed using 3 mg Cl/kg BW to account for faecal endogenous losses, 2 mg Cl/kg BW for urinary endogenous losses and 1 mg Cl/kg BW to account for cutaneous endogenous losses, totalling 6 mg Cl/kg BW daily. Another 14 mg Cl/kg BW daily are required to replace losses through sweat, indicating that a minimum of 20 mg Cl/kg BW daily is needed for maintenance with an absorption efficiency of 100%. However, as 80 mg Cl/kg BW daily were required to prevent changes in acid:base balance and hypochloraemia, this amount is given as the minimum daily maintenance requirement (NRC 2007). Coenen (1999) suggested that the Cl requirements for growth of foals was maintenance plus 13 mg, 5 mg and 2.5 mg Cl/kg BW daily up to 6, 6–12 and 12–24 months, respectively. Hence NRC (2007) estimates the daily requirements for Cl by growing foals up to 6 months is: (0.08 g/kg BW) + (0.013 g/kg BW). By equivalent calculations the requirements are 0.085 g Cl/kg BW daily and 0.0825 g Cl/kg BW from 6 to 12 and from 12 to 24 months, respectively. Pregnancy requirements for Cl equal maintenance plus 2 mg Cl/kg BW daily, or 0.082 g Cl/kg BW. Lactation requirements for Cl equal maintenance plus 11 mg Cl/kg BW daily, or 0.091 g Cl/kg BW (Coenen 1999; NRC 2007).

Coenen (1999, 2005b) suggested Cl requirement for exercising horses equals maintenance plus 5.3 g Cl/L sweat, so that in total the requirement is (0.08 g/kg BW) + (5.3 g/kg loss in BW during exercise). Details of the requirements are given in Table 3.7 (Coenen 1999) and NRC (2007) estimates in Table 3.3.

Sulphur (S)

Sulphur is a component of sulphur-containing amino acids, several water-soluble vitamins, heparin, and chondroitin sulphate, and constitutes 1.5 g/kg BW. Horses must meet their requirements for S from organic forms of the element, although 10–15% of plant S is inorganic. Most is present in the amino acids of plant proteins and some inorganic S will be utilised by gut microorganisms in protein synthesis. The NRC (2007) recommends 1.5 g S/kg dietary DM.

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Table 3.7 Chloride recommendations (mg/kg BW/day) (Coenen 1999).

Endogenous losses (faecal + renal + cutaneous)	3 + 2 + 1
Cutaneous maintenance losses by perspiration	14
Maintenance, minimum	20
Maintenance, recommendation	80
Maintenance exercise, 5, 20 or 50 g sweat/kg BW	108, 190 or 355
Maintenance pregnancy up to 90 days ante partum	80
Last 90 days of pregnancy	82
Maintenance lactation, 3rd month	89–93
Maintenance growth until 6th month	93
Maintenance growth, 6th to 12th months	85

TRACE ELEMENTS

Most stabled horses now receive supplements containing variable quantities of trace elements, and the horse seems able to cope with some measure of abnormal intake without showing clinical signs of toxicity or deficiency. Those trace elements of prime importance in the diet of horses are discussed here. The great variety of geological strata underlying UK soils yields grazing areas that cause clinically recognizable signs of specific deficiencies in cattle and sheep. There is biochemical evidence to show that horses and ponies in these areas similarly reflect their nutritional environment. Tables 3.8 and 3.9 give average serum values for some trace elements. Abnormalities in leg growth and development of foals and yearlings have been reported to be associated with dietary deficiencies of copper (Cu), manganese (Mn) and selenium (Se), and toxicities of iodine (I) and lead (Pb).

The extent to which pasture plants extract trace metals from soil depends on the soil's pH and moisture content and the plant species. Effects are attributable to the root systems of plants, as legumes and many herbs have deeper roots than grasses. The levels of trace elements in herbage are clearly of importance, but the horse will consume soil while grazing, depending on the density of the herbage. In certain conditions cattle and sheep consume more than 10% of their daily intake of DM as soil.

Mixed supplements

Ott & Asquith (1995) measured the response of growing TB and Quarter Horses from 340 to 452 days of age to trace mineral supplements, when given concentrates to appetite and coastal Bermuda grass *(Cynodon dactylon)* hay at the rate of 1 kg/100 kg BW daily. The unsupplemented total diet contained 195 mg Fe, 36 mg Mn, 41 mg Zn and 4.8 mg Cu, each per kg DM. The supplements had no influence on growth and development, but mineral deposition in the third metacarpal was increased by the trace mineral mix, although not by Cu or by Cu plus Zn. The results suggested that the other trace minerals (Fe, Mn, Co or I) were more critical. Previous work by the same group (Ott & Asquith 1989) indicated that bone mineral deposition in yearlings was increased when a trace mineral mix containing Fe, Mn, Zn and Cu was added to a natural diet containing lower concentrations of these elements than those recommended by the National Research Council (NRC 1978).

Mineral content of horse hair

The mineral content of animal hair varies not only with mineral intake, but also with season, breed, age, hair colour and body condition, yet the assessment of trace element status by their determination in hair is of questionable value (Asano *et al.* 2005). Nickel (Ni) intake is apparently correlated with Ni in hair, and toxic intakes of Pb cause elevated hair Pb, but other heavy metals, at least at subtoxic intakes, are not. There is generally no simple relationship between minerals of nutritional importance and their concentration in horse hair.

Copper (Cu) and molybdenum (Mo)

Cu is essential for several Cu-dependent enzymes required in the synthesis and maintenance of elastic connective tissue, mobilisation of iron stores, preservation of the integrity of mitochondria, melanin synthesis and detoxification of superoxide. Hypocupraemia occurs widely in grazing cattle, attributed frequently to an excess of molybdenum (Mo) derived from the underlying Mo-rich strata, particularly Lias clays and marine black shales of Jurassic and Carboniferous ages. The high levels of Mo associated with relatively low Cu lead to Cu : Mo ratios in the herbage narrower than 6 : 1, causing Cu deficiency on the so-called teart pastures in these areas. Hypocupraemia can also occur, owing to low Cu levels *per se* in the soils and herbage.

The horse is not as susceptible to clinical signs of Cu deficiency as are ruminants, but signs have been described, such as erosion of the articular cartilage

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Table 3.8 Values for four trace elements (μmol/L) in normal serum or plasma and milk of mares (Smith *et al.* 1975; Blackmore & Brobst 1981; Cape & Hintz 1982; Schryver *et al.* 1986; Lawrence *et al.* 1987b; Bridges and Harris 1988; Saastamoinen *et al.* 1990; Anderson 1992; Suttle *et al.* 1996; Brommer & Sloet van Oldruitenborgh-Oosterbaan 2001 (see also Chapter 12 for Cu determinations).

	Blood	Milk		
	Serum or plasma	Partum	1–8 days	9–120 days
Cu				
TB, USA mainly stabled	24–35	16	10–12	4–6
TB	8–18			3[1]
TB, 7, 14, 28 days of age	6, 14, 25			
Quarter Horse	5–31			
Horse/pony, gestation[2]	16.3			
Horse/pony, lactation[2]	14.7			3.8
Horse/pony, yearlings	15.3–26.1			
Horse/pony, 2–3 years old	21.3			
Horse, adult	17			
Fe				
TB	28 ± 7			
Quarter Horse	28			
Arabian	23	24	17	12
Standardbred	30 ± 7			
Shetland	19			
Dutch Warmblood foal	38			
Zn				
TB, USA, stabled	—		46	60
TB, USA, pastured				30–46
TB, UK, pastured	17 ± 7	98	52	36
TB, UK, stabled	26 ± 8			
TB, 7, 14, 28 days of age	17, 12, 12			
Horse/pony, gestation[2]	5.8			
Horse/pony, lactation[2]	7.6			31.3
Horse/pony, yearlings	10.7, 12.5			
Horse/pony, 2–3 years old	10.1			
Pb				
TB and Standardbred:				
suckling to adult (mean)	0.8–1.9			
mixed breeding	7–8			

[1]Unpublished data of author.
[2](Saastamoinen *et al.* 1990). Finnhorse breed.

of joints, and anaemia and haemorrhage in parturient mares. Moreover, Mo and sulphate do not have the same impact on Cu status in horses as they have in cattle. Thiomolybdates, which bind Cu ($CuMoS_4$ being very insoluble), have not been detected in horses with dietary Mo concentrations of 10 mg/kg. Their formation depends on the presence of a rumen and its microbial activity, that is, an extensive microbial activity in a region of the GI tract proximal to absorption sites for Cu. Thus, Rieker *et al.* (1999, 2000) have shown that a concentrate

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Table 3.9 Normal blood characteristics of horses.[1] Ranges indicate approximately ±1 SD from mean.

Sample	Characteristic	Units per litre	Yearlings	Horses in training	Mares	Foals
Plasma	Albumin	g	27–28	34	27	25
Serum	AST	iu	140	150–400	140	70–120
Serum	CK	iu	43	50–70	43	53–57
Serum	SAP	iu	100–120	85–95	70–80	150–400
Serum	ALT	iu	1–6.7	1–6.7	1–6.7	NA
Serum	GGT	iu	18–30	20–30	18–19	13–16
Red cell	GSH-Px	u/mL[7]	15–25	15–25	—	—
Serum	LDH	iu	45–100	45–100	—	—
Serum	SDH	iu	0.8–1.2	0.8–1.2	—	—
Serum	Bilirubin, total[2]	µmol	34	34–39	26	38–55
Serum	Fasting glucose	mmol	3.5–5	3.5–5	3.5–5	3.1–4.2
Serum	T_4	nmol	5–39	5–35	3–56	10–150
Serum	T_3	nmol	1.5–2	1–2	0.9–1.4	2–7
Serum	Creatinine	mmol	140	170–185	140	150–190
Serum	Cu	µmol – stable	15.2[3]	12.4	15[4]	6–20[5]
Serum	Cu	µmol – grass	—	15.9	16–26	20[8]
Serum	I	µmol	—	—	0.6	0.8–0.85
Serum	Zn	µmol – stable	—	26	9–12	13
Serum	Zn	µmol – grass	—	17	9–12	—
Serum	Mo	µmol	—	0.31	—	—
Serum	Se	µmol	0.8–1.3	1.5[6]	0.8–1.2	0.6–1.1
Whole blood	Se	µmol	—	1.6[6]–3.0	0.8–1.1	0.5–0.7
Plasma	α-Tocopherol	mg	1–3	1.5–4.4	1.8–2.6	1.1–2.4
Plasma	α-Tocopherol	µmol	2.3–6.8	3.4–10	4.1–5.9	2.5–5.5
Plasma	Retinol	µg	180	180	150–300	150
Serum	25-Hydroxy vitamin D	µg	—	—	2.9–3.6	2.2–2.5
Whole blood	Cyanocobalamin	µg – stable	3.7–6.6	1.2–6.6	3.7–6.6	3.7–6.6
Whole blood	Cyanocobalamin	µg – grass	2.8–20	2.8–20	2.8–20	2.8–20
Serum	Folate	µg – stable	4.5–12	4.5–12	4.5–12	4.5–12
Serum	Folate	µg – grass	5.3–13.5	5.3–13.5	5.3–13.5	5.3–13.5
Whole blood	Thiamin	µg	28	30	33	24
Plasma	Ascorbic acid	mg	—	2.5–4.5	—	—

[1]Data are drawn mainly from TBs. See Table 3.1 for serum Ca, P, Mg, Na, K and Cl values.
[2]Mainly unconjugated.
[3]Ponies.
[4]Whole blood.
[5]Rising from 1 week of age to plateau at 4 weeks.
[6]Minimum adequate.
[7]One enzyme unit of GSH-Px activity = 1 µmol NADPH oxidized/min.
[8]Plasma.

Abbreviations: ALT, alanine amino-transferase; AST, aspartate amino-transferase; CK, creatine kinase; GGT, γ-glutamyl-transferase; GSH-Px, glutathione peroxidase; iu, international unit; LDH, lactate dehydrogenase; SAP, serum alkaline phosphatase; SDH, sorbitol dehydrogenase; T_3, triiodothyronine; T_4, thyroxine.

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containing 5–20 mg Mo/kg given to geldings with grass hay (50 : 50) had no adverse effect on the absorption and retention of Cu.

Measurement of Cu status and dietary requirements

Excessive intakes of Cu are hazardous to sheep and, to a lesser extent, to cattle. Ruminants differ from nonruminants in the propensity of the former to store Cu in their livers at low dietary Cu intakes. Very high dietary Cu levels (<4 mmol/kg DM) are required to increase liver Cu of ponies substantially (referred to by Suttle *et al.* 1996), while any increase above the minimum dietary requirement seems to lead only to some additional hepatic Cu storage. For this reason horses are less subject to intoxication by dietary Cu. An excess of dietary Cu of up to 791 mg/kg diet in ponies for six months (Smith *et al.* 1975) led to liver Cu concentrations of over 4000 mg/kg DM, apparently causing no liver damage and no adverse effect on fertility or other characteristic.

There is a curvilinear relationship in ruminants between liver Cu and plasma Cu concentrations. In ruminant plasma, or serum, Cu values plateau and, excepting toxic crises, they rarely exceed 16 µmol/L, even with the liver at >800 µmol/kg fresh weight. In 44 horses, Suttle *et al.* (1996) found a linear relationship:

$$Y = 7.00811 + 0.086019X \left(R^2, 0.168\right)$$

where Y is µmol Cu/L serum, X is µmol Cu/kg fresh liver (values were restricted to liver Cu <190 and serum Cu <29) and R^2 is the proportion of the variation, or sum of squares, attributable to the model (in this case a value of 0.168, i.e. 16.8% of the variation, is accounted for by the relationship described). In 48 horses, Suttle *et al.* (1996) found mean Cu concentrations of:

- 16.7 µmol/L serum, with 13.5 and 19.5 µmol/L as lower and upper quartile values; (1 mmol ≡ 63570 µg)
- 113.7 µmol/kg fresh liver;
- 172.5 µmol/kg feed (11 mg/kg) with 61.1 and 233.8 µmol/kg as lower and upper quartile values.

The animal has a need to maintain a normal cellular Cu concentration, measurable as hepatic concentration. In the horse, adequate liver levels would be achieved by a dietary concentration of 20 mg/kg DM and these liver and dietary concentrations correspond to 16 µmol/L serum (Suttle *et al.* 1996). Suttle proposes 16 µmol/L as a threshold value to distinguish the normal from subnormal serum values and a serum level of 11.5 µmol/L to distinguish deficient from marginal liver Cu concentrations of 52.5 µmol/kg fresh weight. Unfortunately, as Suttle *et al.* point out, high serum Cu values occur following inflammation, infection or vaccination and liver samples are then preferable, but not readily available. (Plasma Cu is an unreliable guide to status, as about 70% of the circulating Cu is present in the form of caeruloplasmin [EC 1.16.3.1], an acute phase protein.) Therefore an alternative cellular source is preferred as a means of assessing Cu status.

Table 3.10 Range in leucocyte Cu and Zn contents in seven Shetland ponies receiving poor hay only, before and after supplementation with Cu and Zn for 50 days (author's unpublished data).

	Cu (µg 10^{-9}) in leucocyte cells	Zn (µg 10^{-9}) in leucocyte cells
Before supplementation	0.11–0.18	2.57–6.25
After supplementation	0.40–2.86	2.57–10.57

Cu deficiency decreases the activity of Cu–Zn superoxide dismutase (EC 1.15.1.1) of leucocytes and platelets. The measurement of this activity is not easy. The author's colleagues (Williams *et al.* 1995) have routinely used mononucleated leucocyte and platelet Cu contents and platelet cytochrome c oxidase (EC 1.9.3.1) activities as guides to status. Mononuclear cell Cu shows good promise (Table 3.10). The Cu contents will be present mainly as enzyme cofactor Cu (see Chapter 12).

Cu is transferred to the foetal liver, which, like the neonatal liver, contains more Cu than that of older foals, or of their dams. The low serum Cu concentration of neonatal foals does not rise to normal adult levels until about 28 days of age (Kavazis *et al.* 2001). In New Zealand it was shown that when the dietary Cu of the grazing pregnant mare was increased from 6 mg to 30 mg Cu/kg DM throughout the last 4 to 5 months of gestation, the Cu content of the foal's liver at birth was increased by two-thirds. Pearce *et al.* (1998a) studied grazing mares and their foals during the second half of gestation, raised on a pasture that contained 4.4–8.6 mg Cu/kg DM. Half the mares were supplemented with 0.5 mg Cu/kg BW daily. The foals from control and supplemented mares were either retained as controls or supplemented at 0.2 mg/kg rising to 0.5 mg/kg BW at 49 days of age. Plasma Cu and

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caeruloplasmin concentrations in the foals were unaffected, whereas liver Cu concentration increased, with supplementation. Mare supplementation alone (Pearce *et al.* 1998b) decreased radiographic indices of physitis in the distal third metatarsal bone and the prevalence of articular cartilage lesions at 150 days of age, without an effect on clinical DOD.

Mare's milk provides less Cu than the suckling foal requires daily (Cu content of milk is about 3 µmol/L or 0.19 mg/L), whereas pasture grass and grass hay contain 4–9 and 10–12 mg Cu/kg DM respectively, cf. cereal grains 1 mg/kg and cane molasses 80 mg/kg. Therefore, with the object of increasing the Cu and Zn contents of the milk, and decreasing the risk of osteochondritis dissecans (OCD), Baucus *et al.* (1987) doubled the Cu and Zn contents of the mare's diet to 53 mg Zn/kg and 12 mg Cu/kg at parturition without effect on milk Cu or Zn contents.

The suckling foal requires a source of Cu to tide it over the period during which it eats little grass and dry food. These needs are normally met when the foetal liver stores about 300 µg Cu/g liver DM, or more. The stores should range between 300 and 600 µg/g when the pregnant mare has received adequate Cu in her diet.

Natural feed sources of Cu have a digestibility ranging from 10 to 50%. Hoyt *et al.* (1995b) found that miniature horses absorbed 42.2–50.7% of the total Cu from diets containing 12 mg/kg. Asquith (1994) reported that serum Cu and Zn concentrations in foals were increased only when their mothers received chelated trace minerals, rather than inorganic sources, during pregnancy. The absorption of Cu from the oxide, sulphate and an organic chelate is reported to be 5.3, 6.6 and 2.8%, respectively, indicating a greater apparent digestibility (Wagner *et al.* 2005; NRC 2007) and retention of Cu from the sulphate than from a Cu-proteinate (Baker *et al.* 2005). By contrast Miller *et al.* (2003) found greater apparent Cu digestibility and retention from the proteinate than from sulphate – differences that might be explained by dietary variation, as Cu interacts with Mo, S, Zn, Se, Ag, Cd, Fe, and Pb (Underwood 1981). A secondary Cu deficiency was induced in weanling foals given a ration containing 7.7 mg Cu/kg DM and either 1000 or 2000 mg Zn/kg DM, but not when given 29.1 or 250 mg Zn/kg DM (Bridges & Moffitt 1990).

The obligatory losses of Cu in the faeces of ponies amount to about 3.5 mg/100 kg BW daily in the presence of low levels of dietary Mo (Cymbaluk *et al.* 1981a,b). Hudson *et al.* (2001) reported endogenous losses to be 2.9 mg Cu/100 kg BW and 3.8 mg Cu/100 kg BW daily and true digestibilities of 41.8 and 54.5% for sedentary and exercising horses, respectively. Pagan (1994) estimated endogenous losses to amount to 6.9 mg Cu/100 kg BW with a true digestibility of 40%.

In order to allow for adverse interactions with other trace elements and to maximize iron (Fe) retention, a dietary intake of 15–20 mg/kg dry feed is recommended for growing horses. Foals need 25–30 mg/kg feed to reduce, but not eliminate, the risk of cartilage erosion. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and, assuming a growth rate of 1.0 kg/day on pasture, estimated the daily requirement for Cu to be 26 mg/day. NRC (2007) proposes a dietary need of 0.172 mg Cu/kg BW daily, indicating a minimum dietary concentration of 8.6 mg Cu/kg DM of feed; but where absorption efficiency is 35% the requirement for maintenance is 0.2 mg Cu/kg BW daily, or 10 mg Cu/kg DM feed. For growth, Grace *et al.* (1999b) determined that 1 mg Cu was deposited daily per kg of BW gain with an absorption efficiency 30% resulting in a requirement of 0.017 mg Cu/kg BW daily in addition to maintenance. The daily dietary requirement is therefore 0.217 mg Cu/kg BW for growing horses, or 8.7 mg Cu/kg DM feed (NRC 2007). NRC (2007) has retained its previous recommendation for heavy exercise, 0.25 mg Cu/kg BW daily (Table 3.11). Gee *et al.* (2006) reported that giving copper by injection, as calcium copper edentate, was an unsuitable method of copper supplementation.

Cu and cartilage formation

(See also 'Growth of foals', Chapter 8). The bones and cartilage of Cu-deficient animals show increased defects and fragility, and contain an enhanced proportion of soluble collagen. This solubility is caused by a reduction in the cross-linking within the molecules of collagen and elastin that require Cu as the cofactor of lysyl oxidase (EC 2.3.2.3). Osteoblast function is inhibited by Cu deficiency, whereas osteoclast function is unaffected. Excess dietary Cu can interfere with bone metabolism causing inhibition of collagen synthesis and a loss of bone density. Firth (1998) indicated that Cu supplementation of the foal can reduce bone mineral density of the radius, third metacarpal and third carpal and neither organic nor inorganic supplements of Cu and Zn influenced blood carboxy-terminal propeptide of type I procollagen, or bone density, in exercised yearling geldings given a basal diet meeting NRC (1989) recommendations (Baker *et al.* 2003). Moreover, amongst 629 Hanoverian warmblood foals in Germany, 222 presented with osteochondrotic lesions not correlated with growth rate or with protein, Ca, P, Cu, Zn or Se intakes (Coenen *et al.* 2003a; Vervuert *et al.* 2003a;

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Table 3.11 Trace element requirements of growing, working and breeding horses, based upon NRC (2007) recommendations (mg/kg BW per day, except for ADG). N.B. These requirements assume positive linearity with BW.

	Copper	Zinc	Manganese	Iron	Iodine	Selenium	Cobalt
Maintenance, foals*	0.22	0.82	0.82	0.8	0.007	0.002	0.001
Growth (mg per kg ADG/d)[A]	3.33	30.0	30.0	80.0	0.35	0.10	0.040
Maintenance, adults*	0.20	0.80	0.80	0.8	0.007	0.002	0.001
Maintenance for light exercise[A]	0.20	0.80	0.80	0.8	0.007	0.0020	0.0010
Maintenance for moderate exercise[A]	0.23	0.90	0.90	0.9	0.008	0.0023	0.0012
Maintenance for heavy exercise[A]	0.25	1.0	1.0	1.0	0.009	0.0025	0.0012
Maintenance for very heavy exercise[A]	0.25	1.0	1.0	1.0	0.009	0.0025	0.0012
Gestation, months 1–6[B]	0.20	0.80	0.80	0.8	0.007	0.002	0.001
Gestation, months 7–8[B]	0.20	0.80	0.80	0.8	0.007	0.002	0.001
Gestation, months 9–11[B]	0.25	0.80	0.80	1.0	0.008	0.002	0.001
Lactation, Foaling to 3 months[BC]	0.25**	1.0	1.0	1.25	0.009	0.0025	0.0012
Lactation, 4–5 months[BC]	0.25**	1.0	1.0	1.25	0.009	0.0025	0.0012
Lactation, >5 months[BC]	0.25**	1.0	1.0	1.25	0.009	0.0025	0.0012
Stallion, nonbreeding	0.20	0.80	0.80	0.8	0.007	0.002	0.001
Stallion, breeding	0.20	0.80	0.80	0.8	0.007	0.002	0.001

*Maintenance is the dietary intake to achieve equilibrium, replacing endogenous (faecal, urinary and cutaneous) losses, and each requirement assumes an absorption efficiency of 0.3 to 0.35 for Cu.

**NRC (2007) retains the estimate of 0.25 mg/kg BW (NRC 1989), although the current estimate is 0.217 mg/kg BW.

[A]Where appropriate growth requirement should be added to maintenance requirement, e.g. yearlings in training.

[B]Including maintenance.

[C]Requirements assume milk yield is directly and linearly proportional to BW.

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Winkelsett *et al.* 2005), although the dietary levels of these nutrients did not meet recommendations (GEH 1994).

In vitro studies indicated that copper reversed proteoglycan depletion of cartilage culture in the presence of synovial fluid in a dose-dependent manner, i.e, Cu had a *chondroprotective* effect (Davies 1998). Davies hypothesised that Cu exerts an anti-inflammatory action; but parenteral Cu supplementation of brood mares in late gestation had no effect on the Cu content of their foals at birth (Gee *et al.* 2000). Yet Pearce *et al.* (1998b) showed that, on Cu-deficient pastures, Cu supplementation of pregnant mares, but not of their foals, decreased the radiographic indices of physitis in the distil third metatarsal bone of their foals at 150 days of age. This led to the conclusion that supplementation improved foal skeletal development *in utero* rather than through increasing liver Cu reserves in the neonate. A note of caution was raised by Pearce *et al.* (1998a) because the Zn:Cu ratio was three-fold greater in the unsupplemented group cf. the supplemented group. Moreover, the Ca and P intakes were low with a Ca:P ratio below 1.0 for 6 of the 11 months of the trial (Pearce *et al.* 1998b). Van Weeren *et al.* (2003) found no relationship between foal, or mare, liver Cu concentration and osteochondrosis status at 5 or 11 months. However, with increasing age, the rate of decrease in number and severity of osteochondrotic lesions was less where Cu status was low at birth cf. those with a high Cu status at birth. So there might be an effect of high Cu status on the natural process of repair of early lesions, indicating the pre-eminence of Cu status of the pregnant mare.

For months 9, 10 and 11 of gestation and for postnatal foal growth the NRC (2007) concluded that the requirement, including replacement of endogenous losses, is 0.25 mg Cu/kg BW daily. This is equivalent to 12 mg/kg diet. Mare's milk contains approximately 0.185 mg Cu/kg and assuming an absorption efficiency of 0.35 and a production rate of 3.2% of BW daily, the daily dietary requirement for maintenance plus production is (0.2 mg + 0.017 mg

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Cu)/kg BW for the lactating mare. The 0.217 mg Cu/kg BW is equivalent to 8.7 mg/kg DM of diet. NRC (2007) retained the previous recommendation for lactating mares of 0.25 mg Cu/kg BW daily (Table 3.11).

Zinc (Zn)

A deficiency of dietary zinc in many domesticated animals, including the horse, depresses appetite and growth rate in the young, causes skin lesions and a depression of Zn concentrations in the blood. A deficiency of Zn in the rat and several other species causes abnormal development of ribs and vertebrae, cleft palate, micrognathia (undersized mandible) and agenesis (absence) of long bones, but there is little direct evidence of this in the horse. Excess Zn exacerbates bony lesions induced by low Cu diets.

Zn as enzyme cofactor

Zn is a cofactor for over 200 enzymes in animals, either as part of the molecule or as an activating cofactor. The enzymes include ALP (EC 3.1.3.1), collagenase (EC 3.4.24.3) and carbonic anhydrase (EC 4.2.1.1), all required in bone formation. Alkaline phosphatase also requires Mg, and excess dietary Zn inhibits the enzyme if Mg is displaced. A deficiency of Zn thus has fairly widespread physiological effects, but high dietary levels are required for toxicity.

Zn supplementation

Siciliano *et al.* (2003a) observed positive total immunoglobulin and IgM humoral immune responses in weanling horses given a supplement of Cu, Zn and Mn. Zn has a potent immunomodulatory capacity, particularly influencing T-helper-cell organization and cytokine secretion. *Culicoides* hypersensitivity in horses, causing a pruritic skin disease, resembles the early and late phases of type I hypersensitivity reactions in man. Stark *et al.* (2001) determined that the severity of *Culicoides* hypersensitivity in Icelandic horses expressed a low, but significant, negative correlation with plasma Zn concentration. However, their more recent observations indicate that *Culicoides* bites are a cause of Zn redistribution between plasma and blood cells, and as plasma holds only 10–23% of total blood Zn, plasma Zn level is not a reliable indicator of nutritional status. Thus, the depression in plasma Zn is quite likely to be a reaction to the bite of this fly.

Zn supplements are claimed to benefit hoof wall development, although Siciliano *et al.* (2001) could not influence the trace mineral content of hoof wall, or wall growth rate, hardness and tensile strength, with supplements of Cu, Zn or Mn, given to light mares as inorganic salts, or as amino acid chelates. Siciliano *et al.* (2003a) increased hoof wall hardness, but not other characteristics of the wall, with similar supplements of Cu, Zn and Mn.

Absorption and retention of zinc

Many feedstuffs contain 15–40 mg Zn/kg DM. Inorganic salts possess a higher availability than do phytate salts of zinc in cereal grains and oilseed meals. The efficiency of Zn absorption in all forms is probably affected more by diets rich in phytate than is the absorption of other trace elements, but even so, high phytate concentrations are unlikely to depress the utilization of Zn by more than 30–40%. Sources of supplemental Zn include Zn sulphate, Zn oxide, Zn chloride, Zn carbonate and various organic Zn chelates. Wagner *et al.* (2005) found absorption of Zn as the oxide, sulphate and organic chelate was 13.9, 12.8 and 10.6%, respectively, and Baker *et al.* (2005) reported that mature horses retained more Zn when it was given as the oxide rather than as the organic chelate. In contrast, Miller *et al.* (2003) reported increased Zn retention by yearlings given organic as opposed to inorganic Zn. An increase in dietary Zn from 26 to 100 mg/kg progressively increases serum Zn concentrations.

Availability of organic v inorganic trace minerals

Various organic mineral sources differ in their individual mineral content and bioavailability. The **ligands** include a wide range of metal–ligand structures: one or more amino acids, proteins, polysaccharides, or propionate. Mineral availability from these organic sources is dependent upon the type of bonding (ionic or covalent) between metal and ligand, ligand size and how each is influenced by pH (NRC 2007). Weaker metal–ligand bonds (ionic) and small ligand size result in a less stable molecule, whereas strong bonds (covalent) and large ligand size result in greater stability, but create concerns over availability (Hynes & Kelly 1995). A number of studies to measure the relative efficacy of organic mineral sources have been reviewed (Ott & Johnson 2001; Baker *et al.* 2003, 2005; Miller *et al.* 2003; Siciliano *et al.* 2003a; Naile *et al.* 2005) by NRC (2007). The conclusion is that there is minimal, or no, difference in biologic use, or animal performance cf. inorganic sources; although results with weanling and yearling horses tended to show a positive response from organic mineral sources (Ott & Johnson 2001; Miller *et al.* 2003; Siciliano *et al.* 2003b). In comparisons of the efficacy of organic and inorganic sources of Cu and Zn, two out of three studies indicated more effective use of organic sources in young horses (Miller *et al.* 2003; Siciliano *et al.* 2003a,b), but the third in adult horses indi-

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Table 3.12 The true digestibility, endogenous loss and estimated daily requirements for Cu, Zn and Mn in sedentary (SED) and exercised (EX) TB geldings of 11 years of age and 534 kg BW (Hudson *et al.* 2001).

		Cu	Zn	Mn
Digestibility (%)	SED	41.8	25.4	57.9
	EX	54.5	14.3	40.2
Endogenous loss (mg/day)	SED	15.7	65.2	304.8
	EX	20.3	69.6	163.6
Requirements (mg/day)	SED	44.2	274.4	528.6
	EX	35.0	461.3	408.3

cated low absorption efficiencies of both sources (Wagner *et al.* 2003).

Hudson *et al.* (2001) measured the true digestibilities and requirements for Cu, Zn and Mn from a mixture of organic and inorganic sources in sedentary and exercised horses. They found a Zn requirement of 461 mg/day in exercised horses, cf. sedentary horses of 274 mg/day, as digestibility in sedentary horses dropped from 25 to 14% when exercised (Table 3.12). The endogenous losses of Zn lie between 50 and 70 mg/d for a 500–550 kg horse (Pagan 1994; Hudson *et al.* 2001), or 0.1–0.13 mg Zn/kg BW daily. True digestibility was calculated to be 20.8% (Pagan 1994), indicating a dietary requirement of only 0.47 mg Zn/kg BW daily. Grace *et al.* (1998b) measured the mineral composition of horses aged 150 days (approximately 190 kg BW) and, assuming a growth rate of 1.0 kg/day on pasture, they estimated the daily requirement for Zn to be 152 mg. NRC (2007) has retained their previous estimate of 40 mg/kg DM of diet, assuming a feed intake of 2% of BW daily (Table 3.11).

Zinc toxicity

Zinc generally does not accumulate in tissues with continued exposure (Casteel 2001) and it is one of the less toxic of the essential trace metals, yet where there is industrial pollution of pastures, grazing animals show signs of toxicity. Zinc intoxication is associated with reduced Ca absorption. Toxic dietary concentrations probably exceed 1000 mg/kg BW from industrial pollution and very high intakes, e.g. 5.4 g Zn/kg diet, cause anaemia, enlarged epiphyses, stiffness of gait (including breaks in the skin around the hooves; Willoughby *et al.* 1972a,b) and lameness (Eamens *et al.* 1984), probably caused by secondary copper deficiency (Bridges & Moffitt 1990).

Cu and Zn interactions

These metals probably compete for the same transport mechanisms. The amount of Zn necessary to cause a deficiency of Cu depends on the hepatic Cu reserves; but whether the competition between these elements occurs during absorption or post-absorption is unclear. In man and most other domestic animals there is an antagonism between Zn and Cu. Excessive intakes of Zn, especially where dietary phytate, Ca and Cu are not abundant, can cause Cu deficiency; but Hoyt *et al.* (1995b) found that dietary Zn concentrations of 73–580 mg/kg, provided as zinc oxide, had no influence on either Cu absorption or retention by horses from diets containing 12 mg Cu/kg. Yet as little as 100 mg Zn/kg diet has been shown to increase the faecal loss of Cu and to lower blood Cu by about 10%; although this might not reflect any change in Cu absorption efficiency. Nevertheless, as Cu adequacy seems to be critical for proper bone development we should assume that dietary Zn at concentrations above 100 mg/kg are to be avoided. Amino acid chelation of trace elements avoids any interruption to Cu absorption by other metals and the competition for absorption is then between amino acids.

Manganese (Mn)

A severe deficiency of Mn can give rise to resorption *in utero*, or death at birth, and lesser deficiencies provoke irregular oestrous cycles. The young seem to suffer lameness and incoordination of movement if they lack sufficient Mn, and a deficiency is a possible explanation of tiptoeing where suckled foals are on pasture containing <20 mg/kg DM. A deficiency is thought to be a cause of enlarged hocks, and, by affecting the growth plate, to shorten legs with characteristic knuckling-over of joints. In the USA, excessively high concentrations of Ca in samples of alfalfa are said to precipitate a flexural deformity of the legs of growing horses, which is rectified by Mn supplements.

Mn is essential for carbohydrate and lipid metabolism. It is required as a cofactor for glycosyltransferases, which catalyse the transfer of a sugar from a nucleotide-diphosphate to an acceptor, and so Mn is essential in several stages of glycosaminoglycan–chondroitin sulphate synthesis in cartilage formation. Epiphyseal cartilage and bone matrix formation are compromised by a deficiency of Mn. It is also required as a cofactor in Mn-containing superoxide dismutase.

A sample of mare's milk contained 0.014 mg Mn/kg (Anderson 1992). Forages contain 40–140 mg Mn/kg DM and most concentrates contain 15–45 mg Mn/kg DM.

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Wagner *et al.* (2005) reported that Mn oxide, sulphate and an organic chelate were absorbed to the extent of 13.6, 8.6 and 15.5%, respectively. Pagan (1994) reported true Mn digestibility to be 28% in mature horses, although Hudson *et al.* (2001) showed the true digestibility of Mn to decrease from 58 to 40% with exercise (Table 3.12). This wide range of values implies that it is impossible to give a dietary requirement of Mn with any degree of precision. It was shown by Sobota *et al.* (2001) that a total dietary Mn concentration of 36 mg/kg (using a concentrate containing 23 cf. 131 mg/kg and coastal Bermuda grass hay of 55 mg/kg) is inadequate in yearling TB and Quarter Horses for maximum weight gain, long bone growth and biochemical indices of bone formation.

Lawrence *et al.* (1987b) reported normal plasma Mn values of 100–180 µmol/L serum in yearling ponies. Hudson *et al.* (2001) calculated that endogenous losses of Mn are between 0.31 and 0.57 mg/kg BW daily, indicating a dietary maintenance requirement of 0.76–0.99 mg Mn/kg BW daily. Pagan (1994) estimated endogenous losses to be 0.2 mg Mn/kg BW daily resulting in a dietary requirement of 0.7 mg Mn/kg BW daily. These results indicate a need for a dietary concentration of approximately 35 mg/kg DM. By contrast Sobota *et al.* (2001) reported decreased growth rates in yearlings receiving 36 mg Mn/kg ration compared with those receiving 101 mg Mn/kg ration and concluded that the minimum requirement for growth is 40 mg Mn/kg DM of diet. This concentration has been retained by NRC (2007) as the overall dietary recommendation (Table 3.11).

Iron (Fe)

Most natural feeds, apart from milk, are fairly rich sources of iron, although the availability is questionable, but deficiencies are unlikely unless the horse is anaemic through heavy parasitization. Many forages contain 100–250 mg Fe/kg DM, but grains usually contain <100 mg/kg DM. Absorption efficiency is likely to be <15 % where there is enough in the diet. The foal is born with an adequate store of liver Fe and the foal's grazing activity is normally an adequate supplement to the mare's milk, which contains meagre amounts of most trace elements, with the possible exception of selenium (Se) in mares supplemented with this element. The levels of Fe in Arabian mare's milk are shown in Table 3.8.

Fe is contained in haemoglobin (Hb, 60%), myoglobin (20%), storage and transport forms (20%), cytochromes and many enzyme systems (0.2%). The body of a 500 kg horse contains approximately 33 g Fe. A deficiency of Fe causes anaemia, but evidence of dietary Fe deficiency anaemia in horses is rare. Signs of deficiency include microcytic, hypochromic anaemia, but this is not a practical problem if foals or mature horses have access to soil. Brommer & Sloet van Oldruitenborgh-Oosterbaan (2001) reported lowered Hb concentrations and PCV in exercised and unexercised box-stalled weanling foals cf. those raised on pasture. A dietary concentration of 50 mg/kg DM should be adequate for growing foals, whereas 40 mg/kg is said to meet the maintenance requirements of adults. The basal diet used by Lawrence *et al.* (1987b) contained 436 mg Fe/kg (typical of many natural ingredient diets) and the addition of 500 or 1000 mg Fe/kg as citrate, brought no benefit.

True dietary Fe deficiency is often confused with pseudo-Fe deficiency. Anaemia is not synonymous with Fe deficiency. Hb is synthesized preferentially to nonHb Fe-containing proteins in Fe-depleted animals. Blood PCV and Hb is decreased by parasitism. Loss of red cells occurs during TB exercise when some are haemolysed, and 0.6% of dietary Fe occurs in sweat (Inoue *et al.* 2003). In this situation the apparent absorption efficiency of Fe is increased, preventing Fe deficiency.

Fe status is assessed reliably by serum ferritin concentration. Total serum Fe is an unreliable measure of status and declines during microbial invasion, tissue injury, immunological reactions and in any inflammatory process, causing an acute phase response, initiated by IL-1. Normal ferritin values fall between 100 and 200 µg/L serum. Deficiency is assumed when values fall below 50 µg/L, and Fe overload exists when values exceed 400–450 µg/L. The dietary Fe requirements proposed by NRC (2007) are given in Table 3.11.

Adverse effects of Fe supplements

Fe-containing haematinics are widely used in the horse industry. Fe supplements and injectable Fe are used in the mistaken belief that PCV and Hb concentration can be regularly increased. The risk of Fe toxicity is a real one and antagonistic interactions with Zn and Cu occur. Fe injections can be fatal. Most Fe is stored in the liver and spleen. If absorbed dietary Fe exceeds the Fe-binding capacity of the serum, the resulting free-Fe reaching the liver can cause liver necrosis and failure (Schryver 1990), as there is no mechanism for disposal of excess. Neonatal foals are particularly susceptible to iatrogenic Fe toxicity. Ferrous-Fe (divalent) is more soluble than ferric-Fe (trivalent) and therefore ferrous sulphate or fumarate is generally used in supplements, presenting a greater risk. Ingestion of a large dose of ferrous fumarate shortly after birth are particularly dangerous and can cause death within

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five days. Prior to death foals can exhibit diarrhoea, icterus, dehydration and coma and *post mortem* signs include pulmonary haemorrhages and Fe deposition in, and degeneration of, the liver. Daily administration of as little as 300 mg Fe in the ferrous form has been associated occasionally with signs of Fe toxicity in adult TBs. Although large amounts of dietary Fe can be toxic, adult ponies given oral ferrous sulphate daily at a rate of 50 mg Fe/kg BW for a period of nearly 8 weeks showed no signs of toxicity (Pearson & Andreasen 2001). Large, but natural, intakes of Fe in cattle can cause Cu deficiency. An induced Cu deficiency of this kind is less likely in horses, although reductions of splenic Cu and Mn, associated with intakes of 1400 mg Fe/kg diet, were observed by Lawrence *et al.* (1987b). These workers noted depressed serum and liver Zn with dietary Fe intakes of only 890 mg/kg. Interactions of this kind arise from competition for binding by intestinal transferrin and/or other transport mechanisms. The toxic Fe level depends on several factors, including concurrent disease processes, previous liver damage and vitamin E and Se status, as Fe is involved in oxidation–reduction reactions. When glutathione peroxidase activity is depressed and vitamin E status is reduced with an Fe overload, catalase activity increases, causing the liver damage, coagulopathy and raised mortality. Fe overload in the spleen results in lymphopenia, frequently observed in neonatal cases.

In extended Fe intoxication there is frequently:

- hepatic dysfunction, expressed as lethargy;
- yellow discolouration and petechial haemorrhages of mucous membranes; and
- thrombocytopenia and elevated activities of serum gamma-glutamyltransferase (GGT; EC 2.3.2.2) and ALP.

These enzyme changes are indicative of cholestatic hepatopathy, with periportal bile ductule proliferation, as one origin of these enzymes is the biliary epithelium.

Fluorine (F)

Fluorine readily substitutes for the hydroxyl ion in bone and teeth hydroxyapatite, creating a more stable crystal of Ca, P and Mg. It does not diffuse into formed bone, but becomes incorporated during bone formation. F increases osteoblast number by increasing osteoprogenitor cell proliferation. Risks of dietary excess (fluorosis) in grazing horses probably exceed risks of deficiency, as F has a narrow therapeutic index. Excess contamination of pastures, especially from brickworks, causes a softening, thickening and weakening of bones, through defects in mineralization, which are not prevented by Ca and vitamin D. A decrease in industrial effluent in many countries has ensured that few cases now occur. Forages naturally contain between 2 and 16 mg F/kg DM, whereas grains usually contain 1–3 mg F/kg DM.

A world shortage of sources of digestible P has led to an increase in the use of rock phosphates; as some of these are rich in F a careful scrutiny of their composition is essential before purchase and use.

Horses seem to excrete more F in faeces than cattle and can tolerate 50 mg F/kg ration for extended periods without detrimental effects (Shupe & Olson 1971). Nevertheless, the maximum tolerable concentration of dietary F has been retained at 40 mg/kg DM intake (NRC 2007). Any potential dietary need for F is met by natural feed ingredients.

Iodine (I)

Iodine is a relatively rare element in the earth's crust and is not required by mono- and dicotyledonous plants, in which concentrations are low. The digestibility of I is high as renal excretion increased linearly with increasing intake from 0 to 80 µg I/kg BW daily, while faecal excretion remained low and relatively constant (Wehr *et al.* 2002). In man and animals both I deficiency and toxicosis result in hypothyroidism (toxicosis causes hyperthyroidism in some individuals). Goitre can then occur, with hyperplasia and hypertrophy of the thyroid gland, induced by the elevated secretion of thyroid-stimulating hormone (TSH) originating in the anterior pituitary in the absence of feedback inhibition. In both a deficiency and excess of dietary I, TSH production is increased in an attempt to overcome the reduced concentrations of thyroid hormones and alleviate hypothyroidism. This increase in TSH production causes an increased size of the thyroid gland, i.e. goitre. A high I intake when Se is deficient can lead to thyroid tissue damage as a result of low activity of circulating glutathione peroxidase (GSH-px). This is not necessarily the outcome of I toxicosis in the pregnant mare (see below).

In respect of both foetal I deficiency and toxicity the mare is at risk throughout gestation, and even shortly before fertilization. When receiving insufficient dietary I mares can show no external signs, but exhibit abnormal oestrous cycles and then produce hypothyroid foals. Serum triiodothyronine (T_3) and thyroxine (T_4) concentrations in the mare are low, generally below 1.3 and 19 nmol/L (0.7 and 15 µg/L), respectively (Table 3.13). The I concentration of deficient mare's milk is <20 µg/L. A deficiency with these signs occurs when mares graze inland pastures that are frequently deficient in I.

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Table 3.13 Normal ranges for plasma T_3 and T_4 concentrations in TBs (nmol/L).

	T_3	T_4
Pregnant mares	0.6–2	3–56
Foals*	2–10	10–150
Adults in training	0.3–2	5–28

*T_3 levels increase to a maximum at 48 h of age and then decline.

Deficiency signs in foals

Deficiency signs in foals are enlarged thyroid glands, weakness, persistent hypothermia, respiratory distress and high neonatal mortality. There is an increased susceptibility to infectious disease and respiratory infections are frequent. T_4 levels in foal serum will be low, whereas T_3 concentration is normal. Plasma T_3 and T_4 levels are generally not too helpful in diagnosis and their correlation is poor. Nevertheless, T_4 concentration is depressed in hypothyroidism. Of greater value is inspection of feed and determination of I in feed samples and plasma.

Toxicity signs in mares and foals

In mares given 300–400 mg I daily in the feed, infertility and abortions occur. Some mares develop hyperthyroidism, expressed as elevations in the plasma concentration of both T_3 and T_4 and suppressed plasma TSH, or they develop hypothyroidism, expressed as normal T_3 and depressed T_4, although clinically they can appear euthyroid. At birth the foal is hypothyroid, frequently with colloid goitre, variably sized thyroid follicles containing a single layer of low cuboidal cells and there are low concentrations of circulating T_3 and T_4. Interestingly, the tracheal mucosa reveals squamous metaplasia, reminiscent of vitamin A deficiency.

In man, excess circulating iodide induces what is called the Wolff–Chaikoff block which inhibits I uptake, preventing the synthesis of T_4 from I. In many species this is a temporary phenomenon, but in the horse the block may depend on the precise level of I intake, although in the author's experience it is temporary. In the neonatal foal, I toxicity blocks the release of T_3 and T_4 from the follicle, apparently by interfering with colloid proteolysis in the acini. Some pathologists give weight to the T_3:T_4 ratio although, in the author's experience, the two hormones are poorly correlated in healthy horses.

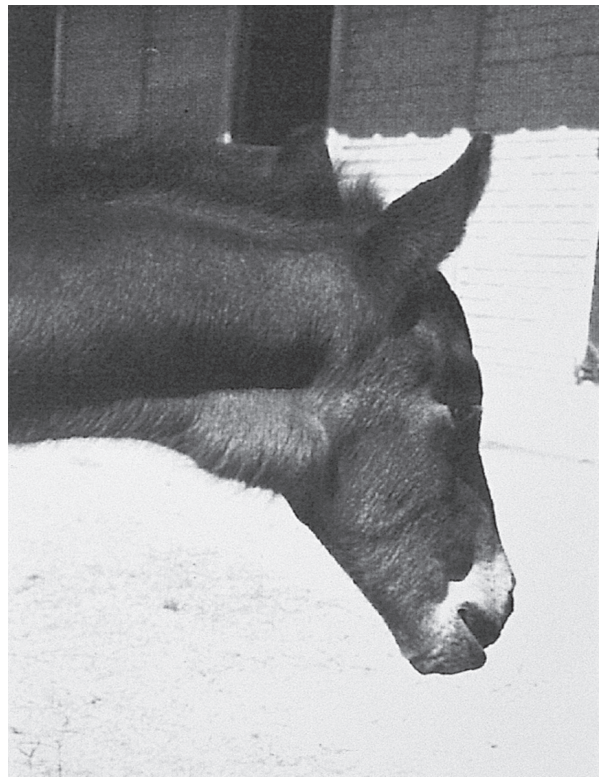
Algae, such as red wrack seaweed, contain high concentrations of I and the feeding of dried kelp, a seaweed, to pregnant mares is frequently a cause of iodism in foals, with similar clinical signs to those of I deficiency. There have also been cases where foals given excessive thyroprotein supplements develop focal depigmentation. If the daily I intake of a pregnant 550 kg mare is between about 40 and 400 mg I, goitre appears in her offspring. Eroksuz *et al.* (2004) reported goitre in new-born foals whose dams had received 299 mg I/day during the last 24 weeks of gestation. At the upper end of the range the mare has goitre. Intakes of 30–50 mg I daily by the mare might just cause enlargement of the thyroid in her offspring, but the foal can be euthyroid and present no other abnormality, although hirsute foals are seen. If the intake exceeds about 100 mg I/day for any length of time during pregnancy, the newborn foal is likely to show signs of hypothyroidism, expressed as weakness, lethargy, high neonatal mortality, poor muscular development and, most patently, osseous dysplasia of long bones. The author's observations indicate that these include angular deformity, tendon contraction and hyperextension of the lower limbs with poor ossification of the carpal and tarsal bones. This extension causes the foal to walk on its heels with its toes raised from the ground. Abnormal growth of long bones occurs at the epiphysis and in the shank. These are thin and small with cortical thickening, which appears to result from a greater reduction in osteoclasia than in osteogenesis. Protruding jaws (mandibular prognathism) and parrot mouth (brachygnathia) have also been observed (Plate 3.1).

The reason for these consequences is that I is concentrated in the placenta which delivers excessive amounts to the foetus, causing congenital malformations and concentrations of I in foetal blood two to three times that of the dam's blood, while T_3 and T_4 can be depressed. I is also concentrated in the mammary gland and is secreted in the milk. If the excessive I source given to pregnant mares is excluded from the diet well before parturition the euthyroid state can be reimposed and normal foals result. Involution of the thyroid, congenitally enlarged in young foals owing to I excess, is brought about by removal of the I source(s). This includes removal of the mare's milk. Normal development occurs, as long as bony abnormalities have not already developed. The foal will be slightly undersized, as hypothyroidism is associated with decreased synthesis and release of growth hormone.

Goitrogens

I is absorbed from the gut principally as iodides. Some feeds (raw soya beans, white clover, kale, mustard and

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(a)



(b)

Plate 3.1 Signs of iodine intoxication at birth of foals out of mares receiving excessive amounts of dietary iodine during pregnancy. The principal signs in the stable were thyroid enlargement and bony abnormalities of neonatal foals. (a) Goitre in a foal, out of a euthyroid mare. (b) Protruding lower jaw (prognathism) of intoxicated foal. (c) Muscular weakness and long bone abnormalities of intoxicated foal. (d) Enlargement of stifle joint in intoxicated foal.

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(c)



(d)



Plate 3.1 *Continued*

cabbage) contain goitrogens (see Chapter 5) which can inhibit the uptake of free I by the colloid proteins of the thyroid, preventing its incorporation into tyrosine. Heating is necessary to inactivate the enzyme responsible for this effect in these feeds. Thioglucosides, thiocyanates and perchlorates can be goitrogenic. The effects of these substances can be overcome by increasing the I intake. However, the goitrogenic effects of thiouracil-, thiourea- and methimazole-containing drugs cannot be overcome in this way, as they interfere with T_4 synthesis. (See 'Selenium and iodine interactions', this chapter.)

Supplements

Diets supplemented with 0.2 mg I/kg should adequately meet the requirements of horses. The I is preferably added as potassium iodate, rather than as potassium iodide, as iodides are readily oxidized to I which is volatile and so gradually lost from premixes and diets. However, iodates

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are more toxic in a concentrated form and should be handled with care.

Treatment of hypothyroidism

Young foals diagnosed with goitre should not be treated with iodide, or iodate, salts in the first instance. After receiving colostrum they should be removed from their mother's milk to a source known to contain normal amounts of I. These foals are first treated with T_4 while other analyses are undertaken to determine the cause of the malady. The author's evidence is that the T_4 rapidly raises blood T_4, whereas treatment with I salts has only a marginal initial influence on plasma T_4.

Requirements

Endogenous losses are assumed to be 7 µg I/kg BW daily (Wehr *et al.* 2002; NRC 2007). Assuming a near 100% absorption efficiency, the maintenance requirement is 0.007 mg I/kg BW daily, or 0.35 mg I/kg DM diet. This value is assumed for all classes of horse, apart from brood mares during late gestation for whom the concentration has been set at 0.4 mg I/kg DM of diet (NRC 2007).

Selenium (Se)

Function and natural sources

Selenium has gained prominence following the realization that it forms an integral part of the GSH-Px (EC 1.11.1.9) molecule. This enzyme catalyses peroxide detoxification in bodily tissues during which reduced glutathione (GSH) is oxidized. It is closely involved with the activity of α-tocopherol, which protects polyunsaturated fatty acids from peroxidation. The requirements for α-tocopherol and Se are increased in the presence of high levels of dietary polyunsaturated fatty acids – cod liver oil, linseed and corn oils – and, in fact, pasture grass. A possible case for fish oils is put in Chapter 5, although they require higher levels of protection from vitamin E, owing to a greater degree of unsaturation than of vegetable oils.

Vegetable oils are generally much richer sources of polyunsaturated fatty acids than are hard animal fats. Although linoleic acid (an *n*-6 fatty acid) is perhaps the most abundant polyunsaturated fatty acid in vegetable oils, some oils, including those present in grasses, are richer in α-linolenic acid (an *n*-3 fatty acid). This is particularly sensitive to peroxidation in tissues (α-linolenic acid has benefits; see Chapter 5, 'Fat supplements').

The alkali treatment of roughage to improve its digestibility has attracted interest. Such treatment destroys α-tocopherol and β-carotene, and unless appropriate supplementation is given, signs of myopathy could occur in animals consuming significant quantities of such roughage.

The Se content of herbage, cereals etc. depends on the Se content of the soil in which they grew. Areas of deficiency include New Zealand, and low soil concentrations occur in parts of Scotland and the eastern and western states of the USA (Scottish wheat 0.028 cf. Canadian wheat 0.518 µg Se/g DM, Barclay & MacPherson 1992). The Great Plains and central southern states of the USA have regions of high Se concentration in crops. These differences are reflected in blood Se concentration, in GSH-Px activity and in performance. In the western USA, blood concentrations of Se are negatively correlated with the incidence of reproductive diseases in mares (Basler & Holtan 1981). Blood concentrations ranged from 1.2 to 3.1 µmol/L and dietary concentrations ranged from 0.045 to 0.461 mg/kg, i.e. deficient to slightly excessive. Normal blood Se ranges from 0.8 to 2.8 µmol/L in adult mares (Blackmore & Brobst 1981), possibly indicating that some 'normal' animals have impaired reproductive capacity. Serum Se levels are correlated with that in whole-blood, and serum values are a satisfactory measure of status.

Deficiency signs

Owing to a narrow **therapeutic index** signs of both deficiency and toxicosis occur under natural conditions, and toxicity has occurred through over-supplementation (Edmondson *et al.* 1993). Se deficiency produces pale, weak muscles in foals and a yellowing of the depot fat. This form of muscular dystrophy in foals is related to subnormal levels of blood Se, depressed activity of GSH-Px, but elevated serum CK, AST, potassium and blood urea-N. Nevertheless, it is suggested that neither a low serum Se, nor a low GSH-px activity necessarily indicates a physiological deficiency of Se without other evidence, as Vervuert *et al.* (2001) noted that low plasma Se concentrations (66.7, SE 47.2 µg/L) in Icelandic horses possessing apparent good health and low GSH-px activities were not correlated with either Se intake or Se plasma concentrations (Wichert *et al.* 2002). Normal serum concentrations for adult horses range from 130 to 160 µg/L (1.6–2.0 µmol/L), but for foals the range is typically from 70 to 80 µg/L (0.9–1.0 µmol/L), and values of <65 µg/L (<0.8 µmol/L) could be indicative of deficiency (Blackmore *et al.* 1982).

Se supplements and their metabolism

Se supplementation is given as either sodium selenite, or sodium selenate, and evidence (Podoll *et al.* 1992) indicates there is no difference between them in potency.

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Janicki *et al.* (2001) reported greater concentrations of Se in serum, colostrum and milk of mares and higher serum Se concentrations, GSH-Px activity and influenza antibody titre, possibly associated with greater B-cell proliferation, in their foals when the mares were given 3 mg Se/day, as Se-yeast, cf. mares receiving 1 mg or 3 mg Se/day as sodium selenite. On the other hand, Richardson *et al.* (2006) observed no difference in status between two sources of oral Se (0.45 Se mg/kg diet as $NaSO_3$ or as zinc-L-selenomethionine) given for 56 days in conjunction with a control diet containing 0.15 mg Se/kg. Measurement in laboratory animals shows that organic plant sources of Se are more potent than inorganic, although barium selenate possesses a high availability. Se in forages, seeds and Se-yeast occurs as selenocystine, selenocysteine and selenomethionine. These are not chelates in which the metal ion is bound to one or more ligands, but rather the Se has replaced the S atom and absorption occurs via amino acid transporters, increasing its presence in tissue amino acids cf. inorganic Se (Pagan *et al.* 1999b, Gunter *et al.* 2003); but the more effective provision of Se, cf. this inorganic Se, as the cofactor for several enzymes, e.g. red blood cell (RBC) GSH-Px, has not been demonstrated (Richardson *et al.* 2003). Selenocysteine, the predominant form of Se in whole blood and plasma, and GSH-Px activity can be increased by both Na selenite and Se-yeast, but the selenomethionine content of whole blood and plasma is increased with Se-yeast, but not by Na selenite (Calamari *et al.* 2009). Exercise increases the urinary loss of Se from selenite. After absorption RBCs take up inorganic Se and return it to the plasma during exercise in a reduced form, hydrogen selenide. Some of this, not bound to plasma protein, is excreted through the kidneys and some is transported to the liver to form part of the selenoprotein pool. By comparison, organic Se is transported in the blood bound to amino acids and is less likely to be lost through the kidneys (Pagan *et al.* 1999b). It is concluded there is a difference between sources, not only in digestibility, but also in metabolism, and the conclusions drawn depend upon the response measures adopted.

Se in reproduction and exercise

It is essential that pregnant mares receive adequate Se, as their status affects their foals as well as their performance at parturition, while their milk provides only modest amounts of Se (Lee *et al.* 1995). Deficient pregnant mares given at least 500 mg vitamin E and 25 mg Se by intramuscular injection two or three weeks prior to parturition, responded with shorter placental retention times and a shorter interval between parturition and effective mating (Ishii *et al.* 2002). Although the foal's serum Se was raised by treatment of their dams it was still deficient. Serum Se values fall to <0.3 µmol/L in foals and reduced amounts among TBs in the UK have been associated with poor racing performance (Blackmore *et al.* 1979, 1982). Only in extreme deficiencies, not normally seen in adult animals, is there sufficient muscle damage for extensive membrane leakage of enzymes such as aspartate aminotransferase (AST; EC 2.6.1.1) and creatine kinase (CK; EC 2.7.3.2) to be detected. Normal peroxidation occurring in muscles after exercise is not reduced by supplementary Se. Se deficiency *in vitro* changes neutrophil function and so a deficiency could depress resistance to infection.

Permitted dietary levels of Se

In 2004 the US Food and Drug Agency (FDA) permitted the use of Se-yeast at a rate not exceeding 0.3 mg/kg of added Se in total diet (Weese 2003). NRC (2007) and Kavazis *et al.* (2002) concluded that dietary concentrations of 0.1 mg/kg DM should prevent classical signs of deficiency and that the only justification for feeding Se at slightly greater concentrations, i.e. 0.5 mg Se/kg DM, might be for optimum immunity, as Janicki *et al.* (2001) observed a higher influenza antibody status in foals out of mares receiving 3 mg Se/day cf. 1 mg Se/day.

Toxicosis

Se is highly toxic to animals and to persons handling the salt in concentrated forms. The minimum toxic dose of Se through continuous intake is 2–5 mg/kg feed and acute toxicity is caused in sheep given amounts ≥0.4 mg Se/kg BW in a single dose. Serum Se concentrations of 1–5 mg/L (12–63 µmol Se/L) occur in chronic Se toxicosis and values of 2–2.5 mg/L (24–32 µmol/L) occur in acute intoxication of horses (Blackmore *et al.* 1982). Chronic intoxication involves a steady increase in tissue Se accumulation over months to a plateau at which excretion in urine and faeces keeps pace with intake. Saturation values of liver, kidney, hair and hooves are approximately 20–30 mg/kg DM for which Se, replacing sulphur, is incorporated in tissue proteins as selenocystine and selenomethionine.

Outside the UK there are areas where soils can contain in excess of 0.5 mg Se/kg and amounts of 5–40 mg/kg DM are found in certain accumulator plants and their consumption results in toxicity (Finley 2005). The predominant forms of Se in these plants are methylselenocysteine and selenocystathionine. Concentrations of up to several thousand mg/kg have been detected in species of milk vetch (*Astragalus*). Various species of woody aster (*Xylorhiza*) and goldenweed (*Oonopsis*), which grow in low-rainfall

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areas, are also indicator plants, containing relatively high levels of Se. Toxicity is more common in dry regions, but horses select grasses rather than these toxic weeds where there is adequate grazing, as the indicator plants are unpalatable. Where grass is sparse, animals suffer from 'alkali disease' in which excessive Se causes a loss of hair on the mane and tail, lameness, bone lesions, including twisted legs in foals, and sloughing of hooves. Plate 3.2 (S. Ricketts, personal communication) depicts signs in a Se-intoxicated adult horse. The diseased hooves were hollow and there was greying of hair in the mane and tail, a characteristic not previously noted. The abnormal hoof horn and hair contained much higher Se concentrations than did the healthy material.

Doses of Se received by intoxicated horses are normally impossible to estimate accurately from field evidence, owing to an accompanying loss of appetite. There is no simple remedy for this intoxication apart from removal of the animals from the region if destruction of the seleniferous plants is impractical.

Se and I interactions

Selenium functions not only as a component of GSH-Px (EC 1.11.1.9) but also as part of the enzyme 1,5′-iodothyronine deiodinase (EC 3.8.1.4), required in the conversion of T_4 to T_3. As T_3 is the more potent hormone, Se deficiency is said to be associated with human cretinism in northern Zaïre. There are no reports of goitre in Se-deficient foals, so that the equine Se requirement for the functioning of this deiodinase is probably less than that for normal function of GSH-Px.

Chromium (Cr)

In the 1950s it was shown by Schwartz and Mertz that impaired glucose tolerance in rats could be caused by a deficiency of the glucose tolerance factor, the active constituents of which are trivalent Cr, niacin, glycine, glutamic acid and cysteine.

Thus, chromium is essential for normal carbohydrate metabolism as a potentiator of insulin action, and so it is found in insulin sensitive tissues where it stimulates glucose clearance. When given as an inorganic supplement, trivalent Cr^{III} at dose levels of 300–500 µg Cr/kg diet accelerates glucose clearance, although no improvements occurred in geriatric mares when a supplement of Cr-L-methionine (0.01 mg and 0.02 mg/kg BW daily) was given (Ralston *et al.* 1999). In some species, apparently, Cr improves both cell-mediated and humoral indices of immunity. However, by supplementation of an alfalfa/maize concentrate with Cr-L-methionine, Dimock *et al.* (1999) were unable to demonstrate any improvement in geriatric mares, whose immunocompetence could have been poor. Trivalent Cr is relatively nontoxic, whereas hexavalent Cr, present in chromates and dichromates and acting as a pro-oxidant, is orally 10–100 times more toxic. Rats show no adverse effect from 100 mg Cr^{III}/kg diet. The recommended safe daily intake of Cr^{III} for humans ranges from 50 to 200 µg.

Ott & Kivipelto (1999) gave yearling horses coastal Bermuda grass (*Cynodon dactylon*) hay supplemented with a concentrate providing chromium tripicolinate at 0, 105, 210 and 420 µg Cr/kg total diet. The tolerance to an intravenous glucose injection was improved by the highest Cr supplementation. The Cr could have reduced insulin resistance and stress and facilitated energy metabolism. Nevertheless, doubts have been expressed over the safety of picolinate sources in human nutrition. Chromosome damage in hamsters occurred with treatment by Cr picolinate, whereas it did not occur with Cr nicotinate, nicotinic acid or trivalent Cr chloride hexahydrateat equivalent doses (Stearns *et al.* 1995).

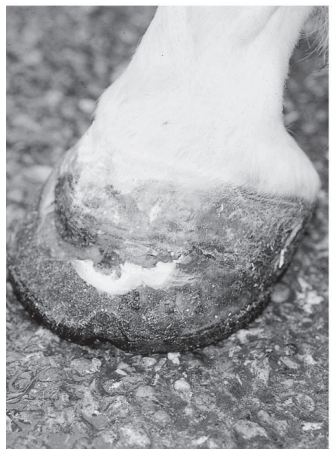
Organic Cr, as the glucose tolerance factor found in brewer's yeast, is more potent than inorganic Cr. Absorption of inorganic forms has been reported to be 0.4–3.0% (Anderson 1987), whereas that from brewer's yeast was 10–25% in rats (Underwood 1977). Barley bread has been given traditionally to individuals suffering from diabetes mellitus in Iraq. Barley, a richer source of Cr than wheat, typically contains 6 mg Cr/kg. Some yeasts are rich sources of organic Cr. Exercised horses given 5 mg Cr, as yeast, in a diet providing 12 mg Cr/day, showed a decrease in both glucose and insulin responses and during exercise a lower plasma cortisol response and raised plasma TAG concentration (Pagan *et al.* 1995). As insulin inhibits the activity of intracellular hormone-sensitive lipase in adipocytes, a lower insulin response would allow more NEFAs to be mobilized for both oxidation and return to the liver. There NEFAs would be re-esterified, entering the circulation as TAG contained in VLDL. Vervuert *et al.* (2005c) reported that Cr yeast did not improve glucose 'handling' and caused higher heart rates and blood lactate following a standardized exercise test. Moreover, Cr propionate (4 mg Cr/day) did not alleviate elevations in plasma leptin and insulin in horses with a high BCS (Cartmill *et al.* 2005). Horses with polysaccharide storage myopathy display abnormal insulin sensitivity, so their Cr supplementation could be counterproductive (Valberg 2005).

Jackson (1997) suggested the Cr requirement for exercising horses is greater than that for sedentary horses,

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(a)





(b)



(c)

Plate 3.2 A selenium-intoxicated horse. (a) Loss and greying of tail hair; (b) crumbly hoof below coronary band; (c) two feet showing healthy hoof wall distal to (prior to) a period of intoxication during which the wall detached from the underlying tissue. (Photographs courtesy of Sidney Ricketts.)

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but whether there is value in any Cr supplementation is unclear. The maximum tolerable concentration in rations has been set (NRC 2007) at 3000 mg/kg DM for the oxide form and 100 mg/kg DM for the chloride form of trivalent Cr.

Cobalt (Co)

The gut microflora of horses use dietary Co in the synthesis of vitamin B_{12} (cyanocobalamin) required for blood cell formation. Soils of the world deficient in Co, including those of the lower Atlantic Coastal plain and parts of New England, USA, and areas in Australia, New Zealand, East Africa, the border counties of England and Scotland and Norway, are noted for a disorder known as 'pining' in sheep and cattle. There are no known field-, or experimentally induced-cases, of either Co or of vitamin B_{12} deficiency in horses. The NRC (2007) has set a minimum dietary concentration of 0.05 mg Co/kg dietary DM for horses and a maximum tolerable dietary concentration of 25 mg Co/kg DM. Recently the EU has indicated there is a potential risk from inhalation of dust containing Co from highly concentrated premixes.

Nickel (Ni)

Nickel (Ni) is both essential for and toxic to the functioning of ruminal microorganisms, and so the dietary concentration is critical for the horse. In humans, toxicity from Ni, Cr^{VI} and Fe is mostly confined to pollution from metal industries.

Silicon (Si) and binders

Although Si is the second most abundant element in the earth's crust, its absorption is influenced by dietary Mo and aluminium (Al) and its retention by animals is sufficiently low for it to be classified as a trace element. Grains contain considerable amounts of Si (Pennington 1991). However, Si is found naturally as silica (SiO_2) which is not easily absorbed. (NB The best bio-available source of Si in human nutrition is beer! Jugdaohsingh *et al.* 2002.) It is a component of animal glycosaminoglycans, and their protein complexes. It appears to be essential for bone matrix formation and so is an important component of connective tissue, osteoblasts, hyaluronic acid and articular cartilage. It has a structural function, acting as a cross-linking element in the polysaccharide chains of the proteoglycans linked to the collagen. Collagen synthesis also requires Si for the optimal activity of proline hydroxylase (EC 1.14.11.2), and it may also be required for bone mineralization, as deficiencies in other species include abnormalities in the skull and leg bones and, independent of vitamin D-induced abnormalities, long bones have a reduced circumference, a thinner cortex and reduced flexibility.

Sodium zeolite A (SZA, $Na_2Al_2Si_2O_8.xH_2O$) is a Si source that is converted into orthosilicic acid (OSA, $Si(OH)_4$, also termed monosilicic acid) in the stomach which can be absorbed. Feeding natural zeolite ($Al_2O_3.2SiO_2.2\ H_2O$), as compared with SZA does not increase plasma Si content (Mazzella *et al.* 2005). Lang *et al.* (2001a) supplemented brood mares at foaling with 220 g Si/day, as SZA. By two months after foaling, increases in Si concentrations were found in mare's blood plasma and milk and in the foal's plasma. Si also showed a tendency to increase mare's serum **osteocalcin**. The supplementation of growing Quarter Horse foals at 6–18 months of age with SZA at the rate of 18.6 g/kg diet (Nielsen *et al.* 1993), increased plasma Si concentration and speed. It also extended work time before leg injury, reducing its frequency and delaying withdrawal from training, as greater rates of bone formation and lower rates of bone resorption , cf. controls occur in horses supplemented with SZA (Lang *et al.* 2001b). The critical period seems to be up to one year of age in Quarter Horses, as supplementation commencing at the yearling stage of growth had no physiologically important effects on running performance from 18 months of age.

Al salts can also protect against toxic metal absorption. Whether this is a factor in the observation of Nielsen *et al.* (1993) is unknown. Al and Mo can inhibit tissue accumulation of Si and inhibit bone formation by reducing osteoblastic activity, osteoid mineralization and matrix formation. Whether the Al component plays a useful part by precipitating excess P in the gut, or whether the toxicity of Al is simply exercised by inhibiting Si use is not known. It should be questioned whether an Al silicate is the most appropriate vehicle for providing Si. O'Connor *et al.* (2008) compared SZA, 200 g/day, with oligomeric OSA, 28.6 mL/day. SZA increased faecal and urinary Si excretion, Ca retention and apparent Ca digestion. It also maintained plasma Si concentrations compared with an unsupplemented diet. Supplemental OSA increased plasma Si concentrations, retention of Ca and tended to increase Si retention. Only OSA, a supplement without substantial Al, was able to significantly alter Si retention, digestibility and plasma concentration. It is therefore a viable option for Si supplementation. Oligomeric Si, free of Al sources, appears to reduce Al absorption and toxicity (Jugdaohsingh *et al.* 2000).

Zeolites are able to exchange constituent cations without a major change of structure. The inclusion of zeolite, in

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a breeding pig diet, contaminated with zearalenone, improved reproductive performance (Papaioannou *et al.* 2002), as zeolite increases faecal excretion of zearalenone and of aflatoxin B_1.

Frape *et al.* (1981, 1982) demonstrated that a cell wall component of wheat grain, the modified acid detergent fibre (MAD) extract, bound aflatoxin B_1 and greatly increased its faecal excretion. MTB-100 (Alltech, Inc., Nicholasville, Kentucky, USA), an esterified glucomannan of yeast cell wall, binds aflatoxin, deoxynivalenol, fumonisin, T-2 toxin, ochratoxin and zearalenone (Anon 2002), and clay binders bind aflatoxin. It is possible that a part of the response to zeolites is their ability to bind intestinal toxins.

Boron (B), gallium (Ga) and vanadium (V)

No equine information is available on these elements.

STUDY QUESTIONS

(1) The use and function of calcium is now well understood. What factors affect calcium adequacy and use and why is it associated with a continuing problem in horses?

(2) Most feeds contain adequate potassium and dietary sources are generally well used. Why is potassium of any concern from nutritional and physiological viewpoints?

(3) If weanlings were said to have problems with (a) mineral status, or (b) trace element status, how would you set about resolving the position?

FURTHER READING

Stephens, T.L., Potter, G.D., Gibbs, P.G. & Hood, D.M. (2004) Mineral balance in juvenile horses in race training. *Journal of Equine Veterinary Science*, **24**, 438–50.

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4
Vitamin and Water Requirements

The drink of all brute creatures being nothing but water, it is therefore the most simple … as it is the proper vehicle of all their food, and what dilates the blood and other juices, which without sufficient quantity of liquid, would soon grow thick and viscid.

W. Gibson 1726

VITAMIN REQUIREMENTS

Vitamins are nutrients that horses require in very small quantities, although the actual needs for each differ considerably. For example, the dietary requirement for niacin or for α-tocopherol (vitamin E) is 1000 times the weight of that for either vitamin D or vitamin B_{12}. However, measurements of vitamin requirements lack precision; there is little direct evidence of the requirements for any of the vitamins in the horse, and assertions are largely based on measurements in other domestic animals.

Like other mammals, horses require vitamins for normal bodily functions. These requirements will be met by vitamins naturally present in feed, supplementary sources, tissue synthesis and, in the case of vitamin K and the water-soluble B vitamins, additional amounts supplied from microbial synthesis in the intestinal tract. The tissue requirements are complicated by the synthesis of ascorbic acid from simple sugars in the horse's tissues, the production of vitamin D in the skin as a reaction to ultraviolet light, the tissue synthesis of niacin from the amino acid tryptophan, and the partial substitution of a need for choline by methionine and other sources of methyl groups.

Dietary requirements for specific vitamins are therefore affected by circumstance; for example, where horses are kept indoors or are maintained in very high northern latitudes, or indeed have highly pigmented skins and thick coats of hair, their dietary requirements for vitamin D will be greater. Young foals possess a poorly developed large intestine so are assumed not to be dependent on it for B vitamin or vitamin K synthesis. Foals grow fast and, in common with other domestic animals, their tissue requirements exceed those of adults, so their dietary needs are far greater. Tissue demands will also be greater for lactating than for barren mares, but as the former are likely to be eating more, this tends to lessen the difference per unit of feed.

Adult horses are able to draw on much larger reserves of some vitamins to see them through periods of deprivation. A good grazing season on high-quality grass can satisfy the mare's vitamin A requirement through two months of the ensuing winter. In some instances, however, old horses have a diminished ability to assimilate nutrients, particularly the fat-soluble vitamins, through a decline in digestive efficiency with age and possibly through the debilitating damage of intestinal parasites. There is also some evidence that the fertility of old barren mares benefits from larger than normal doses of vitamin A. A role for β-carotene, other than as a precursor of vitamin A, is equivocal in the horse, and indeed nothing is directly known of possible functions of the hundreds of other carotenoids in equine metabolism, although some are suspected.

Inferences drawn from other domestic animals cannot be used in estimating the effect of strenuous work on vitamin needs. It has been asserted, with some justification, that the dietary requirements for certain B vitamins involved in energy metabolism are increased for animals in heavy work, both in total and per unit of feed. This conclusion is reinforced by the frequent decline in appetite during extra hard work. The nutritional requirements of work should, however, not be confused with pharmacological responses. For example, thiamin given parenterally in single doses of 1000–2000 mg is said to have a marked sedative effect on nervous racehorses. NRC (2007)

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estimates requirements for vitamins on the basis of BW. In doing so they have transformed earlier data expressed per unit of DM intake, assuming the weight of daily intake of DM is 2% of BW for maintenance, breeding, gestation and light work, assuming daily DM intake is 2.25% BW for moderate work and 2.5% of BW for all other feeding classes.

Recommended dietary allowances for vitamins are given in Tables 4.1, 4.2 and 6.21 and a summary of deficiency signs is given in Table 4.3.

Table 4.1 Adequate concentrations of available vitamins*/kg total diet (assuming 88% DM). Compare with NRC recommendations, Table 4.2.

	Mature horse					
	Maintenance	Intense work	Mares: last 90 days' gestation, Stallions	Lactating mare	Weaned foal	Yearling
Vitamin A (iu)	1600	1600	3500	3000	3000	2500
Vitamin D (iu)	500	500	700	600	800	700
Vitamin E (mg)	50	80	60	60	70	60
Thiamin (mg)	3	4	3	4	4	3
Riboflavin (mg)	2.5	3.5	3	3.5	3.5	3
Pyridoxine (mg)	4	6	5	6	6	5
Pantothenic acid (mg)	5	10	5	8	0	5
Biotin (µg)	200	200	200	200	200	200
Folic acid (mg)	0.5	1.5	1	1	1.5	0.5
Vitamin B_{12} (µg)	0	5	0	0	15	0

*There is no evidence of a dietary requirement for vitamin K, niacin or ascorbic acid in healthy horses.

Table 4.2 Vitamin requirements of growing, working and breeding horses, based upon NRC (2007), in mg/kg BW per day, unless otherwise stated. N.B. These requirements assume positive linearity with BW.

	Vitamin A (IU)	Vitamin D (IU)	Vitamin E (IU)	Thiamin (mg)	Riboflavin (mg)
Maintenance, foals*	41	13	2.0	0.07	0.04
Growth (mg per kg ADG/d)[A]	950	2000	20	2.0	2.0
Maintenance, adults*	30	6.6	1.0	0.06	0.04
Maintenance for light exercise[A]	45	6.6	1.6	0.06	0.04
Maintenance for moderate exercise[A]	45	6.6	1.8	0.11	0.045
Maintenance for heavy exercise[A]	45	6.6	2.0	0.13	0.05
Maintenance for very heavy exercise[A]	45	6.6	2.0	0.13	0.05
Gestation, months 1–6[B]	60	6.6	1.6	0.06	0.04
Gestation, months 7–8 [B]	60	6.6	1.6	0.06	0.04
Gestation, months 9–11 [B]	60	6.6	1.6	0.06	0.04
Lactation, Foaling to 3 months[BC]	60	6.6	2.0	0.075	0.05
Lactation, 4–5 months[BC]	60	6.6	2.0	0.075	0.05
Lactation, >5 months[BC]	60	6.6	2.0	0.075	0.05
Stallion, nonbreeding	30	6.6	1.0	0.06	0.04
Stallion, breeding	45	6.6	1.6	0.06	0.04

*Maintenance amount is to ensure healthy cellular metabolism in the non-growing animal.

[A]Where appropriate growth requirement should be added to maintenance requirement, e.g. yearlings in training.

[B]Including maintenance.

[C]Requirements assume milk yield is directly and linearly proportional to BW.

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Table 4.3 Signs of advanced vitamin deficiency in the horse and pony. The status should always be kept well above that leading to these signs to provide positive benefits.

Vitamin A	Anorexia, poor growth, night blindness, keratinization of skin and cornea, increased susceptibility to respiratory infections, infertility, especially in older mares, lameness
Vitamin D	Reduced bone calcification, stiffness and abnormal gait, back pain, swollen joints, reduction in serum calcium and phosphate
Vitamin E	Pale areas of skeletal muscles and myocardium, red cell fragility, reduced phagocytic activity
Vitamin K	Extended blood-clotting time (prothrombin time), but vitamin K deficiency is rarely seen unless it is induced by drugs. Vitamin K is also required for osteocalcin function and bone health.
Thiamin	Anorexia, incoordination, dilated and hypertrophied heart, low blood thiamin and elevated blood pyruvate
Folic acid	Poor growth, lowered blood folate
Biotin	Deterioration in the quality of the hoof horn, expressed as dish-shaped walls that crumble at the lower edges so that shoe nails fail to hold

Fat-soluble vitamins

Vitamin A (retinol)

Grazing horses derive their vitamin A from the carotenoid pigments present in herbage. The principal one of these is β-carotene and fresh leafy herbage contains the equivalent of 100 000–200 000 iu vitamin A/kg DM for most domestic animals (1 iu equals 0.3 µg retinol [vitamin A alcohol]). The horse, however, seems to be relatively inefficient in the conversion of β-carotene to vitamin A, and the total carotene in good-quality grass or lucerne hay is estimated to possess only a fortieth of the value, weight for weight, of retinol. Although fresh pasture herbage would normally provide well in excess of the requirement, hay used for feeding horses in the UK provides meagre amounts of carotene and, particularly where it is more than six months old, should be considered to contribute none, unless it is visibly green. Greiwe-Crandell *et al.* (1995) found that reserves in horses deprived of pasture were depleted in about two months.

Various signs of vitamin A deficiency have been recorded, as it has several important functions, among them the integrity of epithelial tissue, normal bone development and night vision. One of the earliest signs of deficiency includes excessive lacrimation (tear production); a protracted deficiency causes impaired endometrial function in the mare. K.M. Greiwe-Crandell (personal communication) observed increased frequencies of retained placenta at parturition and lower birth weights, slower growth rates and contracted tendons in foals, following vitamin A depletion of the dam.

Figure 4.1 indicates that these clinical signs of deficiency occur under extreme conditions of deprivation. As many horses are stabled for most of their time, when they consume little or no fresh herbage, the possibility of this deprived state exists. However, few cases of overt vitamin A deficiency are recognized among stabled horses in Western countries as most routinely receive supplementary synthetic sources. There is evidence of responses in several animal species to rates of intake above the minimum requirement level (Figure 4.1) under the stress of certain chronic transmissible diseases. Some forms of infertility, particularly in elderly mares, might respond to vitamin A therapy and responses among TBs in training suffering tendon strain and lameness have been noted (Abrams 1979).

Measurement of vitamin A status from blood values

Blood plasma retinol has a relatively low concentration in horses. Butler & Blackmore (1982) give a range of 60–300 µg/L for TBs in training in the UK. This range is consistent with that given for other countries, but a marginal dietary deficiency causes little change in this. Plasma retinol is sustained by hepatic reserves and so varies only to a small extent with intake. The vitamin A status can be assessed by use of the relative dose response (RDR). For the horse this is determined in jugular blood as:

$$100(A_4 - A_0)/A_4 = \text{RDR}\%$$

where A_4 is plasma retinol concentration four hours following feeding 10 000 iu vitamin A, and A_0 is fasting plasma retinol concentration (Jarrett & Schurg 1987). A deficiency is indicated where RDR% is greater than 20%.

The response at four hours, measured as total vitamin A, represents a combination of retinyl ester, in the process

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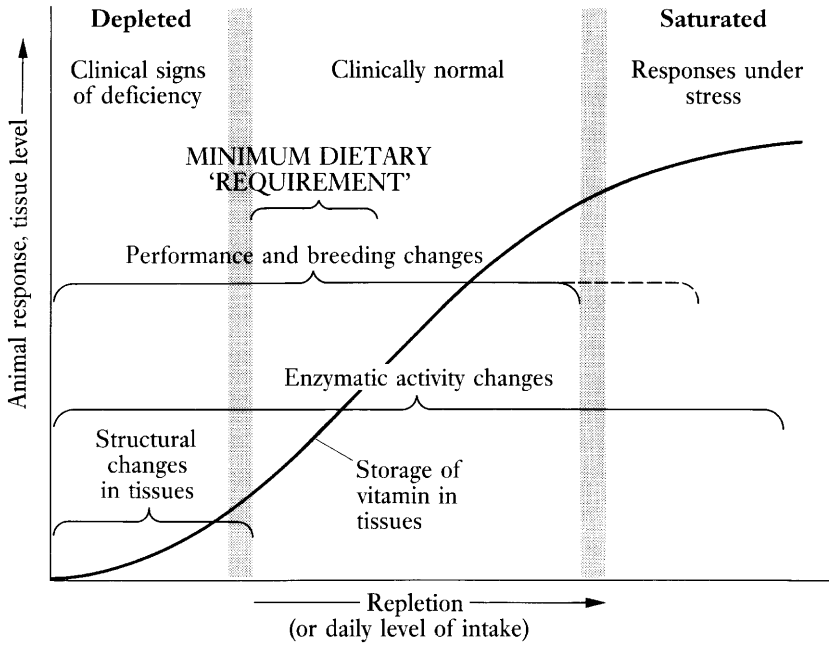


Figure 4.1 General relationship of animal response to level of vitamin intake.

of absorption, plus the subsequent release of retinol from the liver. However, the object of the assay is to measure hepatic release of retinol, which peaks at 12–15 hours post-dose of the ester. Thus, a more sensitive RDR test is obtained by measurement of plasma retinol only, by high performance liquid chromatography (HPLC), at 0 hours and at about 14–15 hours following the dose. The revised procedure employs an oral dose of 123.5 mg retinyl palmitate (224 152 iu) (Greiwe-Crandell *et al.* 1995):

$$100(A_{14} - A_0)/A_{14} = \text{RDR}\%$$

By this test a deficiency probably occurs when RDR% exceeds 10–12%.

Requirement

The synthetic forms of vitamin A are retinyl-palmitate and -acetate. These forms are more stable in the diet than retinol. These esters are hydrolysed within the lumen of the small intestine to retinol prior to absorption. One iu of vitamin A is equivalent to the biological activity of 0.550 µg of all-*trans*-retinyl palmitate, or 0.344 µg of all-*trans*-retinyl acetate.

NRC (2007) concludes that the daily vitamin A requirement for maintenance is 30 iu/kg BW; for growth it is 45 iu/kg BW; for brood mares it is 60 iu/kg BW and for exercise it is 45 iu/kg BW (Table 4.2). Most commercial feeds are supplemented with at least 10 000 iu/kg. Donoghue *et al.* (1981), however, reported that the optimum retinol intake for normal growth rate of foals was 17 µg/kg BW daily, for adequate liver-secreted serum constituents 65 µg/kg BW daily and for normal haematopoiesis 120 µg/kg BW daily. As 120 µg/kg BW is equivalent to 15 000 iu/kg feed the optimum intake is clearly not well established. Moreover, the presence of respiratory infections could increase the requirement, as these are frequently associated with a decrease in plasma vitamin A concentration. A dose rate of 120 µg/kg BW daily is equivalent to 150 000–200 000 iu daily, and, at an intake five times this, toxic reactions might occur.

Preformed vitamin A and β-carotene, in common with all other fat-soluble vitamins, are unstable, so that natural feed gradually loses potency. Synthetic forms of vitamin A are stabilized and, when undiluted and well stored, they retain more than three-quarters of their potency for several years. The grain/protein concentrate portion of the diet supplemented to the extent of 10 000 iu vitamin A/kg (3 mg retinol/kg) should allow for all foreseeable vitamin A demands (with the possible exception of haematopoiesis in foals, as indicated above). In practice, deficiencies arise from failure to supplement feed, or from the provision of badly stored old feed.

Vitamin A deficiency can also be induced by other dietary abnormalities. Evidence from several grazing species indicates that signs of deficiency can arise in stock subsisting on poor forage marginal in carotene, which, like

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much hay, contains <7% of crude protein and is deficient in zinc. Authenticated evidence for such interactions in equids is unavailable, but they probably explain the observations of Jeremiah (14:6): 'The wild asses stand on the bare heights, they pant the air like jackals; their eyes fail because there is no herbage'. Thus, stock under range conditions should also be given adequate supplementary protein and trace elements.

Vitamin A toxicosis

Vitamin A toxicity has been reported to cause bone fragility, hyperostosis, exfoliated epithelium and teratogenesis (NRC 1987) and has been implicated in DOD in growing horses (Donoghue *et al.* 1981; Kronfeld *et al.* 1990) in relation to endochondral ossification (Frape *et al.* 1959). Plasma total vitamin A concentrations greater than 400–600 µg/L are indicative of toxicosis (Lewis 1995), when retinyl-ester concentration of plasma increases relative to that of retinol (Donoghue *et al.* 1981).

β-Carotene

The plant pigment β-carotene, is the precursor of retinol; however, the pigment appears to function in the animal body independently of this precursor function. There is evidence that cows deprived of β-carotene respond to its supplementation by an improvement in fertility. However, it could simply function as an intracellular antioxidant, for in rats stressed with high intakes of corn oil, the consumption of β-carotene lowered the activity of liver superoxide dismutase. However, the corpus luteum accumulates β-carotene and 1 g/mare daily has been shown to stimulate ovarian activity in both the follicular and corpus luteum phases and to increase circulating progesterone during dioestrus. Treated mares are said to display an enhanced oestrus, an increased pregnancy rate and reduced cycling disorders. This treatment of stabled breeding mares has, in some studies, led to blood β-carotene concentrations similar to those found in pastured mares. However, problem barren mares are said to accumulate lower amounts in the blood.

A report from Edinburgh (Watson *et al.* 1996), on the other hand, indicates that water-soluble synthetic β-carotene is not absorbed by the horse as no increase in plasma β-carotene occurred with dietary supplements of either 1.8 or 10 mg/kg BW *per os* daily. Moreover, no change in cyclical ovarian activity, or in plasma progesterone concentration, was produced in that study, and poorer pregnancy and foaling rates, cf. retinyl palmitate, were observed in another study (K.M. Greiwe-Crandell personal communication). On the other hand, Kienzle *et al.* (2002) found a water-dispersible source that was effective in raising plasma β-carotene concentration.

Pasture legumes are particularly rich sources of β-carotene and these natural sources are absorbed by horses, although the equine response is relatively low. Mixed grass–clover pasture contains at least 400 mg β-carotene/kg DM during the growing season, providing many times the equivalent of the vitamin A requirement. Fonnesbeck & Symons (1967) recorded plasma concentrations of 70 µg/L β-carotene from daily intakes of 650 mg lucerne β-carotene in comparison with 40 µg/L β-carotene from 170 mg β-carotene derived from fescue grass (also see Lucerne, Chapter 5). NRC (1989) assumed that 1 mg β-carotene found naturally was equivalent to 400 iu vitamin A. There has been no conclusive evidence for NRC (2007) to change this assumption. Breeding mares deprived of pasture during the winter can become depleted of the fat-soluble vitamins A, D and E, as grass hay, or silage, is generally not an adequate source of these vitamins. Blood concentrations tend to be lowest in late winter (Mäenpää *et al.* 1988a); moreover, mares are depleted of vitamin A 2–3 months following removal from pasture, or by mid-winter in pastured mares (Greiwe-Crandell *et al.* 1995).

Vitamins D_2 (ergocalciferol) and D_3 (cholecalciferol)

Function

The widespread occurrence of bony abnormalities in horses and misunderstandings concerning the interpretation of Ca and phosphate values in blood justify a short summary of the functioning of vitamin D, which has been elegantly unravelled. Vitamin D is required for the maintenance of Ca and phosphate homeostasis, impairment of which produces the lesions in bone called rickets, or osteomalacia and the risks of lameness and bone fractures.

Metabolism

It has been realized that a fuller understanding of vitamin D function and the calcium economy of a horse requires an assay of the active form of parathyroid hormone (PTH), secreted by the parathyroid gland. Standard assays use an antibody against the carboxy-terminal- and mid-regions, and another against the amino-terminal region of the molecule (intact human PTH), or simply two antibodies against the amino-terminal region (rat assay). Estepa *et al.* (1998) reported that both assays detected a rise in PTH concentration of horse plasma when blood ionised Ca decreased, and a decline in PTH concentration with hypercalcaemia. Modulation of ionised Ca occurs as a

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consequence of the conversion, under the influence of PTH, of vitamin D to its metabolically active forms: $25(OH)D_3$, $1,25(OH)_2D_3$ and $24,25(OH)_2D_3$ (the two dihydroxy-forms are also known as calcitriol). Their well-known target organs are: intestine, kidney and bone. In addition, vitamin D influences cell growth and differentiation (Norman 2001); but these intriguing functions have not been investigated in the horse. The active forms are derived from both ergo- and chole-calciferol. The vitamin fits the classic model of a steroid hormone, but $1,25(OH)_2D_3$ is present in extremely low concentrations (55 pmol/L plasma or approximately 24 ng/L) in horses 1–30 years of age (A. Breidenbach personal communication), in comparison with human plasma (20–40 ng/L). The intermediary metabolite 25-OH-vitamin D, formed in the liver, is present in much higher concentrations (15–38 µg/L in human plasma) and is therefore more easily measured. Horse blood contains relatively low concentrations of the 25-OH metabolite. In Finland, Mäenpää *et al.* (1988b) reported winter mean concentrations of 2.14 µg/L and 8.05 µg/L, respectively, for 25(OH)vitamin D_2 and 25(OH) D_3. In summer, the respective mean values were 2.16 and 16.6 µg/L. Vitamin D_3 is synthesized in the skin when it is exposed to solar radiation with an emission spectrum of 280–315 nm. Vitamin D_2, ergocalciferol, is a poor source, derived from the action of ultraviolet light on cut leafy forage, i.e. hay.

The low equine concentration of even the 25-OH metabolite in blood, following vitamin D injections (Harmeyer *et al.* 1992), indicates that the hormone has a lesser function in the horse than in other domestic animals, where it is critical for the intestinal absorption of Ca. Nevertheless, blood plasma concentrations of Ca in the horse are higher than those in the gut lumen, so that active absorption is taking place, presumably requiring a Ca-binding protein. A vitamin D-responsive Ca-binding protein has been identified in the equine duodenum, but it seems to be of less metabolic significance than in other domestic species. It is possible that dietary vitamin D should be given in larger quantities than those recommended, as it is also essential in the prevention of dyschondroplasia. Nevertheless, toxic doses cause signs similar to those of a deficiency.

The 1,25(OH)D hormone, calcitriol, with three alcohol groups, is nearly undetectable in the horse (Harmeyer *et al.* 1992); it increases gastrointestinal Ca absorption, stimulates osteoclastic Ca resorption from bone, facilitates the effect PTH has on bone resorption, and increases renal tubular absorption of Ca. Production of calcitriol, from the 25-OH metabolite, by the cells of the proximal tubule of the nephron in the kidney, through the action of 1α-hydroxylase, is stimulated by hypocalcaemia and hypophosphataemia. The vital objective of these two hormones, together with thyrocalcitonin, is to sustain a constant level of blood Ca. It is a fascinating fact that they modulate both Ca and P nutrition, but with different signals. When the diet is deficient in Ca, but adequate in P, then a fall in plasma Ca ions triggers the release of PTH from the parathyroid gland. This stimulates renal Ca reabsorption and the production of the vitamin D hormone. Intestinal absorption and bone mobilization of both Ca and phosphate are facilitated so that blood Ca and blood pH are returned to normal. Blood phosphate does not rise because of the PTH blocking effect on the renal reabsorption of phosphate. Serum vitamin $1,25(OH)_2D$ and parathyroid hormone concentrations are higher in osteochondrotic foals than in those without, or with few, lesions (Sloet van Oldruitenborgh-Oosterbaan *et al.*1999). This might indicate that higher plasma hormone concentrations are needed to maintain plasma Ca concentration in these foals.

Thyrocalcitonin counterbalances and modulates the effect of PTH by increasing the net deposition of Ca in bone stimulated by a raised serum Ca. By contrast, deficiency of dietary P depresses blood phosphate, which in turn directly raises ionized Ca in the blood, but also stimulates production of the vitamin D hormone. The combined effect of this is to suppress PTH production, increasing phosphate retention by the kidney (negating phosphate diuresis), stimulating Ca and phosphate absorption from the small intestine. Blood Ca, however, does not rise excessively as the lack of PTH increases the urinary loss of Ca (see also Chapter 11).

When vitamin D nutrition is adequate for a given age of horse, but not so Ca or P, blood Ca is held within fairly well-defined limits and phosphate will be more variable. In the absence of vitamin D the efficiency of Ca absorption from the intestinal tract and the mobilization of bone Ca are depressed, so that blood Ca levels will fall. Some mobilization of bone Ca will continue, however, so that osteomalacia, or gradual bone decalcification, occurs in the adult horse and rickets, or reduced calcification of bones, is displayed by the young. Some authorities dispute the existence of true rickets in growing horses and vitamin D is probably of less metabolic significance than in the young pig. Nevertheless, a loss of appetite, discomfort on standing, lameness, increased risk of bony fractures and a thinning of the cortex of long bones have been described in foals deprived of sunlight and dietary vitamin D. In young horses, the growth plate (epiphyseal plate) of long bones is irregular, widened and poorly defined and the epiphyses are late in closing.

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Dietary requirement

The daily vitamin D requirement for adult horses with limited exposure to sunlight is 6.6 iu/kg BW. For growing horses between birth and 24 months of age the daily requirement declines from 22.2 iu/kg BW at birth to 13.7 iu/kg BW for the period 19 months to 24 months of age (NRC 2007, Table 4.2). If the cereal/protein concentrate component of the diet is supplemented with 1500 iu vitamin D/kg (37.5 µg cholecalciferol or ergocalciferol), then the daily requirement for vitamin D should be met. Moderately large doses of vitamin D can, to some extent, compensate for low dietary Ca by promoting further Ca absorption, particularly where dietary P is in excess. However, large doses of vitamin D (in excess of 2000–3000 iu/kg BW daily, or 60 000–100 000 iu/kg diet) will cause similar signs to those of deficiency and eventual death, owing to the effect of vitamin D hormone on bone mineral mobilization and a consequential calcification of soft tissue.

Natural sources of toxicity

The horse seems to differ from other species during vitamin D intoxication, in that plasma Ca concentration does not increase, but that of inorganic P does (Harmeyer & Schlumbohm 2004). Several plant species, not found in the UK, actually synthesize this highly active hormone as the calcitropic principal, $1,25(OH)_2D_3$-glycoside. (e.g. *Cestrum diurnum*, a member of the potato family, sometimes incorrectly called wild jasmine, found in Florida and other subtropical US states, including Texas and California). Thus, horses grazing areas where the plants exist will develop rickets and soft-tissue calcification.

Vitamin E (α-tocopherol)

Vitamin E donates a hydrogen atom from its phenolic group to lipid peroxyl radicals produced during auto-oxidation of membrane polyunsaturated fatty acids, forming a more stable lipid peroxide and stable tocopheryl radical. The lipid peroxides are further degraded by Se-dependent glutathione peroxidase. Potency is possessed by several tocopherol and tocotrienol isomers and undoubtedly all these have some antioxidant value in the feed and in the GI tract. Analysis has shown that α-tocopherol is the only isomer to be found in significant amounts in equine tissues and so is the only form to have significant vitamin potency. Natural forms of vitamin E (D-α-tocopherol and its acetate salt) have a somewhat greater bioavailability in the horse than the racemic mixture of synthetic forms (DL-α-tocopherol and its acetate salt) and free alcohols are superior to the salts (Hargreaves *et al.* 2001). Nevertheless, there is evidence (Christen *et al.* 1997) that α-tocopherol displays an active metabolic role in several species.

As tocopherols have an antioxidant property, they are themselves destroyed by oxidation. This is accelerated by poor feed storage, mould damage and by ensilage of forage or the preservation of cereals in moist conditions. Müller *et al.* (2007) measured the effect of degree of fermentation and wilting time on the α-tocopherol and β-carotene contents of grass silage and haylage. The DM contents were 300 g DM/kg, 500 g DM/kg and 600 g DM/kg. The α-tocopherol contents ranged from 0.39 to 0.60 of the initial contents and those of the β-carotene ranged from 0.33 to 0.86 of the initial contents. It was apparent that good conservation, as measured by a high lactic acid fermentation, favoured the preservation of both α-tocopherol and β-carotene. After the crushing of oats or grinding of cereals, the fats are more rapidly oxidized and vitamin E is gradually destroyed unless the material is pelleted. Fresh, green forage and the germ of cereal grains are rich sources of vitamin E, but feeds are frequently supplemented today with the relatively stable acetate ester. (See 'Selenium', Chapter 3, for effects of vitamin E/selenium supplements.)

Vitamin E status

Evaluation of the vitamin E status of horses is problematic, owing to a relatively low normal plasma α-tocopherol concentration. The normal range is 1.5–5 mg/L. Adipose tissue of horses contains large quantities (10–60 µg/g) of α-tocopherol that are not prone to the short-term fluctuations characteristic of blood levels. TBs tend to have plasma and adipose tissue concentrations at the lower end of these ranges and overall storage is lower than that for vitamin A. A vitamin E deficient adult female Quarter Horse suffered acute heart failure and sudden death (Barigye *et al.* 2007). Multiple pale and firm foci in the heart were consistent with severe myocardial degeneration/necrosis and fibrosis. A suboptimal hepatic vitamin E was noted, whereas hepatic Se concentration was normal. There was no anamnestic indication for ingestion of cardiotoxic plants or evidence of other aetiologic factors.

Vitamin E, ascorbic acid and exercise

Vitamin E adequacy has been measured for many years as the dietary amount required to minimize erythrocyte haemolysis in the presence of dialuric acid, hydrogen peroxide or other haemolytic agents. Early studies (NRC 1978) indicated that the horse required only 10–15 mg/kg diet to

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ensure this. A vitamin E deficiency is known to reduce endurance in rats and the vitamin is particularly important for extended work with a higher red cell GSH-Px activity following exercise (Ji *et al.* 1990).

Ronéus *et al.* (1986) found that to provide adequate Standardbred tissue saturation with α-tocopherol the daily supplement of DL-α-tocopheryl acetate (all-*rac*-α-tocopheryl acetate) should be 600–1800 mg. This is equivalent to 1.5–4.4 mg/kg BW. The question that arises then: Is it necessary to saturate tissues with the vitamin? α-Tocopherol forms an integral part of cellular membranes, where it protects polyunsaturated fatty acids. The peroxidation of these ω-6 fatty acids probably increases during exercise – frequently measured as an increase in thiobarbituric acid reactive substances (TBARs) – yielding *n*-pentane, a hydrocarbon gas that is excreted in the breath, although an assessment of breath pentane following exercise is complicated by an associated increase in breathing rate. McMeniman & Hintz (1992) recorded an increase in plasma ascorbic acid and a tendency to a decrease in plasma TBARs in exercised ponies supplemented with additional vitamin E. However, the additional amount was only 100 iu/day and very few animals were used. Schubert (1990) reported that quite large supplementary levels of vitamin E improved the track performance of racehorses. A supplement of 240 iu vitamin E/kg feed plus 10 g/day ascorbic acid has led to higher blood levels of both α-tocopherol and ascorbic acid in heavily used polo ponies late in the competition season (Hoffman *et al.* 2001c). Amongst endurance horses, a variable vitamin E intake of 1150–4700 mg/day during the weeks prior to an 80 km ride resulted in a positive correlation of intake with plasma α-tocopherol concentration, and a negative correlation of intake with plasma CK and aspartate aminotransferase (AST) activities during the ride (Williams *et al.* 2003b). These data indicate that the mean of 5 mg vitamin E/kg BW for hard exercise is inadequate. Moreover, it has been proposed that oxidant/antioxidant imbalance plays a decisive role in the pathogenesis of chronic inflammatory airway diseases. A supplement of vitamin E, ascorbic acid and selenium (Winergy Ventilate™, Winergy, Pedigree Masterfoods, Melton Mowbray, Leicestershire, UK) improved exercise tolerance and reduced pulmonary inflammation in heaves-affected horses in remission at rest and after a standard exercise test (Kirschvink *et al.* 2002). de Moffarts *et al.* (2005) measured the markers of antioxidant status during training in TBs given oats and hay with and without a supplement of 11 500 mg ascorbic acid, 7000 mg α-tocopherol acetate, 500 mg β-carotene, 187 mg Cu, 769 mg Zn, and 7 mg Se. An antioxidant imbalance was observed after 3 months in the control group measured as decreases in GSH, superoxide dismutase (SOD), GSH-Px, Se and increases in oxidised glutathione (GSSG). Hence, the vitamin E requirement of working horses requires further assessment and it could be up to 300 mg/kg dietary DM (8–9 mg/kg BW daily) cf. that proposed by NRC (2007) (Table 4.2).

Fat supplementation (see Chapter 5)

There is increasing interest in fat supplementation of diet for exercising horses (see Chapter 9). Many vegetable and fish oils, such as corn, soya or cod liver oil, are rich in polyunsaturated fatty acids and oil supplementation of other species is known to increase the vitamin E requirement. In the horse muscle, TBARs have been shown to rise following this supplementation, despite the naturally high concentration of vitamin E in fresh corn or soya oil and the addition of antioxidants during manufacture of the oils. Clearly, stale, badly stored oils should not be used.

Breeding mares

In conditions where the intake of selenium and vitamin E is low, which can occur on pasture, mares give birth to foals suffering from myodegeneration. Pale areas in the myocardium and skeletal muscles are apparent on *post-mortem* examination and muscle-cell damage is seen histologically. If the foals survive, damage is said to be irreversible. Other signs include steatitis, or yellowing of the body fat, and general fat necrosis with multiple small haemorrhages in fatty tissues. As with other causes of muscle damage, the activity of blood CK (EC 2.7.3.2) and AST (EC 2.6.1.1) rises and probably the fragility of red blood cells increases.

Immune function

Vitamin E is required for normal immune function. Baalsrud & Øvernes (1986) reported that a vitamin E supplement given to oat-fed horses increased their humoral immune response to tetanus toxoid and equine influenza virus. An increase in the phagocytic activity of foal neutrophils has been induced by supplementation. Mares given a traditional mix supplemented with 160 iu vitamin E/kg mix, cf. 80 iu/kg, from four weeks before foaling, tended to have higher serum and colostrum concentrations of immunoglobulins, leading to somewhat higher serum IgG and IgA concentrations in their suckled foals (Hoffman *et al.* 1999b). Work is required in relation to neonatal infections.

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Vitamin E in prevention of equine degenerative myeloencephalopathy and equine motor neuron disease

Two neurological disorders of horses have been recognized to involve α-tocopherol status, equine degenerative myeloencephalopathy (EDM) and equine motor neuron disease (EMND). Oxidative injury of myelinated nerve fibres occurs in both diseases. The sheaths of these fibres are rich in unsaturated fat, normally protected by α-tocopherol.

EMND is a neurodegenerative disorder of the somatic lower motor neurons affecting horses 2 years of age and older. Evidence indicates that EMND occurs following a prolonged period of vitamin E deficiency, when serum α-tocopherol concentration is frequently <1 μg/mL (Divers 2005). EMND is typified by weight loss, despite normal or increased appetite, and increased recumbency and trembling of proximal limb musculature (Mayhew 1994). The lifting of one thoracic limb from the ground for a few minutes may induce trembling, especially in proximal limb muscles of the opposing thoracic limb. There is a typical base-narrow stance of the legs and tucked-up abdomen. Other signs include hyperaesthesia (hypersensitive response to stimulation) and low head and neck carriage. Affected horses move better than they stand with a stiff rather than ataxic gait. The clinical signs and neuronal lesions bear some resemblance to the clinical signs and lesions present in autonomic neurons of equine grass sickness (see Chapter 10), apart from the normal feed intake of EMND patients, many of which show abnormal glucose tolerance (Wunberg 2006). A rapidly progressive muscular atrophy, is present in the chronic form of EMND and the tail head is frequently held in a raised position, as neurogenic atrophy from oxidative damage to the somatic ventral motor neuron cells causes contracture of muscle. Abnormal brown pigment deposits occur in the retinal fundus. Serum ferritin and hepatic Fe (Divers *et al.* 2001; Verhulst *et al.* 2001) and spinal cord Fe and Cu (Syria *et al.* 2006) concentrations are frequently elevated, accounting for oxidative stress in the absence of vitamin E deficiency. In eight experimental horses confined to a dirt lot and given a concentrate low in vitamin E and high in Fe and Cu plus 1-year-old grass hay (Divers *et al.* 2006), four developed clinical signs of EMND. Plasma vitamin E decreased in all horses, without changes in plasma concentrations of vitamin A, Se or Cu, or serum concentrations of ferritin cf. controls. The authors concluded that a lack of access to pasture, dietary deficiency of vitamin E, or excessive dietary Cu is a likely risk factor for EMND. A supplement of vitamin E for healthy horses led to increased serum and CSF α-tocopherol concentrations (Higgins *et al.* 2008).

In common with EDM, EMND is typically associated with an absence of access to pasture, with the consumption of poor quality hay and with low concentrations of circulating α-tocopherol. EMND seems to prevail among adult horses of a wide age distribution. Divers *et al.* (1994, 2001) recorded EMND in 28 horses, ranging in age from 3 to 18 years, with the highest frequency among 4–9-year-olds. Mayhew (1994) considered that EMND might require a neurotoxin to initiate oxidative neuronal degeneration in a pre-existing vitamin E deficient state of the adult (also see Grass sickness, Chapter 10). McGorum *et al.* (2006) diagnosed EMND in three horses with low vitamin E status, maintained on lush grass-based pasture. Green forage and vitamin E supplementation at 2000 iu/day are required when access to green forage/pasture is limited. Standard preparations that include Se would normally lead to selenium toxicosis if the dose provides the required amount of vitamin E.

EDM usually develops in growing horses and is possibly more prevalent in some sire lines. EDM is a diffuse degenerative disease of the spinal cord and caudal medulla oblongata of equids. The disease arises in foals where breeding horses have no access to pasture, or where large numbers are crowded on poorly productive, often dry, pasture and are given poor sun-baked hay. The clinical signs include an abrupt to insidious, onset of symmetric paresis, ataxia and dysmetria. Stiffness of the limbs of foals and yearlings may persist to adulthood. The signs are present particularly in the pelvic limbs, but also in the thoracic. In the author's experience, EDM has been associated with absence of pasture and the presence of DOD (see Chapter 8), and signs were not expressed until three to five months of age. Older horses exhibit a striking hyporeflexia over the neck and trunk. Histologically, neuroaxonal dystrophy is widespread, and neuronal atrophy, axonal swellings (spheroids), astrogliosis and lipofuscin-like pigments are prominent in older horses. The signs should not be confused with those of infections, such as those of West Nile virus infection (Cantile *et al.* 2000), including ataxia, weakness and paresis of the hind limbs, occasionally progressing to tetraplegia and recumbency. In EDM, plasma α-tocopherol concentrations range from 1–1.5 mg/L. EDM is vitamin E responsive, if it has not progressed too far, and then plasma levels rise to 2 mg/L. Se does not seem to be involved.

For breeding horses without access to pasture, the daily vitamin E supplementation should be high – 2000 iu daily for breeding mares. For those presenting signs of ataxia,

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Table 4.4 Summary of proposed daily treatment for a horse presenting EDM.

Vitamin E	2000–6000 iu
Rapeseed oil[1]	30–200 mL
Clean, mould-free carrots	2 kg
Dried lucerne[2]	2 kg
Riboflavin	12 mg/kg concentrate
Coarse mix diluted with molassed chaff	To appetite, given *ad libitum* to growing weanlings

[1]If fish oil is used, the minimum vitamin E dose should be 4000 iu/day.

[2]A good source of riboflavin, although the additional 12 mg is an insurance.

6000 iu DL-α-tocopheryl acetate/horse daily should be mixed in 30 mL vegetable oil (see below) and added to 1 kg freshly ground cereal on a daily basis (not stored). Little response is likely for three weeks. Once improvement has been achieved the supplementary dose is slowly reduced to 2000 iu/day. Proof of absorption should be sought by α-tocopherol determinations on blood.

The lipids of plant leaves are much richer in *n*-3 polyunsaturated fatty acids than are those of seeds. The author suggests that the cause of EDM could be a combination of deficiencies of natural antioxidants and of *n*-3 polyunsaturated fatty acids, although this suggestion would need testing before recommendations are advanced. Fish oils are rich sources of the higher *n*-3 fatty acids. As an intermediary step, vegetable oils, such as rapeseed oil, richer in the lower members of the *n*-3 series than corn oil, might be used as the carrier for vitamin E; but the dose of oil would have to be much greater than 30 mL – say 200 mL daily. Natural antioxidants include the carotenoids. The author has found that control of EDM is assisted by providing a source of these carotenoids, such as mould-free carrots, or dehydrated lucerne, and the riboflavin supplementation of a concentrate mix should be increased to 12 mg/kg (Table 4.4).

It is commented on above that DOD seems to be present in some EDM cases. To this end, as discussed in Chapter 8, the author has found that the *ad libitum* feeding of growing foals and yearlings is helpful. Most published evidence suggests that this aggravates the condition; however, in several studies including Savage *et al.* (1993a, b) meal-fed and *ad libitum*-fed horses have been compared when given the same concentrated feed, whereas the author's experience indicates that the feed for *ad libitum* feeding should be a coarse mix or a small pellet diluted with molassed chaff, so that the amount taken in at each 'sitting' provides a relatively small amount of energy, akin to nibbling.

Recommended dietary allowance for normally managed horses

At very high intakes of vitamin E coagulopathy and impaired bone mineralisation have been reported (NRC 1987). So, NRC (2007) has retained an upper safe limit for dietary vitamin E of 1000 iu/kg DM (1 iu is equal to 1 mg DL-α-tocopheryl acetate, i.e. all-*rac*-α-tocopheryl acetate). However, Fiorellino *et al.* (2009) found differences in the efficiency of absorption and retention of an elevated blood level amongst five forms of vitamin E alcohol and acetate, hence influencing an optimum dietary concentration. Moreover, the amount of vitamin E needed in the diet rises in proportion to the level of dietary unsaturated fats (Agricultural Research Council 1981). NRC (2007) has retained earlier estimates of the dietary requirements for vitamin E (NRC 1989). It was recognized that the requirement for optimum humoral immune function is greater than that to maintain erythrocyte stability: for maintenance 50 iu/kg DM, or 1 iu/kg BW daily; for lactation: 2 iu/kg BW daily, based upon the evidence that mares receiving this amount, cf. 1 iu/kg BW, had a greater IgG concentration in their colostrum (Hoffman *et al.* 1999b) (Table 4.2). Larger amounts are proposed for work as exercise induces lipid oxidation in horses (Williams 2004a,b). The status of horses performing rigorous endurance exercise was improved by amounts of supplementary vitamin E exceeding existing estimates (approximately 6 iu/kg BW daily, Hoffman *et al.* 2001c; 11.1 iu/kg BW daily, Williams 2004b). Siciliano *et al.* (1997) reported a decline in both serum and middle gluteal muscle vitamin E concentration over a 90-day exercise conditioning period when horses were given up to 1.6 iu/kg BW daily, but not when they were given 6 iu/kg BW daily. Vitamin E supplementation of 11.1 iu/kg BW daily decreased white blood cell apoptosis and plasma CK activity during and after a treadmill-simulated 55-km endurance race (Williams 2004b).

Vitamin K (phylloquinone)

Vitamin K serves as a cofactor for vitamin K-dependent carboxylase, which catalyzes the post-translational synthesis of γ-carboxyglutamic acid (Gla) from glutamic acid residues contained in at least a dozen precursor proteins. The resulting vitamin K-dependent proteins, also referred

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to as Gla-proteins, are involved in blood clotting, bone metabolism and vascular health (Vermeer *et al.* 2004; NRC 2007). A deficiency of vitamin K results in the presence of under-carboxylated Gla-proteins, e.g. under-carboxylated osteocalcin and impaired blood coagulation. Carboxylation allows osteocalcin to bind hydroxyapatite in bone formation. Vitamin K antagonists such as dicumarol, produced by mouldy sweet clover hay, and other coumarin derivatives, such as warfarin, can impair vitamin K metabolism, causing signs of deficiency.

There are two natural sources of vitamin K. Green plants synthesise phylloquinones (phytylmenaquinone, K_1) and bacteria synthesise menaquinones (K_2). Menadione (K_3) is a synthetic form used in supplements that is metabolised in the body to an active form, i.e. menaquinone-4. Vitamin K_2, prenylmenaquinone-n, along with the B vitamins, is synthesized by functioning gut microorganisms in amounts that should normally meet the horse's requirements. However, this source is inadequate during the first couple of postnatal weeks, or during extended treatment with sulphonamides.

There is some tissue storage of vitamin K, and, owing to intestinal synthesis and that present in green forages supplementation is unnecessary. No correlation between serum osteocalcin, or its binding capacity, with bone density or health was observed in horses given brome grass hay (2.73 g phylloquinone per kg) and a grain-mix (0.4 g phylloquinone per kg) (Siciliano *et al.* 2000a).

Airway haemorrhages in racehorses are an expression of blood vessel fragility and not one of a failure in the clotting mechanism. These are unrelated to vitamin K status, expressed as hydroxyapatite-binding capacity for serum osteocalcin (Siciliano *et al.* 1999a).

The NRC (1987) proposed that oral toxic levels are at least 1000 times the dietary requirement. However, NRC (2007) states that menadione bisulphite, given by injection in single doses of as little as 2.1 mg/kg BW, could cause ill-health and that phylloquinone injectables are safer. The dietary requirement for vitamin K is unknown, as the plasma concentration of under-carboxylated osteocalcin has not been influenced by dietary supplementation. Binding capacity of serum osteocalcin (with lower serum osteocalcin concentrations) increases with age in foals and with earlier weaning, probably reflecting increases in forage intake and in the capacity of the intestinal flora to synthesise vitamin K_2 (Siciliano *et al.* 1999b, 2000b).

Water-soluble vitamins

Normal intestinal synthesis plus the quantities naturally present in typical horse feeds seem to meet the maintenance requirements for the B vitamins riboflavin, niacin, pantothenic acid, pyridoxine and B_{12} (Carroll *et al.* 1949). Should there be a basic change in diet towards root vegetables and certain by-products in the future, then an increase in supplementary needs might result. The needs of lactating mares and weanling foals should be met if good-quality pasture grass is provided. Additional nutrient demands of exercise are discussed in Chapter 9.

Thiamin

Thiamin is required by pyruvate dehydrogenase, α-ketoglutarate dehydrogenase and transketolase, all of which are involved in carbohydrate metabolism. Thiamin is found in cereal grains (3–6 mg/kg DM) and in high concentrations in brewer's yeast (95 mg/kg DM).The classical deficiency signs are beriberi. These signs have been inferred, to a large extent, from studies in other species. However, a deficiency (Carroll *et al.* 1949, Cymbaluk *et al.* 1978) has been shown to cause anorexia, a dilated and hypertrophic heart, bradycardia, muscle fasciculations, ataxia, a decline in blood concentration of thiamin and in the activity of enzymes requiring thiamin as a cofactor. Cymbaluk *et al.* (1978) made four Standardbred horses thiamin-deficient by the daily oral administration of 400–800 mg amprolium/kg BW (the coccidiostat, amprolium, is a structural analogue of thiamin). After one to two months, thiamin deficiency signs of bradycardia, ataxia, muscular fasciculations and periodic hypothermia of extremities, blindness, diarrhoea and weight loss were observed, and erythrocyte transketolase activity was depressed.

Table 4.5 gives normal values for blood thiamin levels. Animals grazing on heathland infested with bracken fern (*Pteridium aquilinum*) can exhibit signs of thiamin deficiency if they eat the bracken. Treatment with thiamin is usually effective. About 25% of free thiamin synthesized in the caecum is absorbed into the blood. Working horses require a higher concentration of dietary thiamin than that given for maintenance, based upon blood thiamin concentrations and pyruvate dehydrogenase activities of working horses (Topliff *et al.* 1981). NRC (2007) has retained the previous estimate for thiamin of 5 mg/kg dietary DM for working horses and 3 mg/kg DM for all other classes. These values are now expressed as mg/kg BW (Table 4.2).

Vitamin B_{12} (cyanocobalamin)

The cyanocobalamin molecule contains the element cobalt (Co). Cattle and sheep grazing areas deficient in this element develop vitamin B_{12} deficiency, as the rumen microorganisms are then unable to manufacture the

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Table 4.5 Normal blood thiamin concentrations (μg/L) in Standardbred horses (Loew & Bettany 1973).

Stallions	2.25 ± 0.16
Geldings	3.03 ± 0.13
Mares and fillies	3.36 ± 0.11
Less than 1 year	2.42 ± 0.26
1–4 years	2.81 ± 0.10
5–10 years	3.53 ± 0.14
10–20 years	3.35 ± 0.17

vitamin. Co therapy rectifies the situation. Horses seem to be more resistant to Co deficiency, but undoubtedly they require Co at a minimum level of about 0.1 mg/kg diet. Supplemental Co raises the vitamin B_{12} status of horses (Alexander & Davies 1969; Stillions *et al.* 1971b), as B_{12} is absorbed from the caecum and colon (Stillions *et al.* 1971b). Synthesis in foals is inadequate, but colostrum contributes to the status of the neonatal foal (Caple *et al.* 1982). Much of the vitamin is stored in the liver, but a supplement of vitamin B_{12} can increase its blood concentration. It is required for cell replication and a deficiency may cause a reduction in the number of red blood cells. Macrocytic anaemia is common to both B_{12} and folic acid deficiencies through a limitation of DNA synthesis.

Although an overt deficiency has not been produced in adult horses, receiving adequate Co in their diet, it has been suggested that a response, including a stimulation to appetite, can be obtained in some anaemic animals. Adult horses in training on high-grain rations might need supplementation if they show a decline in appetite which could reflects a build-up of blood propionate. This VFA is produced proportionately and absolutely in much greater quantities when grain diets are consumed, and its metabolism to succinate requires methylmalonyl-CoA mutase (EC 5.4.99.2) which has adenosylcobalamin as a coenzyme. Vitamin B_{12} deficiency causes an elevation in urine of both methylmalonic acid and homocysteine which distinguishes it from folic-acid deficiency. Early-weaned foals should receive a supplement of 10 μg vitamin B_{12}/kg dietary DM.

Folic acid (pteroylglutamic acid)

Folate is a generic term referring to folic acid and naturally occurring folate (Bailey *et al.* 2001) which occur as conjugates of *p*-aminobenzoic acid (PABA) with five to eight glutamic acid residues joined in γ-peptide linkages, i.e. polyglutamate forms. Folate is required for the transfer of single carbon units in DNA, purine and methionine synthesis, and so folate is particularly important for tissues in which cell replication is occurring (Bailey *et al.* 2001). Both the stability and the availability to the horse of these compounds undoubtedly vary, but there is a scarcity of evidence. Good quality forages, especially in a fresh form, are better sources of folate than cereal grains. Orally administered folic acid may be poorly utilized by the horse (Allen 1984), but it is synthesized by horse gut microorganisms (Carroll *et al.* 1949).

Signs of equine folate deficiency have not been described. In other species megaloblastic anaemia and leukopenia are common. In addition tissues having a rapid rate of cell replication, or tissue regeneration, such as the epithelial lining of the gastrointestinal tract, epidermis and bone marrow are affected (McDowell 1989).

Antimicrobials used to treat equine protozoal myeloencephalitis, sulphadiazine and pyrimethamine, can impair folate status. Synthesis of folate by gut bacteria from PABA is blocked by sulphanilamide, which has a structure similar to PABA and which combines with the bacterial enzyme, dihydropteroate synthetase, required for folic acid synthesis. Thus, enzyme action is compromised by inhibiting the formation of a normal enzyme–substrate complex. Signs caused by these drugs can include haematological defects, hypoplastic bone marrow and dysphagia, resulting from oral ulceration and **glossitis**. Folic acid supplementation is not recommended for horses treated with dihydrofolate reductase inhibitors, e.g. pyrimethamine (Piercy *et al.* 2002). A Quarter Horse mare with suspected equine protozoal myeloencephalitis (EPM) was treated orally for nine months with sulfadiazine, pyrimethamine and 19 mg folic acid daily (Piercy *et al.* 2002). She presented with dysphagia, glossitis, neurological abnormalities, anaemia, leukopenia, hypoplastic bone marrow and hypofolic acidaemia. The abnormalities resolved only after the oral administrations were discontinued and replaced by intravenous administration of folic acid and oral treatment with diclazuril. The folic acid deficiency, induced by prolonged administration of folic acid inhibitors, was exacerbated by oral administration of oxidised folic acid.

The forms of folic acid vary depending on the addition of one-carbon units, the degree of conjugation by glutamyl groups and the degree to which the molecule is reduced by dihydrofolate reductase (to di- and tetrahydrofolate). Folic acid in plants exists in polyglutamated forms and prior to absorption it must be deconjugated by enzymes located in the brush border. Passive and active absorption

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occur, but for active absorption folate must be in a *reduced* form, not the form usually found in supplements. The fully oxidised form requires reduction by dihydrofolate reductase prior to its active absorption. Thus, frequently, folinic acid, a fully reduced form, is administered. Intestinal folic acid, when in the oxidised form, decreases absorption of the biologically active methyltetrahydrofolate, because the former's affinity for intestinal folate-binding protein is approximately six times greater than that of the latter. Moreover, the combined effect of oxidised folate and an inhibitor of dihydrofolate reductase (pyrimethamine) further intensifies the inhibition of reduced folate absorption. (See 'Folic acid', Chapter 12). This conclusion probably explains the observation of Ordakowski *et al.* (2002) that horses treated with anti-folate drugs were refractory to the co-administration of natural and synthetic folates.

Folic acid is closely associated with vitamin B_{12} in single-carbon metabolism. In some domesticated animals, a deficiency causes macrocytic anaemia, through impairment of methionine synthetase (EC 2.1.1.13) function, and so depresses DNA and RNA synthesis. In folic-acid deficient humans and horses (Ordakowski *et al.* 2002), elevated concentrations of homocysteine (but not of methylmalonic acid) occur in blood and urine. Supplementation of stabled TBs receiving an inadequate diet produces an increase in serum folate concentration from about 4 to 9 µg/L and where blood haemoglobin is low the level might be raised. Australian work on folic acid in the horse supports the author's observations suggesting that there is an increased utilization of folic acid by horses in hard work. Green forage legumes are rich sources of the vitamin, but its availability in some sources is low. As horses required to partake in intensive exercise tend to receive less green forage it is recommended that they receive supplementary folic acid, or green forage in their diet. Folic acid supplementation (25 mg/day, 5 days/week) had no effect on the folic acid status, or submaximal exercise assessment, of adult horses managed on pasture and given hay and a commercial concentrate (Ordakowski *et al.* 2003). Serum folate concentrations are lower in horses deprived of pasture (Roberts 1983). Ordakowski-Burk *et al.* (2005) concluded that folate supplementation of mares with access to pasture through the breeding cycle was unnecessary as they received 30–80 mg folate/day from natural sources. Ordakowski *et al.* (2001), found 6.5 mg folic acid/kg DM in an April pasture, at foaling, declining to 2.2 mg/kg DM in June. Mean plasma concentration in TB mares, given supplements containing 1.6 or 1.9 mg folic acid/kg, was 20.6 ng/mL at foaling declining to 17 ng/mL at 3–6 months of lactation. The mare's milk contained approximately 10 times the level in their serum, i.e. 218 ng/mL at foaling declining to 147–196 ng/mL during 3–6 months of lactation (foal serum levels varied with their dam's status). A daily supplement of 2–3 mg folic acid for weaned foals and working horses is appropriate (Table 4.1).

Biotin

Biotin is a co-enzyme for four carboxylase enzymes: acetyl-CoA carboxylase, pyruvate carboxylase, propionyl-CoA carboxylase and β-methylcrotonyl-CoA carboxylase, which are involved in fatty acid, cholesterol and amino acid metabolism (Zempleni 2001) and is essential in cell proliferation. There are eight possible isomers of biotin, of which only *d*-biotin has vitamin activity (McDowell 1989). High concentrations of active vitamin occur in alfalfa and intermediate to low concentrations in grains and soya. Most naturally occurring biotin exists bound to a protein making availability dependent on the digestibility of the specific binding proteins (Baker 1995). Biotin supplementation is the only water-soluble vitamin supplement to have brought about clinically observable responses in otherwise healthy horses. This could have occurred because the biotin contained in wheat, barley and milo (sorghum) grains and in rice bran is bound and almost completely unavailable for utilization. That contained in oats is only slightly more digestible. However, all the biotin in maize, yeast and soyabean is accessible, together with most of that in grass and clover foliage. Biotin is synthesised by the gut microflora (Carroll *et al.* 1949).

Deficiency signs include severe dermatitis in other animals. In the horse Kempson (1987) identified two types of hoof defect common in horses: the first is characterized by a loss of structure and horn in the stratum externum of the hoof wall, responsive to biotin supplementation, and the second by a loss of tubular structure in the inner layers of the hoof wall that may result from protein and calcium deficiency. Kempson (1987) reported that biotin did not overcome the second hoof defect of poor attachment of horn keratin squames. Supplementary limestone (7.5 g/day) with biotin improved this defect. Nevertheless, it is possible that this additional response resulted from the more extended use of the biotin. Horn is primarily composed of protein, rich in sulphur-containing amino acids, and many supplements contain methionine, or methyl sulphonyl methane (MSM), as a source of biologically available sulphur (see 'Dietary vitamin and mineral supplements, Chapter 5), in addition to biotin, which is an asset where a low-protein diet is given. Kempson (1987) also noted that infection of the keratin squames by Bacteroides

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nodosus is responsive to metronidazole, although these organisms are probably secondary invaders.

Slater & Hood (1997) reported that 28% of the horses in their Texas survey had some type of hoof wall problem. Many cases of weak, misshapen, cracked and crumbly hooves, that tend to separate from the sole, have responded to supplementation with biotin. A greater response, as measured by horn hardness, tensile strength and possibly growth rate, has been evoked with 15 mg/horse daily than with 7.5 mg (Buffa *et al.* 1992) (Plate 4.1, a and b). Hoof horn and capsule growth rates were also increased over a period of five months when a high-fibre pony cube, containing 100 g biotin/kg, was supplemented with 8 mg biotin/kg feed (0.12 mg/kg BW daily) (Reilly *et al.* 1999). These supplementary amounts are considerably larger than should normally be adequate as a daily maintenance dose, but no definitive dietary requirement has been determined.

It is also essential that the hooves are properly shaped and trimmed. Long, unpared hooves exert excessive pressure on the heels and this restricts blood flow and hence impedes adequate nutrition of the foot, leading to poor quality and crumbly, unsatisfactory growth of walls, sole and frog. The hoof wall grows linearly from the coronary band at the rate of 8–10 mm/month, so that 9–12 months are required for the wall of the toe to grow from the proximal to the distal border (Pollitt 1990) and at least 10–12 months are required for an adequate response from a horse to a dose of 2–5 mg/d supplementary biotin (the sole and the frog are replaced every two months). Comprehensive evidence indicates that a dose level of 20 mg/day might be required by large horses, for up to three years, for the maximum benefit to be wrought (Josseck *et al.* 1995; Zenker *et al.* 1995). Plasma biotin concentrations of 350 ng/L were found in control, untreated horses and 1000 ng/L in treated horses (Josseck *et al.* 1995).

Riboflavin, niacin, pantothenic acid, pyridoxine, choline and lipoic acid

No evidence of a dietary deficiency of riboflavin, niacin, pantothenic acid, pyridoxine and lipoic acid has been established, owing presumably to adequate biosynthesis by the gut flora. Whether supplementation is of benefit in special circumstances, e.g. ill-health or extreme exertion, has also not been established. Thiamin, as thiamin pyrophosphate (TPP), niacin, as nicotinamide adenine dinucleotide (NAD), riboflavin, as flavoproteins, and lipoic acid, all function in the tricarboxylic acid (TCA) cycle for the aerobic combustion of acetate to CO_2 and water with the production of energy, eventually provided to muscle cells as high-energy phosphate in ATP.

Niacin is the generic term for nicotinic acid and nicotinamide, which are equivalent in their vitamin activities per mole. Niacin is essential for the coenzyme nicotinamide adenine dinucleotide (NAD) and for NAD-phosphate (NADP), involved in many biological oxidation–reduction reactions. Natural sources are hydrolysed in the intestinal mucosa yielding nicotinamide. NAD provides the substrate for three classes of enzymes that transfer adenosine diphosphate (ADP)-ribose units to proteins involved in DNA processing, cell differentiation and cellular Ca mobilization (Jacob 2001). Niacin is widely distributed in plants, but a variable amount of this is in a bound, unavailable form. It is produced by microorganisms in the gut (Carroll *et al.* 1949) and can be synthesised from tryptophan by hepatic tissue. Toxicity of niacin in horses has not been described.

Riboflavin is a precursor of the coenzymes flavin adenine dinucleotide (FAD) and flavin mononucleotide (FMN). Riboflavin is found, normally as FAD and FMN in high concentrations in forage legumes, such as alfalfa, and clover (13–17 mg/kg DM) and slightly lower concentrations in grass hays (7–10 mg/kg DM), but low concentrations in cereal grains (1.4–1.7 mg/kg DM) (NRC 2007). Riboflavin is synthesised in the intestines of horses, at higher rates where microbial activity is greater (Carroll *et al.* 1949). Riboflavin has a low toxicity. NRC (2007) has retained its previous estimates of dietary riboflavin requirement of 2 mg/kg dietary DM. This is equivalent to 0.04 mg/kg BW daily for maintenance, breeding stallions and light work and 0.05 mg/kg BW daily for hard work and lactation (Table 4.2).

Pantothenic acid is widely distributed in the diet (McDowell 2000). It functions as a carrier of acyl groups in the form of the protein, coenzyme A, which makes high-energy thioester bonds with carboxylic acids, the most important of which is acetic acid. Acetic acid results from the metabolic catabolism of both fats and carbohydrates. Acetyl groups from these sources, as of acetyl-CoA are further metabolized in fat synthesis, or for energy production in the TCA cycle. No signs of deficiency were observed in horses receiving a diet deficient in pantothenic acid (Carroll *et al.* 1949) and no dietary requirement has been established (NRC 2007).

In carbohydrate metabolism, acetate is derived from pyruvate. Pyruvate requires TPP, or cocarboxylase, the active form of thiamin, for the cleavage of pyruvate, otherwise the formation of lactic acid from pyruvate will be accelerated in intense exercise. It is possible that

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(i)



(ii)



Plate 4.1 (a) Off-forehooves of 8-year-old Irish chestnut gelding, before (i) and after (ii) receiving 15 mg synthetic biotin/day orally for 13 months.

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(i)



(ii)



Plate 4.1 (b) A 5-year-old TB gelding hack, before (i) and after (ii) receiving 15 mg synthetic biotin/day orally for 5 months. (Photographs 4.1(a) and (b) by kind permission of Norman Comben, MRCVS; Comben *et al.* 1984.)

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supplementary pantothenic acid and thiamin could promote the aerobic metabolism of pyruvate to acetyl-CoA, reducing lactate production, although there is no evidence that this would occur in horses given normal feeds.

Vitamin B6 (pyridoxine) is a component of numerous enzymes involved in the metabolism of proteins, fats and carbohydrates, is widely distributed in the diet (McDowell 2000) and no dietary requirement has been established.

Choline and *betaine*. Most mammals are able to synthesise choline and no dietary requirement has been demonstrated for it in the horse. It functions as a lipotrope, and has other metabolic functions. Betaine is a metabolic precursor of choline and is a methyl donor (transmethylation) and participates in the methionine cycle – primarily in the liver and kidneys. Inadequate production of methyl groups leads to hypomethylation in hepatic protein (methionine) metabolism and to inadequate hepatic fat metabolism, steatosis (fatty accumulation) and subsequent plasma dyslipidemia. Warren *et al.* (1999) gave untrained TB horses 80 mg betaine/kg BW daily for 14 days before exercise to fatigue and found it lowered plasma FFA both before and after exercise and it lowered plasma lactate 60 min after exercise, indicating that betaine might influence lactate metabolism following exercise in untrained horses. No effect was observed in trained TBs.

Vitamin B15 (pangamic acid). Pangamic acid is a term that has been used indiscriminately to describe several products. It is not a definable entity and no substantive data appear to have been presented to support claims of beneficial biological effects.

Vitamin C (ascorbic acid). Vitamin C activity originates from two compounds: L-ascorbic acid and dehydro-L-ascorbic acid, which are equivalent in activity (McDowell 1989). The horse has the ability to synthesise vitamin C from glucose (Stillions *et al.* 1971a). Dietary sources are very poorly absorbed, and Löscher *et al.* (1984) concluded that an i.v. dose of 10 g ascorbic acid was needed to raise the blood concentration. Its acidic nature leads to local irritation following subcutaneous and intramuscular administration and so it is administered by i.v. injection for removal of renal calculi. Ascorbic acid, ascorbyl palmitate and ascorbyl-2-monophosphate have been used as vitamin C supplements for horses. Snow *et al.* (1987c) reported that single oral doses of 20 g of ascorbic acid had no effect on plasma concentration, whereas daily administration of either 4.5 or 20 g resulted in an increased plasma level after 5–10 days. Ascorbyl palmitate is more efficient than either ascorbyl-2-monophosphate (Deaton *et al.* 2003), or ascorbic acid (Snow & Frigg 1987a) in raising plasma ascorbic acid concentrations. A relationship exists between decreased blood ascorbic acid concentrations and several diseases in horses, including post-operative and post-traumatic wound infections, epistaxis, strangles, acute rhinopneumonia, recurrent airway obstruction (RAO) and performance insufficiency (Jaeschke & Keller 1978; Jaeschke 1984; Deaton *et al.* 2004a). Oxidative stress is caused by the inflammatory response to organic dust in cases of RAO (Deaton *et al.* 2006), associated with a large decrease in the ascorbic acid content of the lavage fluid and depletion of antioxidant in the tracheal epithelial lining. Mean plasma concentration of ascorbic acid in working horses ranges from 1 to 4 µg/mL (Snow *et al.* 1987c; Williams *et al.* 2004a). Exercise does not seem to decrease blood levels of ascorbic acid (Williams *et al.* 2004a; de Moffarts *et al.* 2005); but it is unclear whether endogenous ascorbic acid synthesis is adequate to meet the needs of all horses (NRC 2007), although no benefit has been demonstrated from supplementation of healthy horses.

WATER REQUIREMENTS AND FLUID LOSSES

There is a total of 62–68% empty body water in adult horses. This is made up of intracellular fluid (ICF) and extracellular fluid (ECF), which includes plasma, intercellular fluid, transcellular fluid and lymph. Of the total body mass, or weight, ICF constitutes 36–46% and ECF 21–25%, including plasma volume (PV) 4% (Fielding *et al.* 2004; NRC 2007). Total body water decreases with age. In foals during the first week it is nearly 71%, so that a disruption of their fluid supply causes a more urgent health threat than it does in adults (NRC 2007).

The horse also needs to take in water with its food to act as a fluid medium for digestion and for propulsion of the digesta through the GI tract, and to make good losses through the lungs, skin and in the faeces and urine. The DM content of faeces is higher in horses given grain-based feeds (34% DM) than it is in those given only hay (15–28% DM) (Zeyner *et al.* 2004; NRC 2007) and this represents 55–63% of their daily water intake (Cymbaluk 1989). In healthy adult horses undertaking light work one estimate showed that 18% of water loss occurred in the urine, 51% in the faeces and the remaining 31% represented insensible losses (Tasker 1967). Restricted water intake will depress appetite and reduce feed intake.

Equids differ in their ability to conserve body water and to withstand dehydration. Asses from the dry tropics can thwart extreme dehydration because they can conserve water more efficiently than horses. A rise in

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environmental temperature from 15 to 20°C increases the water requirement of horses by 15–20%. Work, depending on its severity, will raise requirements by 20–300% above the needs for maintenance through increased losses from the lungs and skin. For obvious reasons, peak lactation can lead to requirements twice those of maintenance (Table 7.5). Dehydration and heat stress are serious welfare issues. Heat stress is associated with increased respiratory rate and depth, head nodding, flared nostrils and apathy, accompanied by an elevated rectal temperature. The standardized skin tent test (pinching a fold of skin at the base of the neck and timing its return to normal) and measures of mucous membrane dryness are not valid, or repeatable, indicators of dehydration when compared with plasma osmolality (P_{osm}), as a 'gold standard' criterion and the test is unrelated to PCV, water intake, or serum total protein concentration in working horses (Pritchard *et al.* 2008). In donkeys, however, the positive skin tent test was associated with a lower serum Posm (Pritchard *et al.* (2006). The volume of water consumed and the number and duration of drinking bouts are the most reliable guide to hydration status of mature working horses (Pritchard *et al.* 2008). Negative **alliesthesia** for water, fear of the environment, behaviour of the horse's owner or water availability, might negate this guide.

The horse obtains water for its metabolic needs from three sources – fresh water, natural herbage and other foods and from metabolic water. Young, growing, herbage contains 75–80% water, so that under many circumstances additional fresh water is not required, but a source should always be provided. In arid conditions herbage is very different, and horses will seek and consume poisonous shrubs and succulent plants unless water and feed are provided.

Metabolic water is that produced during the degradation of carbohydrates, fats and proteins in cellular respiration, e.g.:

$$C_6H_{12}O_6 + 6\,O_2 \rightarrow 6\,CO_2 + 6\,H_2O \text{ for carbohydrate}$$

$$C_{57}H_{104}O_6 + 80\,O_2 \rightarrow 57\,CO_2 + 52\,H_2O \text{ for typical fat}$$

$$2\,C_3H_7O_2N + 6\,O_2 \rightarrow (NH_2)_2CO^* + 5\,CO_2 + 5\,H_2O \text{ for the amino acid alanine}$$

where * indicates urea. Thus, per 100 g glucose, average fat or average amino acid metabolized, there are, respectively, 60, 106 or 51 g water produced. Per kg of feed ingested, it amounts to the equivalent of 350–400 g water, depending on the feed's digestibility. Nevertheless, in circumstances of choice, the water intake of horses is highly correlated with intake of DM and amounts to between 2–4 L/kg DM in stabled horses worked moderately.

Water requirements

Maintenance

For the maintenance of adult horses in an equable environment, the total water requirement is probably less than 2 L/kg DM intake (about 5 L/100 kg BW).

Working horses

Strenuous effort in hot climates increases the water requirement to as much as 5–6 L/kg DM intake (12–15 L/100 kg BW) when there is an inevitable loss of relatively large amounts of Na, K and Cl in sweat. Excessive dehydration can be fatal. Certain breeds of horse and other species of *Equus* (e.g. *E. asinus*; Maloiy 1970) can sustain extensive water loss without apparent discomfort, but horses of temperate breeds succumb to water losses that amount to 12–15% of their body weight (Hinton 1978; Brobst & Bayly 1982). The estimates of the Hanover group (Meyer 1990a) indicate that a 500-kg horse, trotting at 3.5 m/s, in an ambient temperature of 27°C would require a minimum of 10–12 L water/h to replace inevitable losses. Water repletion should be accompanied by balanced electrolytes, although electrolytes often have to be given first in order to induce drinking when isotonic or hypotonic dehydration has occurred (even though electrolyte supplementation increases the risk of gastric ulceration, see Chapter 11). A fit horse should be walked or allowed to graze so that it cools down gradually over an hour before being given substantial amounts of water. Excessive consumption of cold water by hot horses can precipitate colic . Water temperature influences water intake, but the way this occurs depends on environmental temperature – horses drink less cold, than warm, water during cold weather (Kristula & McDonnell 1994). During very cold weather, warm water at a temperature of 7–18°C should be provided. Horses given a saline solution immediately after treadmill exercise in a room at 25°C at a relative humidity (RH) of 60% preferred a solution at 20°C to solutions at 10 or 30°C (Butudom *et al.* 2004). Decreased consumption of water contributes to the incidence of impaction colic and depressed performance in racehorses.

The GI tract contains 300 mg Na, 150 mg Cl and 220 mg K per kg BW, or 15–20% of total body Na, 17% of total Cl and 10% of total K. The GI tract K content is correlated with intake and the content of all three is related to the GI water content (Meyer 1996a,b). This content is aided by the consumption of soluble fibre, so providing a

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Table 4.6 Daily fluid intake per foal (Martin *et al.* 1992).

Age (days)	Milk intake (kg)	Water intake (kg)	Total fluid intake (g/kg BW)	Milk consumed (kg/kg BW gain)
11–18	16.9	nil	246	12.8
30–44	—	3.9	202	15.7
60–74	18.1	5.5	172	16.4

source of electrolytes and water for extended work (see 'Endurance riding', Chapter 9).

Foals and weaned horses

A high-yielding 500-kg mare produces 12 kg water daily in her milk (see Table 7.5, water requirements in the stud). However, a foal has a greater requirement than an adult in proportion to its size because it is less able to concentrate urine. A frequent cause of death in neonatal foals is rapid dehydration through persistent diarrhoea, requiring treatment with a physiological salt solution (see Chapter 11). The fluid intake of foals suckling grazing mares was measured (Martin *et al.* 1992) in Queensland, where the maximum ambient temperature averaged 30°C and the relative humidity (RH) averaged 70% (Table 4.6).

Weaned horses satisfy their water needs by relatively brief periods of drinking, even when meal-fed. Sufit *et al.* (1985) found that ponies drank for 27 min daily, much of which was periprandial.

Water quality

Where it is feasible, water should be provided from the mains. If mains water is unavailable, then well water, or watercourses, must be free from pollution by sewage or fertilizer seepage. Diets containing 1.74–1.85% nitrate were given to non-pregnant mares leading to elevations in concentrations of blood nitrate and nitrite. Yet no changes were detected in methaemoglobin, oxygenated haemoglobin, reduced haemoglobin or haematocrit (Burwash *et al.* 2005), possibly indicating that horses have a higher threshold of tolerance cf. ruminants.

Potentially dangerous microbiological contamination of water can occur. For instance, the urinary excretion of leptospira by rodents can pollute rivers and sewage flooding can cause abortion in mares and death of foals. Potomac horse fever is a febrile gastrointestinal disorder of horses caused by *Ehrlichia risticii*. It occurs in North America and Europe between late spring and early autumn in temperate areas and is associated with depression, anorexia, lethargy, fever, mild colic, watery diarrhoea, dehydration, oedema and leukopenia. It is maintained in a complex aquatic ecosystem from which transmission to horses occurs by ingestion of insects. Madigan *et al.* (2000) demonstrated its transmission by the ingestion of mature caddis flies from polluted water in northern California.

River water is normally safer than pond water. Nevertheless, in addition to risks incurred from any upstream pollution, a decreased rate of flow in summer droughts encourages the growth and accumulation of plant life. Although this assists oxygenation of water, blue–green algae cyanophyceae (Myxophyceae) can proliferate and be held in position by other plant life, in association with the **eutrophication** of worldwide water supplies (de Figueiredo *et al.* 2004). More importantly, the genera of cyanobacteria, *Microcystis, Aphanizomenon, Planktothrix/Oscillataria, Nostoc* and *Anabaena* produce alkaloid cyclic decapeptide hepatotoxins called microcystins, which affect both humans and livestock (de Figueiredo *et al.* 2004; NRC 2007). Cyanophytes can cause taste and odour adulteration of water described as mouldy, musty, grassy or as septic-tank odour. The consumption of *Anacystis cyanea* (Clarke & Clarke 1975) by horses can cause liver damage, icterus and photosensitization or death. A microcystin is probably the toxic agent in this species. Microcystin-producing algae in the US Midwest grow vigorously in surface waters with a high content of suspended solids, chlorophyll, P and N compounds, although these conditions are not invariable (Graham *et al.* 2004). The calculated trigger value for microcystin-LR (one of 80 variants) in water for horses is 2.3 µg/L, or 11500 cells/mL (ANZECC 2000). These values are not based on evidence in horses and incorporate a safety margin for intra- and inter-species variation of lowest observed adverse effects (NRC 2007). Melt water from de-icing salt used on roadways is frequently contaminated with heavy metals: cadmium, lead, copper and zinc (Backstrom *et al.* 2004).

Water losses and deprivation

Renal losses

The nonexercised horse disposes of most excess water by excretion through the kidneys. This water is the vehicle

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for the excretion of excess salts of Na and K, and much of the breakdown products of N metabolism. Whereas Ca salts are excreted in a solid form there is a limit to which the horse can concentrate urea and the highly soluble salts of Na and K. Thus, where diets are rich in salt, or in protein, more dietary water will be required. Based on evidence from other species, an increase in total dietary NaCl from 7.5 to 30 g/kg would be expected to increase the ratio of free-choice water to DM in the diet from 2 : 1 to 3.5 : 1.

Meyer (1990a) reported that 73–89% of total water intake was lost by renal excretion in horses given concentrates, while <60% was lost by this route in horses given hay. Water restriction decreases renal losses, but does not affect sweat losses in normal horses. Where water restriction persists, then a considerable stress is induced in exercised horses, with an increase in plasma protein and urea concentrations and increased breathing frequency during exercise. Meyer (1990a), found urinary urea was 6–8 g/L at rest, but 15–50 g/L after exercise, reflecting the solubility of urea and the relative lack of effect on water conservation. The highest of the urea values occurred with high protein diets and extended water deprivation.

Table 4.7 Composition of sweat following 2 hours of exercise (after Meyer 1990a).

	g/L		mg/L
Na	2.77	Ca	123
K	1.42	Mg	52
Cl	5.33	Zn	11.4
		Fe	4.3
		Cu	0.27
		Mn	0.16
		Se	2–5 µg/L

Table 4.8 Net water lost per unit volume (m^3) of exhaled air and water lost in exhaled air per hour.

	Water lost during expiration (g/m^3)	Expired water lost (g/h/horse)
Resting	38.3	172.5
Trotting (3.5 m/s)	44.2	1356.0

Sweat

Horses lose a large amount of fluid as sweat during exercise. The amount increases greatly with a rise in environmental temperature. Meyer (1990a) recorded losses of 1 L/100 kg BW/h at 18–20°C, but for each degree increase in environmental temperature (range 15–27°C) sweat production increased by 3%. In contrast to fluid losses from the lungs, sweat contains significant amounts of electrolytes and small amounts of trace elements (Table 4.7). During the 2 h of exercise (Table 4.7) losses in sweat were:

	mg/kg BW
Na	28–69
K	17–30
Cl	56–118

Thus, during this exercise, a 500 kg horse could lose 50–90 g NaCl in sweat alone.

Evaporative losses from the lungs

The evaporation of sweat, or of water from the lung surface, absorbs heat and so cools the horse (the isothermal evaporation of 1 kg insensible water absorbs 2256 kJ). The water loss from the surface of the lungs increases greatly during exercise, owing to an increase in body temperature and an increase in both respiratory frequency and tidal volume. Heat loss through the lungs at 20°C and 60% relative humidity (RH) was calculated to be 289 kJ/h at rest and 3059 kJ/h while trotting (Table 4.8).

An increase in body temperature during exercise increases the moisture content of exhaled air by 15%, but this, together with the increased respiratory volume during exercise, increases the expiratory water loss nearly eightfold (Table 4.8). Water losses of exercising horses, in sweat and from the lungs, cannot be reduced during water deprivation if body temperature is to be contained within physiological limits, whereas urinary losses are decreased.

Determining water deprivation and response to thirst

Apart from various clinical signs of fluid loss, including skin turgor, the PCV of blood is not a guide to dehydration and water deprivation. The shrinkage of red cells and the changes in the release of red cells from the spleen during dehydration and exercise make this an unreliable guide. Total plasma protein, however, increases by 10–12 g/L (say, from 62 to 73 g/L) with a fluid loss causing a 12–15% decrease in body weight. Changes in plasma and urine electrolytes and urea depend on several associated factors. Fluid losses of this extent in dehydrated TB geldings increased the concentration of serum and urinary urea by 68 and 130%, respectively (Brobst & Bayly 1982). The

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Table 4.9 Mean fluid contents of the GI tract in ponies given 18 g DM/kg BW as hay or as concentrated feed (after Meyer 1990a).

	Hay	Concentrates
3.5 hours postfeeding		
DM (g/kg BW)	19.4	18.3
Water (mL/kg BW)	183	101
Na (mg/kg BW)	398	226
K (mg/kg BW)	289	220
After 1 hour treadmill exercise at 3.5 hours postfeeding		
Reduction in water content (mL/kg BW)	20	8
Reduction in Na content (mg/kg BW)	291	No significant change

specific gravity of urine reached at least 1.042 and urine osmolality increased 30% to 1310 mOsm/kg when the osmolality ratio urine:serum increased to 4.14 : 1.00. Voluntary drinking starts in ponies when plasma osmolality (mOsm/L) increases by 3% from normal. Drinking is also stimulated by inducing the formation of urine with a consequential decrease in plasma volume, measured as an increase in plasma protein concentration (Sufit *et al.* 1985).

Nyman *et al.* (2002) compared water intakes of dehydrated, normal and hyperhydrated Standardbred horses following subsequent exercise. With both osmotic and hypovolaemic thirst stimulus the two former groups rehydrated more rapidly post exercise than did horses that were hyperhydrated before exercise. The loss of plasma volume was slightly less in hyperhydrated than in normal horses. Plasma aldosterone concentration increased to the same extent after 10 min exercise, irrespective of hydration status; but in one test it increased to a greater extent in the hyperhydrated group. The differences amongst horses in their inclination to drink when dehydrated is likely to have both physiological and psychological explanations. Schott *et al.* (2003) demonstrated three distinct groups amongst 18 untrained two-year-old Arabians in the amount of fluid taken in during and after a 60 km treadmill exercise. 'You can take a horse to water …'.

The effect of fluid intake before exercise on performance will depend on many factors including the amount consumed, its solute content, diet, length and intensity of exercise and ambient temperature (see also Chapters 9 and 11). Finally, horses travelling to a competition frequently arrive in a dehydrated state.

Water reserves

Voluntary water intake and water content of the GI tract are greater in horses fed hay than in those given only concentrates. The GI tract following hay feeding can act as a reservoir of water and sodium for metabolic needs (Table 4.9). Chronic dehydration (6% decrease in BW) in donkeys is associated with an increased fermentative activity in the caecum and enhanced fluid retention in the ventral colon. These responses are related to a greater total mucosal and crypt surface area per unit volume of large intestine in donkeys. This adaptation allows appetite to be maintained (Sneddon *et al.* 2006). Sodium chloride dosing is harmful in states of water deprivation, but it will increase voluntary water intake and retention prior to endurance work. Maximum retention occurred at the 3rd–4th hour after providing NaCl in a feed (Meyer 1990a) and this could be the optimum time after a small meal for extended exercise in hot weather (see Long-distance work, Chapter 9).

STUDY QUESTIONS

(1) Are there any circumstances in which you might expect a useful response of horses to a fat-soluble vitamin preparation? If there are, describe the circumstances and give reasons why these might exist?
(2) What factors should be considered in deciding on the adequacy of a water source for horses?

FURTHER READING

Divers, T.J., Cummings, J.E., de Lahunta, A. , Hintz, H.F. & Mohammed, H.O. (2006) Evaluation of the risk of motor neuron disease in horses fed a diet low in vitamin E and high in copper and iron. *American Journal of Veterinary Research*, **67**, 120–6.

Graham, J.L., Jones, J.R., Jones, S.B., Downing, J.A. & Clevenger, T.E. (2004) Environmental factors influencing microcystin distribution and concentration in the midwestern United States. *Water Research*, **38**, 4395–404.

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5
Ingredients of Horse Feeds

Some parts of the kingdom produce no grain so much as oats which probably may be the reason why they have come to be used as our chief provender.

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Some of the main chemical characteristics of the ingredients of horse feeds are given in Appendix C. Ingredients should be selected not only to provide the nutrients required, but also to be uniform in quality, to be free from harmful contaminants and dust and to balance dense energy-rich feeds with more bulky feeds. The rate of consumption of DE should not be excessive and the stomach contents should retain an 'open' physical texture.

ROUGHAGE

Loose hay

Grasses and forage legumes are cut for hay. Most common species of grass are suitable, but the more popular and productive ones include rye grasses (*Lolium*), fescues (*Festuca*), timothy (*Phleum pratense*) and cocksfoot (*Dactylis glomerata*). Many species found in permanent pastures, for example meadow grasses (*Poa*), bromes (*Bromus*), bent grass (*Agrostis*) and foxtails (*Alopecurus*), are also quite satisfactory. Among legumes, red, white, alsike and crimson clovers and trefoils (*Trifolium*), lucerne (*Medicago*) and sometimes sainfoin (*Onobrychis*) are used. Although the CF content of crimson clover (*Trifolium incarnatum*) hay is similar to that of other clovers, the fibre tends to be less easily digested by the horse (forage legume fibre generally is more lignified than that of grass). For serving as horse hay or haylage crops, two reliable seed mixtures are:

(1) three perennial rye grass strains – Melle 5 kg/ha, tetraploid Meltra 15 kg/ha, hybrid tetraploid Augusta 13 kg/ha – or as a two-year crop:

(2) tetraploid broad red clover – Hungaropoly 7 kg/ha – and tetraploid Italian rye grasses – Wilo 15 kg/ha, Whisper 10 kg/ha.

The leaves of forage legumes and grasses are much richer in nutrients than are the stems, as stems contain about two-thirds of the energy and about half of the protein and other nutrients found in the aerial parts. The leaves of legumes tend to shatter more readily than grass leaves so that care is necessary at haymaking to conserve the nutritional quality of legume hay. At the same stage of maturity, legume hay contains more DE, calcium, protein, β-carotene and some of the B vitamins, including folic acid, than does grass hay. Horses consuming hay composed predominantly of forage legume tend to produce more urine with a strong ammonia smell and containing deposits of calcium salts. This is a normal physiological response in healthy animals.

As long as hay is composed of safe, nontoxic, nutritious plants, the stage of maturity of the crop at the time of cutting and the weather conditions and care to which the haymaking is subject are more important characteristics than the species of plant present. The digestibility and feeding value of hay made from various grass species have been determined at similar stages of maturity. The digestibility of Matua prairie grass (*Bromus unioloides*) hay, a cool season brome grass, was shown to be higher than that of coastal Bermuda grass (*Cynodon dactylon L*) (Sturgeon *et al.* 1999). Matua brome grass (*B. willdenowii* Kunth) hays, 11.5% and 15.1% CP, were consumed in similar quantities respectively to alfalfa hays of 15.4% and 20.4% CP by breeding mares and the two species led to a similar reproductive performance (Ball *et al.* 1999; Guay *et al.* 2002). Matua grass hay containing 17% CP, 54% NDF, 5.5% lignin has a higher dry matter digestibility than Matua hay of 9% CP, 67% NDF and 9.5% lignin (Box

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et al. 2001). Coastal Bermuda grass (*Cynodon dactylon L*) hay is consumed in greater quantities and produces greater growth rates than hays of either Florakirk or Tifton 85 Bermuda grass, or those of Florona star grass (Lieb & Mislevy 2001).

LaCasha *et al.* (1999) found that voluntary intake and OM digestibility were both greater for the alfalfa (*Medicago sativa L*) hay than for the grass hays: Matua brome grass (*Bromus willdenowii* Kunth. cv. Grasslands Matua) and coastal Bermuda grass (*Cynodon dactylon*), and the intake of the Bermuda grass hay was poorer than that of Matua. The authors concluded that the Matua grass hay should meet the digestible protein and mineral requirements of yearling Quarter Horses. Three other subtropical perennial grass hays: Tifton 85 Bermuda grass, Florakirk Bermuda grass (*Cynodon spp*) and Florona star grass possessed as good, or better, nutrient contents and digestibility cf. coastal Bermuda grass (Lieb & Mislevy 2003).

The voluntary consumption (and digestibility) of warm- and cool-season grass hays as a sole feed varies from 19–29 g/kg BW and is negatively correlated with their NDF content:

$$y = 124.55 + 0.0155x^2 - 2.5742x \left(R^2 = 0.67\right)$$

(Lawrence *et al.* 2001)

$$y = 90.95 - 0.98x \left(R^2 = 0.68\right)$$ (Reinowski & Coleman 2003)

where x = % NDF in DM and y = g/kg BW per day

The voluntary intake of three warm-season perennial grass hays decreased in the order: big bluestem (*Andropogon gerardii*), Indiangrass (*Sorghastrum nutans*) and eastern gammagrass (*Tripsacum dactyloides*), (NDF contents 700–740 g/kg DM), but timothy hay (613 g NDF/kg DM) was preferred to all warm-season hays (Reinowski & Coleman 2003; Reinowski *et al.* 2003). Hay consumption extends eating time and so is beneficial to otherwise idle horses. Those given only timothy hay can spend 6.7–9.3 hours/day eating with a DM intake of 1.5% BW/day (Shingu *et al.* 2001). As pasture herbage matures, the yield of DM per hectare increases, the moisture content of the crop decreases and, in the UK, the weather becomes warmer. At Jealott's Hill Research Station, Bracknell, Berkshire, many years ago, the average yield of DM from early hay crops was only 57% of that cut at a later date. Even when the aftermath was included, the total yield of the early hay amounted to only 71% of that produced by later cutting. Thus, there is a considerable commercial incentive to produce grass hay at the late-flowering stage. Nevertheless, where hay of good nutritional quality is required for horses, mixtures of grass and clover should be cut before the grass is in full flower when the protein content of the crop lies between 9 and 10% DM and the crop contains high concentrations of Ca, P and other minerals. Hard, mature grass hays, however, frequently contain 3.5–6% CP, lower concentrations of minerals and more crude fibre (CF; Plate 5.1). Hyslop *et al.* (1998a) found that mature pony geldings given *ad libitum* access to mature threshed grass hay (933 g OM, 49 g CP, 796 g NDF and 6.25 MJ DE/kg DM) were able to exceed by 43% their predicted energy maintenance requirement, whereas the digestible crude protein (DCP) intake was 40% lower than the predicted protein requirement. Many samples of mature grass hay, in my experience, are similar to this one, requiring a protein supplement, in particular because much of the protein digested is as inorganic N in the hind-gut.

Using equine faecal flora, Hussein *et al.* (2001) found that DM and OM digestibilities *in vitro* were lower for perennial rye grass (*L. perenne*), than for brome grass (*Bromus inermis*), orchard grass (*Dactylis glomerata*), or tall fescue (*Festuca arundinacea*) at various stages of maturity. Much further work is required to determine appropriate grass species mixtures to meet the various needs of equine pastures and hay. Good-quality hay from pure stands of lucerne or sainfoin is difficult to make when natural drying is relied on because moisture loss from the thick juicy stems is relatively slow and mechanical turning and tedding can result in a considerable loss of leaf, which dries sooner and shatters more readily. For the best product, these legumes should be cut before flowering at the bud stage, because after first flowering the CP content declines at a rate of some 0.5% daily and the DE declines by some 0.75% daily.

Horses should never be given mouldy hay, so the making of satisfactory leafy hays during inclement weather presents a considerable problem, in the absence of a facility for artificial drying. Best-quality hay should be leafy and green, but free from mould dust, weeds and pockets of excess weathering. When ley mixtures are grown for hay, the first cut may contain more weeds, the second cut is generally produced from a faster growth and contains more stem, but the third cut may have the highest nutrient content and leaf, giving a small yield per hectare.

Haylage (see Chapter 10, for grass species, safety aspects and silage, and Chapter 11, Botulism)

Haylage (grass cut between early silage and hay stages and normally containing, after preservation, 50–65% DM) is widely used in place of hay, for feeding horses. However,

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(a)



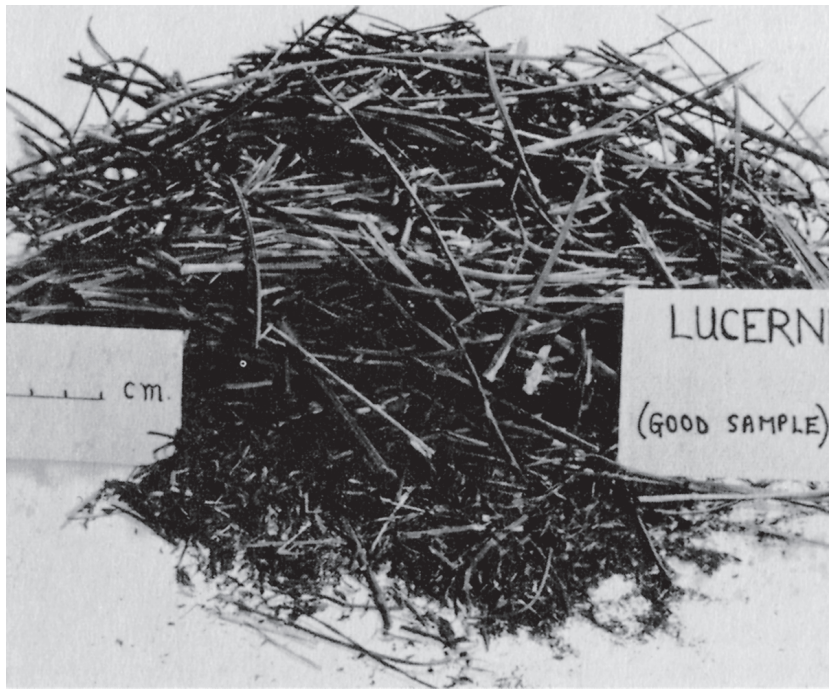
(b)



Plate 5.1 Hay samples of various types. (a) Hard 'seeds' hay cut when the grass has formed seed heads. The material is clean and low in dust but of low nutritional value. (b) Lucerne (alfalfa) hay, which is similarly stemmy and has been sun dried; bleaching destroys its vitamin A potency but it adds some vitamin D potency. Poor harvesting has led to the loss of most of the leaf, so depriving the hay of its most digestible component.

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(c)



(d)



Plate 5.1 *Continued* (c) Good-quality lucerne hay, which has been barn-dried. This is 'rich' material and care should be exercised to avoid loss of leaf during feeding (note the leaf particles at the base of the sample). Artificial drying deprives the hay of vitamin D potency. (d) Meadow hay containing a proportion of timothy (*Phleum pratense*). This sample is of reasonable quality and is free from significant moulding.

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the production of haylage for home consumption can be justified only if:

- there are enough horses to make use of the minimum quantity that can be produced in an efficient manner, and to eat an opened bale within a couple of days;
- there is adequate grassland, that has not been grazed that year by horses, and that can be set aside and fertilized; and
- there is manpower with the appropriate knowledge, equipment and space available for making and storing the product.

The most successful producers and users of haylage find that a product very high in DM content is the most successful, and acceptable to more horses in a stableyard. At the Yorkshire Riding Centre, in England, meadow grass is cut from mid-June, when the grass is starting to flower, about two weeks before a hay crop would be taken. The cut material is allowed to wilt in the field for a day and is then square-baled (Plate 5.2) a day before it would be ready as hay, yielding a product with 50–68% DM and 90–120 g CP/kg DM. Square bales are preferred as 'round' bales are more inclined to mould in the centre, where there is an empty space. Avoid puncturing the plastic bale cover, as moulding will occur at that point. Yet it is normally safe to pull away and discard only the portion that is mouldy, as the mycelium of *most* moulds is benign and does not penetrate well-compressed bales (Plate 5.2). If there is secondary fermentation, occasioned by excessive heating in the centre, then the whole bale should be discarded. A well-made and packed bale can be left open for up to 4 days during cool weather, although for a lesser time in hot weather, before the residue should be discarded.

By weight, at 55–65% DM content, haylage is equivalent in energy value to the same weight of typical grass hay. At the Yorkshire Riding Centre, 0.405 ha (1 acre) provides a year's supply of haylage for one horse. About 375 bales meet the requirements of 40 horses for a year, except for one or two that will not eat haylage. The droppings of most horses are looser when they are introduced to haylage, an effect similar to that which occurs when horses are put out to grass, yet colic does not usually occur. So horses 'coming off' grass adapt more easily to a regime that includes haylage. One bale (≈150 kg) daily is sufficient for 35 horses in winter, given on the floor, two or three times daily (5.5–7.5 kg daily for smaller horses and up to 12 kg for larger horses). This scheme keeps the horses in a mental state that is suitable for novice riders. In summer, when the horses are generally more active, more concentrates are given so that the haylage allowance is reduced by 20–25% and the horses are run on grass at night (Table 5.1). Moreover, competition horses receive less than others, in order to avoid an excessively large 'belly'. The intake and apparent digestibility values for haylage, grass hay, big-bale grass and legume silages and clamp silage have been compared using ponies. The DM intake of clamp silage was the lowest, a response that could have been related to its low pH and the DM intake of hay was lower than that for big-bale silages. The digestibility of hay and haylage was lower than for clamp silage and big-bale silage. All feeds but hay, met the daily DE and digestible CP requirements (Moore-Colyer & Longland 2000; Hale & Moore-Colyer 2001; Tables 5.2 and 5.3). Further, big-bale grass silage was readily accepted and was more digestible (DM, OM, CP, ADF and NDF) than grass hay derived from the same source (Moore-Colyer *et al.* 2003b), although it produced faeces with a lower DM content and higher pH (Coenen *et al.* 2003c). The comparable DM digestibilities were 67% and 49%.

As much of the feeding value of forages is derived from absorption of nutrients from the large intestine, the amino-acid value of forages is low compared with that of concentrate feeds. The true protein and lysine values of high quality alfalfa cubes are only 60% of the comparable values for a concentrate mixture (Coleman *et al.* 2001).

'PROCESSED' FEEDS

Pelleting aids

Binders

Bentonite (hydrated aluminosilicate, principally montmorillonite) and attapulgite clays and kaolin are permitted to a maximum of 2% of total ration. Lignin sulphonate may be incorporated up to 4% of finished pellet, but ball clay is no longer approved as a feed ingredient in the USA (AAFCO 2005). The permitted list is similar in the UK (Anon 2000b).

Hydrated sodium calcium aluminosilicate (also used as a ruminal fluid buffer) and other aluminosilicates (zeolites) are potential mycotoxin adsorbants (Council for Agricultural Science and Technology 2003), calcium bentonite decreases the adverse effects of dietary aflatoxin in pigs; but none of this evidence was obtained in the horse. The ADF fraction of wheat offal has been shown to bind aflatoxin B_1, inhibiting its intestinal absorption in rats (Frape *et al.* 1981a,b,c, 1982). Clays have a large surface area and this characteristic, combined with their buffering capacity, is the reason bentonite is included in products to counter laminitis and gas colics of horses (see Chapter 11).

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(a)



(b)



(c)



Plate 5.2 Haylage. (a) A stack of plastic-wrapped bales: square bales are less inclined to mould in the centre than round bales; (b) surface mould on a bale, adjacent to a puncture; (c) a cut section of the bale, showing that mould penetration of the compressed haylage is slight.

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Table 5.1 Daily rations for horses using grass haylage of 50–60% DM.

	Haylage (kg/day)	Concentrates (kg/day)
Event horses	7–7.5	5–6
Riding horses for novices		
1.68–1.73 m (16.2–17 hands) – summer	7–7.5	1.5–2.5
– winter	11–12	0–1
1.57–1.68 m (15.2–16.2 hands) – summer	5–6	1–1.5
– winter	7–7.5	0–0.5
1.42–1.57 m (14–15.2 hands) – summer	5–6	0.5–1
– winter	6.5–7	0–0.5

Table 5.2 A comparison of grass hay (H), haylage (HY), big-bale silage (BB) and clamp silage (CS) in mature Welsh-cross pony geldings. Feeds were offered at the rate of 1.65 kg DM/100 kg BW daily (g/kg DM unless otherwise stated) (Moore-Colyer & Longland 2000).

	H	HY	BB	CS
DM (g/kg)	922	676	500	337
Organic matter	946	934	937	916
Crude protein	44	70	111	154
Starch	90	50	40	33
GE (MJ/kg)	17.4	17.5	17.8	18.6
Ca	3.1	5.8	4.7	5.5
P	1.3	2.0	3.7	3.3
Mg	1.4	1.8	2.4	2.6
Total NSP	408	293	405	353
DM intake (g/kg LW/day)	14.7	18.4	17.3	9.17
DE (MJ/kg DM)	5.75	9.09	9.83	11.98
DE intake (MJ/kg LW/day)	0.091	0.167	0.170	0.109
DCP (g/kg DM)	6	34	74	104

Table 5.3 Composition, intakes and apparent digestibility coefficients of hay, big-bale grass silage and big-bale red clover silage offered *ad libitum* to ponies (Hale & Moore-Colyer 2001).

	Hay	Grass silage	Red clover silage
Dry matter (g/kg)	852	371	268
Organic matter (g/kg DM)	919	916	866
Water-soluble carbohydrate (g/kg DM)	103	110	8.9
Crude protein (g/kg)	74	104	193
Dry matter intake (kg/day)	5.5	6.1	7.2
Dry matter digestibility	0.36	0.69	0.74
Crude protein digestibility	0.29	0.68	0.80

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It has been considered that clay inhibits nutrient absorption, but the author's own studies show it does not interfere with vitamin A absorption from a retinyl palmitate source.

Pellets and wafers

Straw, chemically processed with sodium hydroxide or ammonia, is discussed elsewhere in this chapter (see Sodium-hydroxide-treated straw). Hay is also occasionally processed; it is ground and pelleted or chopped and wafered. During pelleting, molasses and a binding agent are normally added to achieve a satisfactory product. Despite the additional costs of processing, pellets possess some advantages over long hay:

- The product is easier to weigh and ration.
- There is less waste during feeding and, particularly with leafy legume or grass material, the sifting out of small particles of leaf and their loss in bedding is avoided.
- Less storage space is required.
- Transport costs are lower.
- Horses particularly prone to respiratory allergies (RAO, heaves) are less subject to dust irritation when given pelleted hay. Coughing is reduced in normal horses and bleeders are less prone to episodes of epistaxis.
- Older horses with poor teeth are inclined to masticate long hay incompletely, so occasionally precipitating colic through impaction.
- Hay belly is reduced.

Pellets do, however, have some disadvantages:

- Incorrectly pelleted material is crumbly so that fines are lost
- It is difficult to assess the quality of pelleted hay visually.
- Horses can choke on pellets of about 12 mm (0.5 in) diameter, but episodes are normally overcome without intervention. The problem is said to be more common when pellets are fed from the hand, but might be avoided by placing a large spherical rock (too large to be chewed) in the manger, forcing the horse to eat around it.
- Wood chewing (also see Crib-biting, Chapter 11) and coprophagy (faeces eating) are more prevalent where pelleted hay is given without any long hay. The provision of 0.25–0.5 kg long hay/100 kg BW daily, or good-quality straw bedding, is normally sufficient to minimize these problems. Their incidence seems to be less, but is not eliminated, when wafered hay containing hay particles 4–5 cm (1.5–2 in) long is used. A relationship exists between the caecal environment and the incidence of wood chewing in horses given hay or grain. A diet of all hay induces a higher proportion of acetate in the VFA of the caecal fluid (see Table 1.4), whereas a low caecal-fluid pH and high propionate content induces wood chewing (Willard *et al.* 1977).The propionate content is unlikely to account for the habit of crib-biters, as they spend less time eating and resting than do normal horses and their reduced feed intake (P.D. McGreevy personal communication) could account for an extended large-intestinal transit time for ingesta with a normal pH (see Gastric erosion, Chapters 6 and 11).
- Although the grinding and pelleting of hay do not affect the digestibility of protein, the digestibility of both DM and CF is decreased slightly, possibly owing to a decreased time taken to consume a given amount of feed. From a practical viewpoint the effect on digestibility is more than offset by the reduction in wastage.
- Grinding and pelleting can increase hay costs by up to 10%.

Feeding time can be influenced by the method of processing the hay. Researchers in Hanover (Meyer *et al.* 1975b) recorded that horses took 40 min to consume either 1 kg long hay or 1 kg hard, pressed, wafered hay. They took longer to consume chopped or ground hay but less time to eat soft pressed wafers. Hay of poor quality and high fibre content took longer to eat than better quality hay. Highly digestible chopped maize silage was eaten much more rapidly than hay. Horses of between 450 and 500 kg made between 3000 and 3500 chewing movements in consuming 1 kg long roughage, but only between 800 and 1200 such movements in eating 1 kg concentrate. However, ponies of between 200 and 280 kg required twice as long to eat hay and a concentrate meal and between three and five times as long to eat whole oats or pellets. They made between 5000 and 8000 chewing movements in consuming 1 kg concentrate. The ingesta of horses given chopped hay, or ground hay, passed more rapidly through the stomach than did that of those given long hay and the former led to more fluid stomach contents.

Several other reports have clearly shown that the digesta of horses given ground hay passes more rapidly through the GI tract (Wolter *et al.* 1974), notwithstanding the evidence that horses normally masticate roughage to particles of less than 1.6 mm long before it is swallowed (Meyer *et al.* 1975b). The mean rate of passage of long meadow hay was 37 hours compared with 26 and 31 hours for ground meadow hay, and ground and pelleted meadow hay, respectively. The decrease in fibre digestibility experienced on grinding almost certainly is a function of rate

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of passage through the GI tract. However, by the same token, the faster the rate of passage becomes the greater the capacity of the horse for feed; an extension of the time for each meal of pelleted roughage might, however, improve the digestibility of fibre.

Where horses are given a choice of loose hay, wafered hay and pelleted hay, more is consumed of the latter two than of the loose hay. Horses prefer stables in which several forages, rather than one, are provided and this reduces straw bedding consumption. Nevertheless, horses possess a motivation to move between foraging locations regardless of palatability (Goodwin *et al.* 2002, 2007). Horses prefer grain to either chopped or long hay, and if given a chopped hay–grain mixture they are inclined to sort out the grain; however, such a mixture frequently affords a useful function of depressing the rate of grain consumption by a greedy feeder. The consumption of concentrate feed before, rather than after hay, causes a more intense mixing of ingesta and less variation in the concentration of VFA in the large intestine (Muuss *et al.* 1982). Other evidence (Cabrera *et al.* 1992) indicates that the consumption of roughage before concentrates improves amino acid utilization from digested proteins (see Chapter 2).

For stabled horses, long hay is given on the floor in the corner of the box, in a hay rack or in a net. The latter should be placed sufficiently high to avoid the possibility of a horse entangling its hoof in an empty net. The amount of hay wasted is greatest where it is placed on the floor, but this procedure leads to less atmospheric dust.

Dried grass nuts

Grass, clover, lucerne (alfalfa) and sainfoin crops are frequently cut when green and leafy, artificially dried, and preferably chopped and pelleted with a moisture content of about 120 g/kg. In the UK the product must have a CP content of at least 130 g/kg, on the assumption that the moisture content is 100 g/kg, to be designated 'dried grass'. High protein grass nuts contain approximately 160 g protein/kg. Dehydrated alfalfa manufactured in the USA contains 150–170 g protein/kg (90 g moisture/kg). These products contain little vitamin D_2, but are rich sources of high quality protein, β-carotene, vitamin E and minerals, well suited to horse feeding and of relatively balanced composition. Products rich in legume forage frequently have a Ca : P ratio too wide, and a protein content too high to form the entire diet. They should be supplemented with a cereal product rich in P.

The artificial drying of green forage yields a product more valuable than hay, as the raw material to be dried is less mature, leaves are not shattered and lost, moulding is avoided and dustiness is minimal. Hyslop *et al.* (1998b) reported that the CP and NDF digestibilities were both higher for the artificially dried material than for hay when a perennial rye-grass crop was cut for both purposes on the same day. It was assumed that there was lower leaf loss for the dehydrated grass. The only disadvantages are the absence of long fibre, cost and the fact that contents of β-carotene and α-tocopherol are variable and influenced particularly by length of storage. As much as half the vitamin A potency (which initially can be equivalent to 30 000–40 000 iu vitamin A/kg for horses) can be lost during the first seven months of storage where facilities are not ideal (see 'Feed storage', this chapter).

FUNCTIONS OF HAY AND USE OF OTHER BULKY FEEDS

Haylage

Fibre and bulk are useful attributes for part of the horse's diet. By diluting more readily fermentable material, fibre suppresses a rapid fall in pH of the large intestinal contents and, by stimulating peristaltic contraction, feed with these characteristics aids the expulsion of accumulated bubbles of gas. There are many alternatives to hay as sources of fibre and for horses with sound teeth several are useful where reliable hay cannot be obtained. Best-quality silage and haylage can be fed to most horses. Good-quality acidified grass silage with a high content of DM replaces between one-third and all horse feed; but success depends on its composition, freedom from abnormal fermentation, skill of the feeder, and on the horse. Compensation should be made for its deficiency, compared with grass, in potency of vitamins A and E. Horses suffering from respiratory allergy should benefit most by changing from hay to silage. Very acid silages should be avoided. Silage with a low DM (less than 25%) , and baled or bagged material with a slightly higher DM, but with a pH of around 6, can lead to a greater risk of abnormal clostridial fermentation (see Chapter 10). Also, it occasionally precipitates explosive intestinal fermentation and colic if improperly fed. The reason for this could be that the rate of intake of highly fermentable DM is much greater than it is in the form of long hay.

Waste products

Good-quality spring barley or wheat straw in small quantities acts as a source of fibre for horses with sound teeth but is deficient in most nutrients. The inclusion of various waste products in complete diets has been examined, particularly in France and the USA. These products include

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dried citrus pulp, which is quite satisfactory (Ott *et al.* 1979b), soya hulls and even sunflower hulls, almond hulls, corrugated-paper boxes and computer paper. Digestibilities for the last two seem to be about 90 and 97%, respectively, but are much lower for the sunflower and almond hulls because of heavy lignification. The characteristics of other bulky feeds are discussed elsewhere in this chapter. With increasing competition between domestic animals for feeds, and an expanding world human population, undoubtedly the search will continue for satisfactory and safe means of sustaining a healthy population of domesticated herbivores by the greater use of waste products of human activities.

Alkali treatment of roughage

Treating straw with NaOH – nutritionally improved straw (NIS) – increases its digestibility to the horse (Mundt 1978) and, with dietary adjustments to its sodium content, the product is an important supplier of dietary fibre. Ammonia-treated straw also has promise (Slagsvold *et al.* 1979), but results in cattle and horses have been mixed. The potential digestibility of poor-quality roughage is difficult to predict by chemical analysis, as factors that inhibit complete digestion of plant cell-wall polysaccharides include a difference in structural organization, as well as differences in chemical composition of those structures (see Chapter 2).

Group feeding

Feeding habits and hunger of stabled horses can vary enormously and succulent roughages are sometimes used to stimulate animals with flagging appetites. One French study (Doreau 1978) showed that the intake *ad libitum* of a dry feed by a group of stabled horses varied from 8.1 to 19.2 kg/day and the time spent eating ranged from 6 h 40 min to 15 h 50 min. The horses ate several large meals and some small diurnal and nocturnal meals. The night meals represented 30% of the total intake. Several factors contribute to the fastidiousness of finicky eaters, such as environmental stress and nervousness, unpalatability and monotony of ration, nutritional deficiencies, poor health and teeth, lack of exercise and peck order (hierarchy) among group-fed horses.

Succulents

Many succulent vegetables and fruits (e.g. sugarbeet roots, carrots, apples, pears, peaches and plums) are satisfactory as treats for horses. Peaches and plums should be stoned, and hard root vegetables should be sliced into strips, to avoid choking, and then mixed with compounded nuts or grain. Carrots contain over 100 mg carotene/kg and care should be exercised in the quantities used (not more than 0.5 kg fresh material/100 kg BW daily) if supplements of vitamin A are provided. A similar attitude should apply to other treats as they represent an unbalanced feed and in large quantities (more than 20% of total DM intake) can do more harm than good. It should also be realized that succulents, including both root vegetables and fruit, contain 80–90% water, and on a DM basis they are an expensive source of energy and protein. Only if they are relatively cheap can succulents be justified and bulkiness restricts their role to that of a supplement to normal rations. Of all the main flavour groups, the horse is deterred by sour tastes and attracted by sweet flavours in moderation.

COMPOUNDED NUTS

Several ingredients are incorporated in a ground form, among them the common cereal grains, oilseed meals, milling, brewing and distilling by-products, dried grass and lucerne, fishmeal, and mineral, trace element and vitamin supplements. Their principal role in this form is to provide a balanced source of all nutrients, but they have to be supplemented with loose hay as a source of long fibre, with water and sometimes with common salt. Nuts rich in nutrients and of high digestibility can be given to young foals, high-energy nuts can be given to horses in hard work, and low-energy nuts can be provided for adult horses engaged only in light work. The advantages of nuts include standardized diets for particular purposes, constant quality, extended shelf life, freedom from dust, palatability, and uniform physical characteristics and density, all of which facilitate routine feeding.

Compounded nuts, particularly high-energy, nutrient-dense formulations, should be introduced gradually to give the horse and its microbial flora time to adapt. A too-rapid introduction of nuts, or for that matter of oats, sometimes leads to slightly loose droppings during the first two to three weeks, 'filled-legs' and even to colic. Complete nuts are also manufactured for feeding to horses in the absence of hay, but, generally, these should be used only for a greater control of dust where horses are subject to respiratory allergies. In the absence of loose hay, wood chewing and some other vices, including coprophagy, are more prevalent. Nevertheless, *ad libitum* access to a complete pelleted diet, one containing 29% CF (53% NDF), enabled three-year-old pony mares to establish natural feeding patterns with minimal vices. Feed intake exceeded maintenance during the first four weeks, but returned to maintenance levels subsequently (Argo *et al.* 2002).

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The preparation of feed in nut form has the disadvantage that the user is unable to recognize good-quality from poor-quality ingredients; but some indication of the product's chemical nature can be conjectured by reference to declared analyses required by law in the EU and found on a ticket attached to the bag. The Statutory Statement should give the following information for complementary and complete feeding stuffs:

- the name or trade name, the price, country of origin and address of the person responsible for the particulars of the Schedule;
- the net quantity and minimum storage life (or batch number), and the moisture content of compounds if it exceeds 14%;
- whether a permitted antioxidant, colourant or preservative is included;
- the active levels of vitamins A, D or E, if included, and the shortest period over which the activities apply;
- the name of any Cu additive and the total level of Cu (naturally present plus added);
- the name of the additive and binding agents present;
- details of any enzymes or microorganisms added (see 'Probiotics', this chapter);
- information may be included on total levels of other trace elements and the total levels of other vitamins, provitamins and vitamin-like substances, including the minimum period for their activity;
- ingredients in descending order by weight;
- the percentage by weight of oil (lipids extractable with light petroleum, 40/60°C boiling point without prior hydrolysis, except in the case of milk products);
- the percentage by weight of CP (the nitrogen content multiplied by 6.25);
- the percentage by weight of CF (principally organic substances remaining insoluble following alkali and acid treatment);
- the percentage by weight of total acid-insoluble ash;
- levels of lysine, methionine, cystine, threonine and tryptophan may be included;
- levels of starch, Ca, Na, P, Mg and K may be included (levels of Ca, if over 4.9%, and P, if over 1.9%, must be included).

Table 5.4 gives recommended declarations and some chemical values for compounded horse feeds.

A range of sizes of compounded nuts has been found suitable for feeding to growing and adult horses. However, the optimum seems to be a diameter of 6–8 mm and a length of 12 mm. For very young foals being given a milk substitute nut, a diameter of 4–5 mm and a length of 6–7 mm is probably more suitable. South African work (van der Merwe 1975) indicates that acceptability is not affected measurably by hardness of nut, although most horses dislike nuts that crumble too readily, and those that are excessively hard are occasionally bolted without mastication. Smaller nuts are chewed more slowly and require more time for a given amount to be consumed – a decided advantage.

Where horses are in especially hard work, up to 80% by weight of the total ration can be provided in the form of nuts or grain and supplements, with the remaining 20% composed of hay. Regimens of this nature require considerable skill, four to five feeds per day and regular exercise every day. A much more typical regimen for stabled horses in strenuous work is a ration of 50–60% by weight concentrate and 40–50% hay. As the amount of work is reduced, so the proportion of concentrate can be decreased, or that of lower energy can be used. In stables where horses are given their concentrate by the bowl, compensation for their differences in bulk density and energy density must be allowed. A unit volume of barley is about three times the weight of the same unit volume of wheat bran. Furthermore, the common cereals have different amounts of digestible energy per unit weight. The combined effects of energy density and bulkiness imply that, for example, a unit volume of maize contains nearly double the DE of the same volume of oats. (Table 5.5 gives conversion values.)

COARSE MIXES

Some horses in strenuous work have poor appetites and are more likely to 'eat up' when given a coarse mixture (sweetfeed) than when given nuts in similarly large amounts. Coarse mixtures have thus gained in popularity, although it is evident that poor appetite for nuts is due in part to similar volumes and therefore larger weights of nuts being offered, when refusals would be expected. Coarse mixes should be complete, to be supplemented only with loose hay, water and sometimes with common salt. They tend to be more expensive to produce than compounded nuts, but have an advantage in being less dense, and normally contain a proportion of cooked, flaked cereals and oil seeds and expeller oil-seed cakes. Argo *et al.* (2002) gave pony mares *ad libitum* access to either pellets or a coarse mix of the same composition. They found that DE intake was greater with the pellets, but that each meal was less frequent and of greater duration, with a lower bite rate and a lower DM intake per minute for the coarse mix and horses tend to prefer high-fibre mixes of good quality, to low-fibre mixes (Goodwin *et al.* 2005a).

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Table 5.4 Recommended declarations and chemical values for compounded nuts and coarse mixtures (assuming 88% DM).

	Crude oil (%)	Crude fibre (%)	Crude* protein (%)	Total lysine (%)	Digestible energy (MJ/kg)	Total ash (%)
Foals						
1 month before weaning to 10 months old	4–4.5	6.5–7.5	17–18	0.9	13	7–9
11–20 months old	3–3.5	8.5	15–16	0.75	11	7–9
Adults						
Strenuous work	3.5–4	8.5	12–13	0.55	12	8–10
Light to moderate work, barren mares and stallions	3	14–15	10–10.5	0.45	9	9–10
Last quarter of pregnancy, lactation and working stallions	3	9–10	13–15	0.65	11	8–10

*Actual protein concentrations are less important than are the total lysine contents. Note: lysine and DE are not normally declared.

Table 5.5 Weights of common cereal grains and soya-bean meal and average DE and UFC (MJ/10L) values per unit volume.

	Weights			
	lb/bushel	kg/10 L	DE	UFC
Oats	27–45	3.4–5.6	51.7	3.9
Barley	36–55	4.5–6.9	73.0	5.7
Wheat	50–62	6.2–7.7	98.0	7.5
Milo (sorghum)	51–59	6.4–7.4	89.7	6.4*
Maize	46–60	5.7–7.5	93.7	7.4
Soya-bean meal (solvent extracted, 44%)	47–53	5.9–6.6	83.1	5.7
Wheatbran	17–21	2.1–2.6	25.4	1.8

*NE value of milo estimated.

An advantage that might accrue from the extension of eating time is the lower glycaemic response produced by a sweet feed cf. a 4 mm pelleted concentrate of similar starch content (Harbour *et al.* 2003). On the other hand, the shelf life of mixes is less than that attributed to compounded nuts and their bulkiness demands proportionately more storage space. Storage of all feeds should be in dry, cool, ventilated conditions where there is little variation in temperature, otherwise moulding can occur.

CEREALS

Whereas water is probably the most critical nutrient for the horse's immediate survival, fatness and lack of exercise are the horse's worst enemies. The adequate control of energy intake is the most difficult aspect of optimum and reliable feeding. Cereals (Plate 5.3) are the principal source of energy in the diet of hard-worked horses and therefore a brief discussion of the characteristics of the common cereal grains and their by-products is appropriate.

Cereal grains contain from 12 MJ to more than 16 MJ DE/kg DM compared with about 9 MJ/kg in average hay. Cereal grains embody three main tissues: the husk and aleurone layer, endosperm and the embryo. The endosperm is a rich store of starch required as a source of energy during the early growth of the plant. Of the nitrogen compounds of cereal grains 85–90% are proteins; these are found in each of the tissue regions but are in

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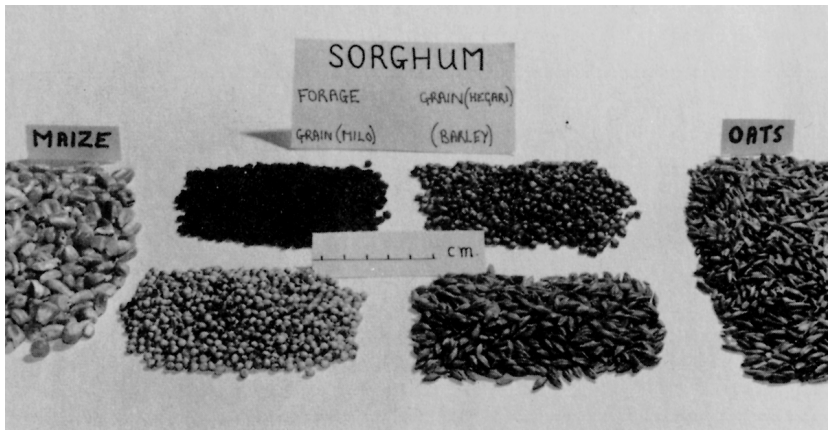


Plate 5.3 Cereal grains. Maize (corn) grains are the largest and can be given whole to horses with sound teeth, but as they can be very hard, cracking is often worthwhile. Barley is smaller and relatively hard and the grains should be crimped or rolled lightly. Oat grains are relatively light and bulky, and crimping or rolling is required only for young horses or for older horses with poor teeth. By comparison, sorghum grains are small, as they are 'naked'. Sorghum is grown in hot dry countries and white varieties are quite satisfactory for horses when coarsely ground, cracked, rolled or cooked. The brown varieties contain large quantities of tannins and are unsuitable for horses.

higher concentrations in the embryo and aleurone layer. Cereal proteins are poorer than oil-seed and animal proteins because they are relatively deficient in lysine, threonine and methionine. The quality of protein (as distinct from the amount) decreases in the order oats, rice, barley, maize, milo, wheat and millet. Oat protein contains slightly more lysine than does the protein of other cereals.

The oil content of cereal grains varies from about 15 to 50 g/kg, with oats containing slightly more than maize, which in turn contains more than barley or wheat. This oil is rich in polyunsaturated fatty acids, of which the principal one is linoleic acid. This generally constitutes about half the fatty acid composition by weight in the oil. Unsaturated oils such as these, are prone to rancidity subsequent to the grinding of cereals unless the meal is compressed into pellets or otherwise stabilized.

Cereal grains are deficient in Ca, because they contain less than 1.5 g/kg, but they have three to five times as much P, principally in the form of phytate salts. These salts tend to reduce the availability of Ca and Zn in the intestinal tract.

Palatability

Although different samples of one cereal species can vary considerably in quality, when given a choice ponies prefer oats to other common cereals. Comparisons made at Cornell (Hawkes *et al.* 1985) showed that the order of preference was oats, cracked maize, barley, rye and cracked wheat. Nevertheless, there was little depression in total intake when only the less palatable cereals were given.

Oats (*Avena sativa*)

Under traditional systems of feeding, in which a single species of cereal grain is given, oats are safer to feed than are the other cereals as their low density and high fibre content make them more difficult to overfeed and the grain size is more appropriate for chewing. They need, therefore, no crimping or rolling for horses aged over one year if the teeth are sound. A greater quantity of oats than of the other cereals must be consumed to produce founder or other digestive problems. However, they tend to be more expensive per unit of energy than the others, as between 23 and 35% of the grain consists of the hull. Figure 5.1 gives a cross-sectional view of oats and, in Appendix C, their chemical characteristics are listed for comparison with the other common cereals.

Naked oats (varieties produced by crossing *Avena nuda* with *A. sativa*)

The groat of *Avena sativa* is enclosed in a husk that constitutes 250 g/kg of the grain's weight. The husk of the

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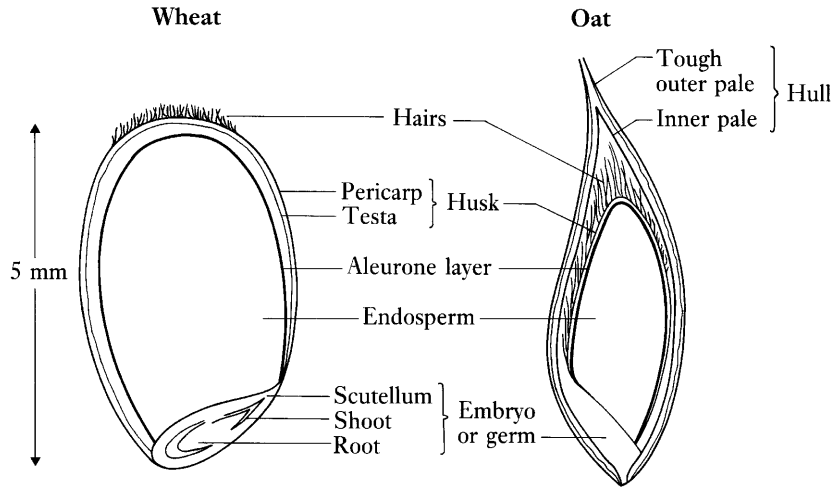


Figure 5.1 Cross-sectional view through wheat and oat grains.

new varieties is loose and falls off during threshing, hence the energy content of naked oats is considerably higher than that of the grain of *Avena sativa*. Naked oats typically contain 120 g/kg CP and 6 g/kg lysine. The P content is high (3.5 g/kg) but is principally present as phytates and the oil is prone to lipolysis during storage. It is advisable to restrict naked oats to 100–200 g/kg of the cereal mix of horses.

Barley (*Hordeum vulgare*)

Oats and barley differ from wheat, maize and grain sorghum in being invested in a hull (botanically known as the inner and outer pales), which the other three cereals have lost during harvesting. All the cereal grains are, however, encased tightly in the thin membrane composed of the less-fibrous fused testa and pericarp (Figure 5.1).

The barley hull constitutes 10–14% of the total grain weight and the hull is relatively smaller and more tightly apposed to the grain than the hull of the smaller grain of oats. For feeding to horses, barley grain should be crimped or lightly rolled to rupture the case shortly before feeding, but it can be fed as the only cereal after a period of gradual adaptation. This period is necessitated by the higher starch content and bushel weight of barley in comparison to oats. Processes that gelatinize starch grains, such as steam rolling or micronization, are discussed later in this chapter (see 'Cooking'). Barley protein contains slightly less lysine than oats, and the oil content is less than 20 g/kg.

Some varieties of naked or hull-less barley, very low in fibre, have been bred. They are comparable with naked oats or oat groats from which the hull has been removed by processing, again yielding a starchy, high-energy, low-fibre product, but their price rarely justifies their use in horse feeding.

Wheat (*Triticum aestivum*)

The grains of wheat (Figure 5.1) are free of hull and relatively small so that they can escape mastication if fed whole. Wheat should be cracked, coarsely ground or steam flaked before use. The two endosperm proteins (known collectively as gluten) are deficient in lysine and can form a pasty mass (similarly for rye grain) impenetrable for digestive juices, especially when wheat is finely ground. In the uncooked state wheat should form less than half the grain fraction of the diet. Moreover, as the starchy endosperm constitutes 85% of the grain, excessive intakes can cause digestive disturbances, particularly if the adaptation period has been short. The bran and germ make up about 13% and 25% of the grain, respectively.

Spelt (*Triticum spelta*)

Spelt is a hexaploid species of wheat. Spelt was an important staple in parts of Europe from the Bronze Age to medieval times; it now survives as a relic crop in Central Europe and has found a new market as a health food. It is a wheat species known from genetic evidence to have originated as a hybrid of a domesticated tetraploid wheat, such as emmer, and the wild goat-grass *Aegilops tauschii*. This hybridisation must have taken place in the Near East, as this is where *Ae. tauschii* grows, prior to the appearance

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of bread wheat (*Triticum aestivum*, a hexaploid free-threshing derivative of spelt) in the archaeological record c. 8000 years ago. It is sometimes considered a subspecies of common wheat (*T. aestivum*), in which case it is known as *Triticum aestivum* subsp. *spelta*. The chemical composition of spelt has been compared with barley, oats and maize (Fayt *et al.* 2008) and is characterized by rather low protein and ether extract (EE) contents. The NDF content of spelt is slightly higher than that of the oat but the ADF content is lower. It is well utilized by horses.

Triticale

Triticale is a hybrid resulting from crossing wheat *(Triticum)* and rye *(Secale)*. It contains more protein than barley, but in variable amounts (100–200 g/kg)that are deficient in the amino acid tryptophan (1.5 g/kg cereal), but richer in lysine than wheat protein. It could contain higher concentrations of trypsin inhibitors and alkyl resorcinols than wheat or barley. In ground form it should have a feeding value for horses slightly greater than that of barley. The triticale cultivar Madonna has a higher small intestinal digestibility cf. wheat, leading to a lower caecal starch flow (Brown *et al.* 2001). Some varieties are subject to ergot infection and samples should be checked for its presence.

Maize (corn) (*Zea mays*)

Maize is the largest of the cereal grains and is acceptable in any form for feeding to horses. If the grains are very hard, they should be cracked, especially for horses with poor teeth. Maize contains twice the energy per unit volume of oats, but oats have a higher small intestinal digestibility, so the glycaemic index of whole oats is greater than that of cracked maize (Pagan *et al.* 1999a). A considerable portion of maize starch is fermented in the hind-gut when large amounts are given, unless the grain is cooked (see Chapter 1 and Figure 6.10). The glycaemic response to steam flaked maize is greater than that to either cracked or ground maize (Hoekstra *et al.* 1999), indicating that cooking improves small intestinal digestibility.

The so-called 'heating' of cereals generally results from the rapid assimilation of their products of digestion and their rapid fermentation by intestinal microorganisms (with a fall in caecal pH). This causes an abrupt increase in the heat of fermentation and a rise in the concentrations of glucose and VFA in the blood, stimulating metabolic rate to a greater extent than occurs after a meal of hay. The grains of maize contain about 650 g starch/kg, but only 80–100 g CP. The endosperm protein, zein, is deficient in tryptophan and to a lesser extent in lysine, but the protein of the germ, in common with the germ of other cereals, is of better nutritional quality.

Yellow and white maize varieties are produced, but the types for stock feeding are predominantly yellow and contain the xanthophyll pigments cryptoxanthin, a precursor of vitamin A, and zeaxanthin. Infrequently, some maize imported into the UK is more than two years old, when it is a poor source of vitamin A.

Grain sorghums (*Sorghum vulgare subglabrescens*)

Sorghums are the main food grain of Africa and parts of India and China where they are grown on land too dry for maize. They are also a major stock feed in dry areas of mid-west USA. The kernel is naked, like that of maize and wheat, but more spherical in shape and smaller than that of wheat. The protein content of the grain is variable (80–200 g/kg). The lysine content of the protein is rather low, and the oil content of the grain is less than that of maize. Owing to its size, the grain should be rolled, cracked, coarsely ground or steam flaked before feeding to horses. This disrupts the waxy bran covering the endosperm. If finely ground it becomes pasty and rolling is preferable. It is a high-energy cereal and, therefore, to avoid digestive disturbances, it should form only a portion of the cereal intake.

Several sorghum varieties are grown, including some for forage use. The grains range in colour from white to deep brown. The white, or milo, varieties of grain sorghum that contain only low concentrations of tannins are widely used as a horse feed. Those varieties that are brown, or purple, contain significant amounts of tannins (Plate 5.3) that can cause colic.

Rice (*Oryza sativa*)

The rice grain is invested in a thick fibrous hull, which is easily removed but which constitutes about 20% of the total weight. The hull is rich in silica and when freed from the grain it is unsuitable on its own for feeding to horses, because the sharp edges cause irritation. Rough rice, that is the grain before the removal of the hull, is more suitable as a horse feed. Rice protein is a reasonable source of lysine.

Millet (*Setaria* spp., *Panicum miliaceum*)

The name millet is applied to a range of grass species. The seeds have a high energy content, are the palatable staple diet of many people and typically contain 110 g/kg protein of low lysine content, 50–90 g/kg CF and 25–35 g/kg oil.

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They are free from toxins except for the unripe seed husks of *Paspalum scrobiculatum*. However, millet is not readily available in Europe and any that is could have been rejected for human consumption for reasons of mould or admixture. The seed is small and the hull is not removed during normal harvesting. It requires coarse grinding, or crushing, for feeding to horses and its feeding value is similar to that of oats.

Processing of cereal grains

Specific procedures for processing each species of cereal grain have already been described. Where cereals are mechanically rolled the process should be one of kibbling, or bruising, rather than the complete rolling of uncooked material, otherwise the chemical stability of the product is jeopardized, no further increase in digestibility is achieved and greater processing costs are incurred. A similar argument can be advanced for coarse, in contrast to fine, grinding. Moreover, finely ground cereal endosperm is floury, unpalatable, less stable and could lead to digestive disturbances. Some other feeds ball up and block the oesophagus if fed dry and are therefore normally dampened, mixed with cut or chopped hay, or fed damp with oats. Cereal grains, or high-energy nuts, should be distributed among as many daily feeds as possible to minimize the risk of colic.

(1) Cooking: expansion (extrusion)

Cereals should be cooked only in the presence of water in order to minimize the risk of heat damage to proteins and oil. Steam pelleting and expansion procedures achieve this objective and, for high-energy materials in particular, have been shown to improve the digestibility of DM, OM, starch and the nitrogen-free extract of cereals without interfering with the digestibility of CP (see Chapter 1). The digestibility of the CF, which represents only a relatively small portion of high-energy products, is either not affected or is only slightly depressed if the food material is ground beforehand. The process of expansion, or popping, relies on the cooking effects of superheated steam injected into a slurry compressed against a die face by a revolving worm and the subsequent rapid fall in pressure during extrusion. Material is subjected to a temperature of around 110–120°C for less than a minute. Processing costs, which constitute more than 10% of the value of the product, are difficult to defend, except for young stock and horses under competition rules. It is hard to quantify some of the indirect advantages, which include increased stability of product, improved palatability, destruction of natural toxins, insect pests and bacterial pathogens, and the avoidance of high-starch concentrations in the large intestine. The last is of prime import.

(2) Cooking: micronisation

Other cooking procedures include the traditional steam flaking of maize (during which the grain is passed through heated rollers), the roasting of oil seeds for the industrial extraction of oil, and the micronisation of cereals and vegetable protein seeds. For micronisation, a moving belt carries a thin, even layer of seeds horizontally beneath a series of ceramic burners that emit infrared irradiation in the 2–6 µm waveband. This results in a rapid internal heating of the seeds and a rise in water-vapour pressure, during which the starch grains swell, fracture and gelatinize. The product is then passed through helically cut rollers and from a cooler to a cyclone. The raw material achieves temperatures ranging from 150 to 185°C for 30–70 s; for each specific raw material there are optimum values within these ranges. These products are frequently included in coarse mixtures for horses; the process increases digestibility and, e.g. in the case of soya beans, will inactivate antiprotease and some other natural toxins.

A high digestibility of starch in the small intestine is important for the avoidance of colic and related metabolic diseases. McLean *et al.* (1998a) incubated monofilament polyester bags containing unprocessed barley, micronised barley and extruded barley in the caecum of ponies offered grass hay *ad libitum.* The initial degradation rate of starch in micronised barley was greater than that of unprocessed barley, or that of extruded barley. The results indicate a preference for micronisation over extrusion, but increased microbial degradation rate does not necessarily imply better small intestinal digestion and rapid bacterial degradation is likely to promote acidosis. Subsequently, McLean *et al.* (1998b) measured the acetate : propionate ratio of VFA in caecal fluid of ponies given micronised, extruded or rolled barley or hay cubes. The ratio decreased in the order hay cubes > micronised barley > extruded barley > rolled barley. This indicated that greater precaecal starch digestion occurred with micronised barley than with either extruded or rolled barley. Vervuert *et al.* (2008) compared diets containing rolled, micronised or extruded barley; the barley intake was adjusted to supply 2 g starch/kg BW daily. Extruded barley was digested to the greatest extent in the small intestine and rolled barley to the least extent, however extruded barley was readily fermented. Whether expansion or micronisation is to be preferred

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depends not only on costs, but on the conditions (temperature, time, moisture content, etc.) under which each process is carried out.

(3) Acidification

The alkali treatment of roughage has been briefly described already (this chapter). Treatment of high-moisture cereal grains with propionic acid has achieved a certain measure of popularity with harvesting in inclement weather. The acid acts as a mould inhibitor and preservative. Grain treated in this way is only marginally suitable for feeding to horses, owing partly to its acidity and more especially to the presence of mouldy patches in the silo. The grain can become infected by the fungus *Fusarium*, which produces the toxin zearalenone known to cause 'poor doing' in all animals, and infertility (see also 'Grass Sickness', Chapter 10). Furthermore, high-moisture cereals are deficient in α-tocopherol so that supplementation with synthetic forms of vitamin E at a level of about 30 mg/kg feed is essential.

Cereals as supplements to hay

C. Drogoul (personal communication) gave ponies chopped meadow hay and rolled barley in ratios of 100% hay down to 50% hay. As the proportion of barley increased, mean retention time increased, but the digestibilities of NDF and ADF decreased, probably indicating that ≥30% barley led to the presence of starch in the hindgut and the partial suppression of cellulolytic bacteria (see also Chapter 2).

Cereal by-products

The industrial use of cereal grains leads to the production of two major by-products: (1) those derived from the milling industries (the seed coats and germ) and (2) those derived from the brewing and distilling industries (principally spent grains, the residues of germinated grains and dried yeast).

Wheat, oat and rye milling by-products

Undoubtedly the major milling by-products fed to horses in Western countries are those derived from wheat milling. The offals of wheat consist of the germ, bran, coarse and fine middlings, which comprise about 28% of the total weight of the grain and collectively are known as wheatfeed, although in some products a proportion of the germ is marketed separately. The germ contains 220–320 g CP /kg and is a rich source of α-tocopherol and thiamin. This particular by-product is too expensive for general use but can be of value to sick animals. Bran is derived from the pericarp, testa and aleurone layers surrounding the endosperm, with some of the latter attached. It normally contains 85–110 g CF/kg and 140–160 g CP. It is sold either as giant, broad or fine bran according to size, or as entire fraction 'straight-run bran'. These grades are similar in chemical composition, although the larger flaked varieties contain slightly more water.

Bran is typically expensive for the nutrients it provides, but it can form, as a mash, a palatable vehicle for oral administration of drugs, and it has the capacity to absorb much more than its weight of water. Thus, it has a laxative action on the intestinal tract. Bran in particular, but also other wheat milling by-products, are rich sources of organic P, as bran contains approximately 10 g/kg, or slightly more, of which 90% is in the form of phytate salts. As bran is deficient in Ca and as phytate depresses the utilization of dietary Ca and Zn, the use of large quantities accelerates the onset of bony abnormalities in young and adult horses.

Coarse middlings are similar to bran but contain more endosperm and therefore contain only 60–85 g CF/kg and about the same amount of CP. Fine middlings contain even more endosperm than the coarse, and consequently only 25–60 g CF/kg. When adjustments are made for the imbalance in minerals, wheat by-products are safe feeds as supplements to horse and pony rations, besides being relatively rich sources of some of the water-soluble vitamins.

Oatfeed

During the commercial preparation of oatmeal for human consumption there are three by-products: the hull, dust consisting of oat hairs lying between the grain and the hull, and meal seeds composed of hull and the endosperm of small seeds. Each of these by-products can be fed to horses when appropriately processed and included in balanced feeds in proportions of up to 20% of low-energy diets. Oat hull has a CF content of 330–360 g/kg with a digestibility little better than that of oat straw. The hull and hairs are combined in a ratio of 4 : 1 to form oatfeed which in the UK should not, by legal definition, contain more than 270 g CF/kg. The lignin content of oat hulls varies from 9–61 g/kg among worldwide varieties. Oatfeed is a safe feed used in mixtures to reduce the energy concentration of starch-rich cereals for nonworking horses on the 'easy list'; but it is bulky and does not flow easily, so is frequently purchased in a pelleted form by manufacturers.

Rye bran

This is of limited acceptability to horses.

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Maize (corn) by-products

The by-products resulting from the industrial production of glucose and starch derived from maize include the protein gluten, a small amount of bran, and the germ. These by-products are similar to the analogous by-products of wheat.

Maize germ meal

The maize germ is removed from the endosperm during the process of corn-starch extraction. Much of the corn oil is then removed from the germ leaving the extracted maize germ to which other maize offal products, bran and gluten, can be added. The composition is therefore variable, depending on the details of the manufacturing procedure. The residual oil is polyunsaturated and subject to peroxidation. It is a palatable feed, the protein has a reasonably good amino acid balance and there are no toxic factors present in a well-manufactured and stored product.

Maize gluten feed

This is the maize protein, gluten, together with maize bran, extracted maize germ and dried residues of the steeping liquors. The product is rich in xanthophylls, approximately 20 mg/kg, giving it a bright yellow colour. The gluten, free from other by-products, contains over 600 g CP/kg and is available as a branded product, Prairie Meal; typical material contains 180–230 g protein/kg, 70–80 g CF/kg and only 6–7 g lysine/kg, depending on the amounts of bran and germ remaining. Although the protein is of poor nutritional quality the feed is palatable, free from toxins, is a good source of some of the water-soluble vitamins and a useful constituent of horse feed mixtures.

Rice bran

The quality of rice bran depends on how efficiently the indigestible, siliceous hulls have been removed before the husk is detached from the grain. The bran from the first-stage milling consists principally of the husk (pericarp and testa), the germ and part of the aleurone layer. These components are incompletely removed and the remaining by-product (second-stage milling) constitutes rice polishings, which also contain some endosperm. The first- and second-stage milling by-products are sometimes combined to form rice pollards.

Large quantities of rice meal, or rice bran, are produced globally. This consists of the husk, aleurone layer, germ and some of the endosperm. Residual hull should be at a minimum. The bran has a crude composition of 90–210 g CF/kg, 100–180 g CP/kg and 110–170 g lipids/kg, the latter consisting of a very unsaturated fat. This fat becomes rancid rapidly and is therefore extracted, leaving a product of better keeping quality. However, stabilized rice bran is available.

Extracted rice bran is widely available and is a good supplementary feed for horses when used as a component of a mixed ration. The extracted by-product has a composition of about 15 g oil, 130 g CP and 120 g CF/kg. However, frequently as much as 60 g silica/kg are present and the ash content of the bran ranges from 100 to 240 g/kg, reflecting the amount of hull that still remains. Extracted rice bran is also a rich and variable source of P (11–22 g/kg), much of which is phytate-P. Where good-quality rice bran is obtained, 150 g/kg is included in balanced concentrate feeds. Nevertheless, many Eastern sources send products to the West already seriously contaminated.

Brewing by-products

Three major by-products are derived from brewing: malt culms, brewer's grains and brewer's yeast. When barley is sprouted for the purpose of hydrolysing the starch, the resulting malt sprouts, which include the embryonic radicle (root) and plumule (stem), remain after the malting process. These are removed and dried to form the malt culms. The remainder of the material is mashed to remove sugars, leaving the grains which are disposed of as a wet by-product or dried and sold as dried brewer's grains.

Malt culms (malt sprouts)

Dried malt culms contain 12–30 g oil and 140 g CF/kg. The CP content is typically 240 g/kg, but is very variable and represented by a proportion of NPN. The lysine content is typically 12 g/kg. The by-product is dusty and not very palatable; it swells on wetting, stimulating peristalsis, but can contain moulds if not fully kiln-dried. An inclusion rate for good material of up to 50 g/kg in feeds is satisfactory, but it should not be fed dry on its own.

Dried brewer's grains

The residual grains after removal of the wort include maize and rice residues, in addition to those of barley, the main constituent. The dried by-product contains 180–250 g CP and 140–170 g CF/kg and it therefore forms a useful adjunct to mixed feeds.

Dried brewer's yeast

The most coveted and expensive by-product of brewing is yeast, which in dry form contains 420 g high-quality protein/kg and is a rich source of a range of water-soluble vitamins and of P. This yeast is frequently fed to horses in poor condition at the rate of 30–50 g daily, but is

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too expensive for regular feeding (see Probiotics, this chapter).

Distilling by-products

The principal residues from the whisky-distilling industry are grains and solubles. The grains in the malt-whisky industry consist solely of barley residues, whereas grain whisky residues can in addition include those of maize, wheat, oats and rice. A proportion of the grains is sold wet, but significant quantities are dried.

Distiller's dried, or light, grains

Distiller's dried, or light, grain is the fibrous residue of barley and of other grains (blended whisky), remaining after washing out the sugars derived from hydrolysis of the starch and used for fermentation to alcohol. For whisky production, the alcohol is distilled off, leaving liquid pot ale from malt whisky production and spent wash from grain whisky production. This by-product is suitable for inclusion in horse feeds.

Distiller's solubles

After distillation of the alcohol, the spent liquor is spray-dried to yield a light-brown powder known as distiller's solubles, quite suitable for use in mixed feeds. Frequently, the dried solubles are added back to the dried grains and marketed as dried distiller's grains with solubles, known also as dark grains.

Distiller's dark grains

The liquid pot ale (or spent wash from maize and other grain distillation) contains unfermentable carbohydrates and products of yeast metabolism, such as protein and vitamins. Where maize is used for alcohol production, this liquid contains maize oil and after evaporation of water and addition of lime the residue is spray-dried. With the grains, the residue produces distiller's dark grains (DDS), or distiller's dried grains with solubles. This by-product, in small quantities, is a valuable supplement to mixed horse feed and American evidence suggests that dried maize grains with solubles stimulate the digestion of cellulose by microorganisms in the horse caecum. Bonoma *et al.* (2008) replaced corn and soyabean meal with **distiller's dried grains and solubles** in the diet of weanling horses. Weight gain and gain/feed ratio were similar for the two treatments, but the corn–soya diet had higher apparent digestibilities of DM, protein, ADF and NDF.

Malt whisky DDS from barley contains 270 g digestible carbohydrate/kg and 70 g ether extract/kg, whereas grain DDS from maize contains 180 g digestible carbohydrate/kg and 110 g ether extract/kg. DDS also contains: 100–130 g/kg CF, 260–280 g/kg CP and 8 g/kg lysine. The by-product from grain whisky is generally more digestible, but both DDS by-products are low in sodium, potassium and calcium. Both are free from toxic constituents, and rich but variable in Cu, malt DDS containing about 40 mg/kg, and grain DDS 80 mg/kg. Both are suitable for inclusion in horse feeds.

Cereal grain screenings

Cereal grain screenings are residues from the preparation, storage and treatment of cereal grains (barley, wheat, maize, sorghums and soya beans). They are extremely variable in quality and include broken grains, chaff, weed seeds and dust, and therefore moulds. Mycotoxins can be present. Each batch differs and their use should be in the pelleted form and strictly limited. The ash content varies from 20–180 g/kg, higher proportions indicating greater soil contamination, and the presence of broken grains contributes to an accelerated rate of rancid oil formation, particularly important in respect of maize.

Chaff and molassed chaff

Chaff is composed of the husk, or glumes, awns and other fibrous waste material derived from the threshing of grain. It is highly lignified, and not well fermented by intestinal microorganisms, but oat chaff, which is the best, is better utilized than straw alone. Chaff derived from barley threshing contains barley awns, which have serrated edges and cause some irritation. Nevertheless, it is a safe material to dilute energy-dense grains and is frequently marketed mixed with molasses at a concentration of about 500 g/kg. This product is palatable to horses and overcomes any dustiness of the chaff. Some molassed products include about 20 g/kg limestone; but these products are deficient in phosphorus. Some chaffs marketed for horses are based on chopped grass hay, 2 cm in length, mixed with molasses and limestone.

Sodium-hydroxide-treated straw

Straw treated with sodium hydroxide (NaOH) is an adjunct to mixed horse feeds and provides a useful source of palatable fibre. It should be recognized that, after NaOH treatment, cereal grains, will be deficient in vitamin E. Where the feed is also deficient in Se, nutritional degenerative myopathy can result.

Hydroponics

The practice of germinating barley seeds in lighted trays under humid conditions produces a high-quality feed of

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Table 5.6 Typical analytical values for hydroponic barley (g/kg DM).

Crude protein	120–170
Crude fibre	80–155
Ash	27–42
Digestible carbohydrate	550–740
Calcium	1.1–1.3
Magnesium	1.5–2
Potassium	4.6–4.8
Phosphorus	4.4–5.5

rapidly growing young plants. However, the cost per unit of DM in particular, owing to the inclusion of a realistic rate of depreciation for capital equipment and the high labour commitment, make the practice difficult to justify on economic grounds. The barley grains should not have been through a grain drier, which would decrease their capacity for germination, nor should they have been treated with mercurial seed dressings. They should be bright and free from broken grains. The time interval from germination to consumption should be minimized by establishing optimum conditions for growth of 20 h light per 24 h and an ambient temperature of 19–20°C. Slow growth increases the likelihood of moulding with its attendant risks. A build-up of mould spores in the room must be avoided by routine hygiene. About three-quarters of the product is moisture and therefore the DE content is only 2.5 MJ/kg despite a high digestibility (Table 5.6). In comparison with pasture grass, hydroponic barley has a very low content of Ca and K, whereas the P contents are similar. The Ca : P ratio is therefore quite different from that of grass.

OTHER LESSER INGREDIENTS AND BY-PRODUCTS

Carob-seed meals (*Ceratomia siliqua*) and locust beans (*Parkia filicoidea*)

The evergreen carob tree *(Ceratomia siliqua)* grows in Mediterranean countries. Its green pods fall from the tree in the autumn and are harvested. The hard seeds are embedded in thick, fleshy maturing pods that are unsuitable for grinding, owing to their high moisture content. They are kibbled, or crimped; the seeds fall out and are used in the confectionery industry, leaving the pieces of pod to be distributed to animal feed manufacturers. These dark-coloured pieces are very sweet, containing 400–450 g sucrose, 35 g CP, about 70 g CF and 160–240 g moisture/kg. It is not advisable to use batches containing more than 200 g moisture/kg for feed. The pods also contain tannic acid. The best sources are a useful feed for inclusion in coarse mixes. Major sources in Europe are: Spain, Portugal and Cyprus and the best quality may be from the latter source.

The West African locust-bean pods, from a spreading tree *(Parkia filicoidea)*, contain dark-brown, sweet, fibrous seeds in fibrous pods that are free from undesirable chemicals. The pod and seed, containing about 300 g sucrose/kg, are drier than those from the Mediterranean area and so can be ground, bulk handled and used in ruminant and horse rations.

Biscuit meal

Bakery wastes are variable and high in energy content, owing to a wide variation in fat content (5–280 g/kg) and high digestibility. In addition to wheat and other flours, the wastes contain sugar and salt. Rancidity and moulding are typical problems and a perennial issue for the Rules of Racing and of Competition is the inclusion of chocolate products in the wastes. In the past, this has frequently been the source of theobromine detected in urine.

Molasses

The crystallization and separation of sucrose from the water extracts of sugar beet (*Beta vulgaris saccharifera*) and sugar cane (*Saccharum officinarum*) leave a thick black liquid, termed molasses, which contains about 750 g DM/kg, about 500 g of which are sugars. The CP in molasses is almost entirely NPN, of minimal feeding value. In beet molasses, a proportion of this is in the form of the amine, betaine (a useful choline substitute) which is responsible for the unpleasant fishy aroma, whereas cane molasses has a pleasant smell. The sweet taste of both is attractive to horses in mixed feeds up to a level of 100 g/kg feed, and in these proportions molasses can act as a relatively effective binding agent in cubes. Cane molasses contains 5–11 g Ca/kg. The K content ranges from 20–40 g/kg in cane molasses and from 45–65 g/kg in beet molasses; cane molasses is reasonably rich in pantothenic acid and both contain around 16 mg niacin/kg (Appendix C).

Molassed and unmolassed sugar-beet pulp (*Beta vulgaris* var. *saccharifera*)

The shredded residue resulting from the extraction of the sucrose contained in the root of sugar beet is a wet product containing 750–900 g water/kg. This could be ensiled for horse feeding, but it is rarely used in this way. Most beet pulp is dried and marketed as dried sugar-beet pulp, or

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mixed with 10% molasses to form molassed sugar beet pulp, providing sugar, pectin and betaine, and sold normally in a cubed form. The glycaemic response as a percentage of that of oats, is 92 for molassed pulp and 72 for unmolassed soaked pulp (Groff *et al.* 2001), and there is no difference in the response to unmolassed soaked and expanded sugar beet pulp shreds (Coenen *et al.* 2003b).

Hyslop *et al.* (1998c) gave mature Shetland ponies 2 kg DM/day as grass hay and *ad libitum* access to either soaked molassed or unmolassed sugar-beet pulp. The total DM intake was lower than for ponies offered threshed hay only, although the DM intake provided 15% more DE than was required for maintenance. The intake and DM, CP and NDF digestibilities were greater for unmolassed pulp than for molassed pulp. Moreover, the partial replacement of oats by unmolassed beet pulp, at concentrations of 152 g and 108 g/kg DM, has little effect on energy digestibility (Lindberg & Karlsson 2001), although the energy sources differ metabolically. Sugar-beet pulp has a high apparent digestibility and appears to limit total feed intake, but molasses would seem to reduce the digestibility of fibre.

The dry cubes should not be given to horses on their own, without soaking, as in the dry form they can cause choke in some animals (see Chapter 11) and if dried beet pulp constitutes a high proportion of the concentrate mix then large quantities of free fluid are absorbed by the mass in the stomach and colic can result. The carbohydrate it contains is rich in pectins, but also cellulose and hemicellose that are fermented by the large intestinal flora of the horse. Mixed with other feeds, the dried form is used successfully in coarse mixtures and larger quantities, soaked, act as a useful cereal replacer in laminitis control ('Laminitis control', Chapters 2 and 11). It has a uniform composition and is free from toxins. It contains more Ca than do cereals (Appendix C), but it is a poor source of water-soluble vitamins, cf. cereals. The protein content and quality are similar to those of cereals. Urea is sometimes added to beet pulp. This is of no value to horses, but causes no harm in those with fully functioning kidneys.

Dried lucerne (dehydrated alfalfa)

Although not a by-product but a useful forage. Several indirect effects have been attributed to unidentified factors in dried lucerne. Its meal stimulates cellulose digestion by equine microorganisms and enhances GE digestion of feed. Lucerne hay is said to be protective in the development of glandular inflammation and to encourage white cell (lymphocyte) and red cell production in foals (Romić 1978). Pelleted lucerne meal is a better source of nutrients than grass hay or sun-cured lucerne (except for vitamin D_2), and the oxalates it contains do not hamper its Ca or Mg value.

The drying occurs at very high temperatures. This will extend shelf-life, inhibit mould growth during storage and so reduce the likelihood of adverse respiratory responses. Pelleting of lucerne leads to increased consumption and its relative bulkiness prevents a rapid accumulation of highly fermentable starch in the stomach, while allowing the penetration of it by gastric juices, ensuring a fall in gastric pH. Horses fed dried lucerne seem to produce better hoof horn than those receiving grass hay, possibly because lucerne provides considerably more S-containing amino acids and Ca than is provided by grass hay. The Persians were famed for their cavalry and when Darius' army invaded the Athenian Empire (490–480 BC) the army is said to have brought with it rich 'Median grass' (lucerne, possibly with seeds) to feed their horses. This 'blue grass' then became a food crop for Greek horses (Fox 2005). (Crops probably replaced areas containing clovers, so that the lucerne could become inoculated by the appropriate strain of *Rhizobium leguminosarum*, the author.)

Dried lucerne leaves contain several green pigments in addition to chlorophyll. One of these, pheophorbide-α, causes photosensitization, causing skin lesions in albino rats. Exposure to light in the visible range is sufficient to cause lesions in these rats and other white animals, given a lucerne-leaf-protein concentrate. The pheophorbide-α is formed by breakdown of chlorophyll under the influence of chlorophyllase, during processing. There is a higher activity of this enzyme in legume forages than in grass, which might account for an absence of dermatitis associated with green grass products.

Carrots (*Daucus carota*)

Carrot roots are very palatable to horses and contain no undesirable chemicals, as long as they have not been moulded. Horses not accustomed to their consumption can bolt them, developing choke, and in that case they should be sliced. They have a DM content of 110–130 g/kg and an energy value rather similar to that of oats per unit DM. The orange-coloured varieties are rich in β-carotene, containing 100–140 mg carotene/kg, 85% of which is present as the β-isomer. This is partially converted to vitamin A by the horse. Large intakes of carrots, and therefore of the provitamin, can cause intoxication in the human. β-carotene improves fertility in mares deprived of a dietary source (Ahlswede & Konermann 1980; Ferraro & Cote 1984; van der Holst *et al.* 1984) (see Chapter 4).

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Potatoes (*Solanum tuberosum*)

Small (chat) and damaged potatoes are sometimes fed to livestock. However, green potatoes and sprouted potatoes contain the alkaloid solanin, which is extremely hazardous to horses. Damaged potatoes and those starting to decompose are equally dangerous and there have been several reported fatalities. Small potatoes also cause choke. Waste potatoes are therefore not recommended for horses. Moreover, Meyer *et al.* (1995) reported that the pre-ileal digestibility of potatoes or cassava was less than 10% compared with 80–90% for oat starch.

Citrus pulp (*Citrus* spp. and *Ananassa sativa*)

Juice extraction from oranges, lemons, tangerines, limes and pineapples (*Ananassa sativa*) yields a residue of pulp (peel, pith and seed) that is dried and pelleted, frequently after adding limestone to neutralize the acid and to aid the removal of moisture (average calcium content then is 12 g/kg). The product is low in protein and P, rich in fermentable fibre (mostly pectins), clean and a useful addition to horse feeds, although the palatability is variable. The content of oil varies from 10 to 70 g/kg and the CF averages 130 g/kg. A high fibre level tends to indicate the presence of citrus seeds that contain relatively high concentrations of limonin, a metabolically interesting compound in human medicine that is toxic at high concentrations to pigs and poultry. The toxic threshold in horses has not been determined, but inclusion rates of up to 50 g pulp/kg in concentrates should be quite safe where seeds are present, and of up to 150 g/kg where they are scarce or absent. Ammoniated pulp is not recommended.

Olive pulp (*Olea europaea, O. sativa*)

The pulp and skin of olive fruits, following oil expression or extraction, is dried and pelleted. Where expression is used the pulp contains 260 g CF /kg and 100–180 g oil/kg which can be rancid and lead to increased vitamin E demands. For extraction, carbon disulphide, trichlorethylene or benzene is used and unsatisfactory solvent residue can be present. The product is equivalent to poor roughage. Pressed cake containing the seeds causes digestive problems, and the digestibility generally is only moderate. The Ca and P contents average 10 g and 2 g/kg, respectively; the CP, 100 g/kg, is of poor quality and low digestibility. Some batches have been of value for horses when included in the diet at up to 100 g/kg, but the variability of the product does not lend it to any general recommendation.

Cottonseed hulls (*Gossypium* spp.)

The hulls of cottonseeds, to which some fibres are attached, are free from gossypol and result from the decortication of the seed. The hulls yield a similar feed intake and growth rate to that of coastal Bermuda grass when each is used as the single roughage source in rations for young horses (Heusner *et al.* 2001).

Soya hulls (*Glycine max*)

Soya hulls are a consistent byproduct, rich in pectin and other soluble fibres (g/kg: 131CP, 606 NDF, 437 ADF). Booth et al. (2003)and Coverdale et al (2004) replaced up to 75% of alfalfa–brome grass hay by soya hulls without loss of acceptability. The consequential increase in dietary soluble fibre caused increases in total caecal VFA production and the proportion of propionate in the VFA, but a decrease in the proportion of butyrate and in caecal pH from 7.0 to 6.45. Total digestibility was unchanged, whereas N digestibility was decreased. Soya hulls are a very useful supplementary feed for horses.

Cassava (manioc) (*Manihot esculenta*)

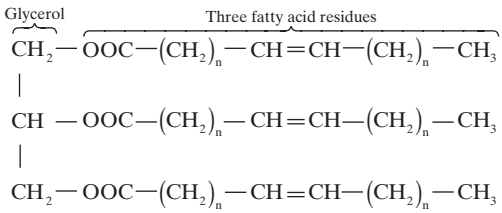
Cassava is a woody herbaceous shrub of tropical and subtropical areas. The tuberous roots provide a human and livestock food that is inferior to cereals and low in protein. The roots contain a glucoside, linamarin, which liberates hydrocyanic acid (HCN) by enzymic hydrolysis. A dried root meal, from which some of the starch has usually been extracted, it is widely available in many Western countries. It should contain a minimum of 620 g starch/kg, a maximum of 50 g CF/kg and 30 g silica/kg (mostly from adherent soil). It also contains a residue of the glycoside and HCN so that its value for horses is limited.

FAT SUPPLEMENTS

There is increasing interest in supplementing horse feeds with edible fats or oils (see Chapter 9). Where oils and fats are by-products of human food production, or of industrial processes, the quality of the fat or oil can be extremely variable. In order to establish the feeding value of a parcel, the fatty acid composition is frequently sought. This is generally inadequate and therefore it is opportune to give some description of fats and of analytical procedures used in their definition.

Crude fat consists of triacylglycerols (TAG, three fatty acid residues combined with glycerol, see Figure 5.2); long chain (aliphatic) alcohols, as found in waxes; choline; sterols (cyclic alcohols), such as cholesterol; moisture; and oxidized, polymerized and cyclized fatty acids. A typical feed fat (material soluble in a fat-solvent, e.g. ethanol,

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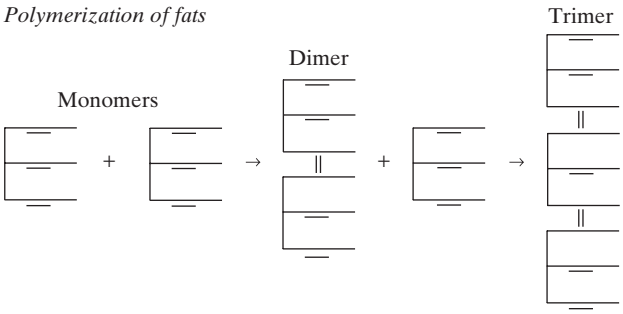


Figure 5.2 A triacylglycerol molecule (simplified to show one unsaturated bond in each fatty acid). Under severe heating of fats, commonly with unsaturated fatty acids present, these monomers combine by rearranging the linkages, forming new carbon–carbon double bonds, resulting in indigestible dimers and trimers.

Table 5.7 Composition of typical feed fat and desirable limits (g/kg).

	Typical	Desirable maximum
Moisture and other impurities	8	10
Unsaponifiables	20	30
Glycerol	25	
Total fatty acid	870	
Free fatty acid	60	60
Oxidized fatty acid	20	25
Oxidized, polymerized fatty acid	77	0
Polythene	—	0.2
Total	1080	

Table 5.8 Desirable GLC profile of fatty acids in fats used as supplements (g/kg fat).

Saturated fatty acids	300
stearic acid (C18:0)	100 maximum
palmitic acid (C16:0)	200
Monounsaturated fatty acids	500–600
palmitoleic acid, oleic acid (C16:1, C18:1)	500–600
Polyunsaturated fatty acids	250
linoleic acid, linolenic acid (C18:2, C18:3)	200 maximum*
arachidonic acid plus others, e.g. eicosapentaenoic acid (all in animal fats) (C20.4+)	50 maximum
Percentage total fatty acid content by internal marker	800 minimum

*See 'Polyunsaturated fatty acids', this chapter.

petroleum or diethyl ether) has the composition given in Table 5.7. Table 5.8 gives the characteristics of the gas-liquid chromatographic (GLC) fatty acid profile of fats suitable as horse feed supplements.

Only the glycerol and fatty acids have a reliable energy value. In order to determine the fatty acid composition of fat it is first hydrolysed by saponification, yielding the sodium or potassium salts of fatty acids and free glycerol. The unsaponifiables (insoluble in water, but soluble in fat-solvents) include hydrocarbons, aliphatic alcohols, cholesterol and phytosterols. These unsaponifiables, together with the high-molecular-weight polymerized and cyclized fatty acids, are not eluted from a column with the fatty acids during GLC analysis and so are not recorded in the standard fatty acid profile of the fat. The value for each fatty acid in this analysis is given as a percentage of the sum total of the fatty acids eluted and the values can therefore appear quite normal for fats containing damaged nonelutable fatty acids, as the total fatty acids will summate to 100%. (Note, this is not a per cent of the total fat supplement purchased.) The author has examined feed fats containing less than 400 g utilizable fatty acids/kg fat (<200 g/kg fat supplement, for most fats are sold on an inert base).

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If an internal marker of an unusual fatty acid, e.g. an odd-numbered carbon fatty acid, such as C17, is included in the analysis, an estimate can be made of the true proportion of the utilizable fatty acids in the total fat.

Polymerization and cyclization of fatty acids occurs during excessive heating of fat (thermal decomposition) (see Figure 5.2). Fat that is heated in air also undergoes oxidative decomposition, and the extent of this tends to be correlated with the extent of polymerization. An indication of the existence of oxidation can sometimes be obtained by measuring the peroxide value of the fat. This is a measure of the oxidation of the double bonds in unsaturated fatty acids and it is likely to be more extensive in polyunsatured oils unprotected by antioxidants. Values in excess of 20 mEq/kg oil must be suspicious. Low peroxide values, on the other hand, do not indicate a lack of damage, as peroxidation is a primary stage in the deterioration of fat quality. Secondary decomposition includes the formation of carbonyl-, hydroxy- and epoxy-derivatives, which are also valueless. The fat can become hydrolysed, liberating free fatty acids, hence the value of determining free fatty acids in raw material that is not intended to contain such acids. (Hydrolysed edible fat containing 50% free fatty acids is also marketed. This is a satisfactory product.) Saturated fatty acids can become rancid only following hydrolysis, as in butter, indicating deterioration by ketone formation.

The importance of using an internal marker in fatty acid analysis should be apparent. If such an analysis gives a value of 18% for, say, the C16 fatty acid as a percentage of eluted fatty acids in a product, which is sold as 50% fat on a nonfat base, then the actual amount of the C16 fatty acid present in the product is only 3.6%, where there are only 400 g utilizable fatty acids/kg in the product referred to above. Typical bases used to prepare fat premixes include palm kernel extractions, ground straw, wood flour and vermiculite, a hydrous silicate.

Damaged fatty acids in fats have a negative value, precipitating a vitamin E deficiency and can cause digestive upsets. Waste oils that are notorious for causing problems are acid oil distillation residues, especially those of fish acid. Other undesirable by-products are 'roller oil' from the steel and tin plating industry, oxidized cod oil, obtained from tanned hides, and recovered vegetable oil (RVO) from food frying outlets. Waste fats can be contaminated with heavy metals, pesticides or polythene. The rate of deterioration of a fat will be accelerated by contamination with, e.g., copper, and retarded by the presence of antioxidants (synthetic or natural). Most seed oils are highly unsaturated, but if fresh are generally rich sources of the antioxidant α-tocopherol (e.g., soya-bean oil). Marine oils will deteriorate more rapidly than, e.g., coconut oil, as a consequence of the difference in degree of unsaturation.

High-quality fats have a valuable role to play in the nutrition of stabled horses but it is imperative that the quality of the fat supplement is assured for this value to be realized. Edible oil reduces the rate of flow of ingesta from the stomach, as the addition of 10% maize oil dramatically decreases the glycaemic index of sweetfeed (Pagan *et al.* 1999a). Moreover, high-quality vegetable oil and animal fat do not adversely affect apparent digestibilities of Ca, P and Mg (Bowman *et al.* 1979, Rich 1980, McCann *et al.* 1987); but they can depress protein digestibility (Bush *et al.* 2001; Kronfeld *et al.* 2002). In general there is an increase in apparent fat digestibility and feed energy when fats are added to forage or grain rations. Fats ease pelleting of fibrous feedstuffs and reduce product dustiness.

NRC (2007) summarized published data concluding that apparent fat digestibilities (D_a) were 55, 81 and 95%, respectively, for forages, mixed feeds with added fat, and added fats. The poor apparent digestibility of forage fat is accounted for by dilution by endogenous fat (0.22 g/day per kg BW), encapsulation within cells, the poor digestibility of lipids that are of non-TAG origin, e.g., waxes, and a poorer true digestibility of lower concentrations of dietary fat. According to Kronfeld *et al.* (2004) the true digestibility of good quality added unsaturated fat approaches 100%, yielding a DE value of 38 MJ/kg. Saturated fats are less digestible, digestibility being inversely related to the melting point of fatty acids. These authors developed the following relationship for the digestibility (D, in %) and the fat content of the feed, (F, ether extract, in g/kg): $D = 92.0 - 92.0e^{(-F/342)}$; $r^2 = 0.81$, $P < 0.001$.

Fat digestibility, or efficiency of absorption, is a function of the rate of lipolysis, especially when residence time in the small intestine is limited.

With high inclusion rates, Jansen *et al.* (2000) reported that 150 g soya-bean oil/kg diet, substituted for isoenergetic amounts of corn starch, reduced the apparent digestibilities of CF, NDF and ADF by 8.0%, 6.2% and 8.3% respectively. Jansen *et al.* (2002) concluded that this excessive inclusion rate increases the amount of fat entering the ileum, caecum and colon, regions of significant microbial activity, which depresses fermentation (Zeyner 2002; Zeyner *et al.* 2002a; Jansen *et al.* 2000, 2001, 2002). The specific inhibitory effect of fat on microbial fermentation causes a depression in fibre digestibility (Table 5.9). Jansen *et al.* (2007) tested the hypothesis that the increase

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Table 5.9 The effect of crude fat and nitrogen-free extract composition of complete diets on apparent total intestinal tract digestibility of structural carbohydrate % of intake (Jansen *et al.* 2002).

	Soya oil	Corn starch	Glucose
Crude fat, g/kg DM	148	20	20
NFE, g/kg DM	493	668	646
Crude fibre digestibility, %	56.6	70.7	71.0
NDF digestibility, %	64.4	73.8	74.4
ADF digestibility, %	55.0	68.3	68.4
Cellulose digestibility, %	55.6	72.5	72.1

in bile acid secretion associated with fat supplementation and linoleic acid derived from soya oil, would depress caecal fermentation of fibre. There was no increase in faecal bile acid excretion with an iso-energetic substitution of soya oil for starch and glucose, and no reduction in fibre digestibility when soya oil was substituted for palm oil, with a six-fold increase in linoleic acid intake. In fact the infusion of linoleic acid into the caecum increased apparent fibre digestibility. No fat absorption occurs in the large intestine of horses (Swinney *et al.* 1995), so even if fibre fermentation is not depressed, fat digestibility would be depressed, imposing a practical ceiling on the inclusion rate. The threshold for this depression by dietary oil is approximately 110–150 g/kg DM in short-term tests, however, accommodation of the digestive system to the higher rates of oil consumption may take a month before fibre digestibility returns to pre-introduction levels (Zeyner 2002; Zeyner *et al.* 2002a). NRC (2007) concludes that there is an upper limit for soya oil inclusion of 0.7 g /kg BW daily. This is approximately 3% added oil in a diet that contains 3% naturally present lipid. NRC (2007) concludes that there is no consensus for the effects of dietary fat supplementation on muscle glycogen storage and that it has no influence on the rate of muscle glycogen utilisation (Pagan *et al.* 1987b; Essén-Gustavsson *et al.* 1991); but fat supplementation at 8% or more of total DM is associated with mitigation of exercise-associated decreases in plasma glucose and leads to increased TAG and NEFA concentrations during prolonged, low intensity, exercise (Hintz *et al.* 1978a; Hambleton *et al.* 1980; Hintz *et al.* 1982; Duren *et al.* 1987; see also Chapter 9). This is evidence of a glucose-sparing effect of fat and increased fat metabolism. This conclusion is supported by a lower rate of glucose utilization during aerobic exercise after 5 weeks adaptation to a diet providing 29% DE from corn oil (Pagan *et al.* 2002) and by a lower respiratory exchange ratio (RER), raised venous blood pH and lower pCO_2 during low to high intensity exercise 3 weeks after initiation of supplementation with 20% DE from soya oil (Dunnett *et al.* 2002) or corn oil (Kronfeld *et al.* 1998). These effects are not apparent with high intensity exercise. NRC (2007) concludes that complete adaptation to fat supplementation can take 2–3 months, but some metabolic responses are evident much earlier, and they are abolished within 5 weeks of withdrawal (Pagan *et al.* 1987b, 2002; Dunnett *et al.* 2002). Frank *et al.* (2004) concluded that, although plasma VLDL and TAG concentrations were elevated in thyroidectomized mares, their response to a high fat diet was similar to that of euthyroid mares, in which corn oil and refined, or crude, rice bran oil cf. water, lowered plasma NEFA and TAG (Frank *et al.* 2005).

The NRC (2007) explains the improved performance of horses adapted to fat supplementation by: (1) reduced weight of gut contents, (2) decreased metabolic heat production associated with feeding and exercise, (3) improved sprint performance, owing to increased energy transduction from anaerobic glycolysis and mitigation of acidaemia.

Polyunsaturated fatty acids

The constituent oils of most seeds and cereal grains are rich in *n*-6 polyunsaturated fatty acids. The sources include corn oil, sunflower seed oil, grape seed oil and even oat oil (Taylor *et al.* 2003). Linseed (flaxseed) oil is rich in the shorter chain *n*-3 fatty acid, α-linolenic acid (C18:3 *n*-3) and soya and rapeseed oils contain a smaller proportion of this fatty acid. These two series are differentiated by the position of the first double bond in the carbon chain, which, in the case of the *n*-6 series, is six carbons from the terminal methyl group. Cold water fish oils are rich in

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the higher (i.e. longer and more unsaturated chains) members of the *n*-3 group. To a limited extent the shorter chain acids are converted by the body into the longer chain acids by desaturase and elongation enzymes. Horses given flaxseed oil increased their plasma concentrations of both C18:3 *n*-3 and eicosapentaenoic acid (EPA; C20:5 *n*-3), but not of docosahexaenoic acid (DHA; C22:6 *n*-3) (Dunbar & Bauer 2002, Hansen *et al.* 2002). The plasma EPA and DHA of mares increased in a dose-responsive manner when given a fatty acid supplement containing EPA plus DHA at 0, 10, 20, or 40 g/day, reaching concentrations 13× and 10× those of controls for EPA and DHA, respectively, in the 40 g/day group by day 7 (King *et al.* 2008). Plasma EPA and DHA had declined by day 9 after cessation of supplementation to near pre-supplementation values. The supplement also increased plasma concentrations of C14:0, C17:1 *n*-7, C18:1 *trans*-11, C18:3 *n*-6, C18:4 *n*-3, C20:3 *n*-6, C20:4 *n*-6, and C22:5 *n*-3 and decreased concentrations of C18:1 *cis*-9.

Linoleic acid (C18 *n*-2), of the *n*-6 series in seed oils, is elongated and desaturated during metabolism to arachidonic acid (C20:4 *n*-6), which is the precursor of the inflammatory eicosanoid prostaglandins. Horses in hard work usually receive a large cereal-based diet, that is, one in which the fat is rich in *n*-6 polyunsaturates. On the other hand, the grazing and browsing horse receives oils derived from leaves which, by happy chance, contain a much higher proportion of the lower members of the *n*-3 series.

Anti-inflammatory effects of n-3 fatty acids

(see Chapter 9 for effects on exercise)

The dietary addition of *n*-3 fatty acids may improve exercise tolerance, decrease inflammation, and improve reproductive function. Competition for elongation, desaturation and cyclo-oxygenase metabolism occurs between *n*-3 and *n*-6 fatty acids as does antagonism of omega-3 derived eicosanoid mediators towards omega-6 derived mediators. So omega-3 enriched diets have promoted anti-inflammatory mediators for various inflammatory cell types (Henry *et al.*1991; Hansen *et al.* 2002; Hall *et al.* 2004a,b). Henry *et al.* (1990) gave horses a complete ration containing 8% raw linseed oil and found that the procoagulant activity and the thromboxane B_2 production by endotoxin-stimulated monocytes decreased by 51 and 71%, respectively, compared with cells from controls. This response was associated with a decrease in the *n*-6 : *n*-3 ratio of the membrane phospholipids of monocytes from treated horses. Similarly Morris *et al.* (1991) found that endotoxin-induced synthesis of TNF-α by macrophages was lower in horses given linseed oil. Comparable results were reported by McCann *et al.* (2000) with respect to the synthesis of the inflammatory mediators, thromboxane and TNF-α by monocytes following intravenous administration of a *n*-3 enriched lipid emulsion cf. a *n*-6 lipid emulsion. Nevertheless, the *in vivo* response of horses to endotoxin (0.03 mg/kg BW infused over 30 min) was not altered (Henry *et al.* 1990). Fish oil (cf. corn oil) increases neutrophil production of leukotriene B_5 (LTB_5) and reduces the quantity of prostaglandin E_2 by endotoxin-stimulated bronchoalveolar lavage (BAL) fluid in horses (Hall *et al.* 2004). Horses given large amounts of flaxseed (454 g/450 kg BW) experienced a lower allergic skin response to *Culicoides* extract (**sweet itch**), although flax oil failed to alter the clinical signs in horses with *Culicoides* hypersensitivity (Friberg & Logas 1999). Using linseed oil as a source of α-linolenic acid (108–110 g fatty acid/day per horse) these authors found no quantifiable effect on pruritic behaviour, or on the dermatologic lesion size caused by *Culicoides* spp. from oil source (linseed *vs* corn oils). The NRC (2007) concludes that, although measured changes in biochemical mediators of inflammation in response to fatty acid source (*n*-3 *vs n*-6) are persuasive, and diets enriched by oils containing omega-3 (*n*-3) fatty acids modulate the inflammatory mediator synthesis by cells harvested from blood, peritoneal fluid or respiratory secretions, the physiological significance of this in the treatment of inflammatory diseases, such as inflammatory airway obstruction (McCann & Carrick 1998) or fly-bite hypersensitivity, is unclear. In the human field there is evidence that cod liver oil reduces inflammatory reactions in joint disease. Techniques have been developed to retain an omega 6 : omega 3 ratio of 6 : 1 in soya oil, which, when added to the diet of strenuously exercised horses at the rate of 100 g/kg, cf. corn oil, reduced the inflammatory response, measured as the rise in blood fibrinogen, an acute-phase protein (Wilson *et al.* 2003a). Although working horses are subject to painful inflammatory leg ailments, the use of *n*-3 fatty acids in their control is not yet proven. Moreover, fish oil *n*-3 fatty acids are more unsaturated than oils of the *n*-6 series and any increase in use of *n*-3 fatty acids should be accompanied by an increase in vitamin E supplementation.

Fat Supplements and Behaviour

(see also L-tryptophan, this Chapter)

Fat supplementation appears to reduce excitability, nervousness, and resting heart rate (Holland *et al.* 1996a; MacLeay *et al.* 2000; McKenzie *et al.* 2003a) and to reduce the exercise-associated increase in plasma cortisol concentrations (Crandell *et al.* 1999). Amongst growing

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foals a fat and fibre supplement cf. a starch-based concentrate lowered plasma cortisol (Holland *et al.* 1996b), reduced apparent distress at weaning and decreased nervousness under test (Nicol *et al.* 2005), although it is unclear whether or not lecithin, a component of the fat, influenced the behaviour. (Choline, present in lecithin, is a component of the neurotransmitter acetylcholine found in sympathetic and parasympathetic ganglia and at the myoneural junction.)

PROTEIN CONCENTRATES

Vegetable proteins

The richest sources of vegetable protein fed to horses and ponies are oil-seed residues, but other sources include peas, beans, yeast and, high-quality dried forages, particularly lucerne meal (alfalfa). Processed soya beans, linseed, cottonseed and, to some extent, sunflower seed, are widely used. Groundnuts are not recommended because of their frequent contamination with aflatoxin, from the mould *Aspergillus flavus*, also found in some other feedstuffs, to which the horse is relatively sensitive.

Two alternative procedures are adopted for the extraction of oil from oil seeds, both of which are preceded by the removal of a thick coat by a process known as decortication, as practised for cottonseed and sunflower seed (undecorticated meals contain very much more fibre). Where the oil is extracted by pressure (expeller process), this is preceded by cooking at up to 104°C for 15–20 min, after which the temperature is raised briefly to 110–115°C. Pressure is then applied by passing the seeds through a horizontal perforated cylinder, in which a screw revolves and the oil is partially pressed out, leaving a residue containing around 35 g fat/kg. This residue has the advantage of incorporating more fat than meals derived from the more efficient chemical-extraction process. The temperatures achieved during the expeller process can damage the protein, however, which generally has a lower **biological value** (BV) than that resulting from solvent extraction. In addition, only material with less than 350 g oil/kg is suitable for pressure extraction. In chemical extraction, the feeds are first subjected to a modified, less extreme, screw press. The seeds are then flaked and the solvent, usually hexane, is allowed to percolate through removing the oil. Solvent residues are evaporated by heating or toasting, which also benefits some meals by destroying natural toxins.

Oil-seed meals are much richer sources of protein than cereals and their balance of amino acids is superior. However, linseed meal is a poorer source of lysine than soya, which is considered to produce the best quality proteins. Sunflower seeds are rich in cystine and methionine, although it is rare for horse diets to be limiting in respect of these amino acids. Oil-seed meals are also relatively reliable sources of some of the B vitamins and of phosphorus, but contain little calcium.

Soya-bean meal (Glycine max)

Raw soya beans contain allergenic, goitrogenic and anticoagulant factors in addition to protease inhibitors. The correct toasting and cooking of the beans (micronization and well-regulated oil-extraction procedures) destroys these factors without detracting from the protein quality. Reliably cooked products are used as the sole source of a supplementary protein in horse feeding.

Standard hexane-extracted soya-bean meal contains 440 g CP/kg. Dehulled meals of uniformly high quality containing 480–490 g CP are also commercially available. Both these meals contain less than 10 g oil/kg. Full-fat soya flour and cooked soya flakes are much more costly, but the latter is widely used in coarse mixes and both contain 180–190 g fat and 360–400 g of CP/kg. The precise composition varies with the CF level, which ranges from 15 to 55 g/kg.

Linseeds and linseed meal (Linum usitatissimum)

Linseeds are unique in so far as they contain a relatively indigestible mucilage at concentrations of between 30 and 100 g/kg. This can absorb large amounts of water, producing a thick soup during the traditional cooking of linseed, and its lubricating action regulates faecal excretion and sometimes overcomes constipation without causing looseness. The cooking also destroys the enzyme linase, which, after soaking, would otherwise release HCN from a glycoside in the seeds, in the presence of water, so poisoning the horse. This implies that the seeds should be added to boiling water rather than to cold water and then boiled. However, HCN is volatile and a proportion of any already present will be driven off by subsequent boiling. Cooked linseed mashes are highly regarded to improve coat condition of horses for sale. Linseeds should not be fed dry because of their water-absorbing propensity, although the linase would be inactivated by gastric secretions.

The low-temperature removal of oil during the production of linseed meal implies that the product is toxic if fed as a gruel, when linase has not been inactivated and HCN is evolved. In comparison, oil removal by the expeller process normally yields sufficient heat to make a safe cake, whether it be fed wet or dry. UK laws stipulate that linseed cake or meal must contain less than 350 mg

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HCN/kg, although this takes no account of any linase activity present.

Coconut meals (copra) (Cocos nucifera)

The dried flesh of the coconut contains variable amounts of oil (50–70 g/kg) depending on the efficiency of extraction. The oil is composed of highly saturated, medium-chain fatty acids and so is less subject to rancid fat development than many vegetable oils. The meal is prone to absorb moisture and mould unless lengthy storage is avoided and an environment of low humidity achieved. It contains a similar amount of protein to that of peas, but of lower BV. The meal typically contains 220 g CP, only 6 g lysine and 125 g CF/kg. It is palatable and contains no toxic factors.

Cottonseed meal (Gossypium *spp.*)

Cottonseed meal is a cake by-product of cotton and oil production and it tends to be dry and dusty, having a somewhat costive (constipating) action. Undecorticated, the cake contains 200–250 g CF/kg. If the hulls are separated from the kernels the fibre content is halved. The oil is removed either by hydraulic expulsion or solvent extraction methods. Glands within the seed embryo contain a toxic yellow pigment, gossypol, which is at a concentration of between 0.3 and 20 g/kg DM in raw cottonseeds. Heating during processing partly inactivates the toxin in the raw material, but at excessive temperatures gossypol binds lysine, depressing protein quality, yet partially inactivating the toxin. Even the bound form is reported to reduce intestinal Fe absorption; this is partly counteracted by further Fe supplementation. Free gossypol can be at a concentration of 1 g/kg in expeller meals and 5 g/kg in solvent-extracted meals. Mixed feeds containing more than 60 mg of free gossypol/kg are unsatisfactory for horses. Only products of glandless varieties are suitable for horse feeding and these are scarce in Europe. Cyclopropene fatty acids are also present in cotton seeds, but at lower concentrations in glandless varieties, although the amount is proportional to the oil residue in the meal. Thus, extracted meals of glandless varieties should be sought, notwithstanding the limitation placed by Feeding Stuffs Regulations on the gossypol content of meals sold in the UK to 1.2 g/kg. Good quality cottonseed meals are palatable and can contain 400 g CP and 15 g lysine/kg.

Gibbs *et al.* (1996) reported that the precaecal N digestibility of solvent-extracted cottonseed meal amounted to 81.2% in comparison with only 57.1% for their sample of soya-bean meal. Although the N digestibilities over the entire GI tract were similar for both, the amino acid value of dietary proteins depends on precaecal digestion (see Chapter 1).

Sunflower-seed meal (Helianthus annuus)

Sunflowers are grown for the oil in their seeds. The oil of the meal residue is unstable and present in greater amounts in expeller than chemically extracted meals. The fibre is poorly utilized and the fibre content of the meal is much lower where the seeds have been partially dehulled (decorticated) and so it ranges from 50 g to as much as 350 g/kg. The protein is rich in sulphur-containing amino acids (double that of soya protein), but variable in amount (from 240 g protein/kg in expeller cake to 500 g/kg in some samples of dehulled extracted meal). The methionine and lysine contents of the meal are 5–14 g and 9–22 g/kg, respectively, and the meal is a good protein source for horses, containing no significant undesirable substances, but it is unpalatable in large quantities.

Palm-kernel meal (Elaeis guineensis)

Expeller and extracted meals are available, but they are not widely used for horse feeding owing to low palatability. The protein and CF contents are typically both 160 g/kg; the protein is relatively impoverished of lysine (40 g/kg protein). Nevertheless, the meals are free from undesirable chemicals.

Groundnut meal (peanut meal) (Arachis hypogaea)

Groundnut or peanut meal is produced following oil removal by expeller and extraction processes. The former process leaves a larger residue of polyunsaturated oil. The protein content of the meal is slightly higher than that of standard 44% soya-bean meal and averages 470–480 g/kg. Groundnut meal is very palatable and contains no undesirable substances apart from contamination with the hepatotoxin, aflatoxin, derived from the mould *Aspergillus flavus*. The horse is subject to severe aflatoxicosis, and, owing to a history of serious damage among livestock, the use of this by-product in Europe has been severely curtailed by EU legislation, otherwise groundnut meal could make a useful contribution to equine nutrition.

Rapeseed meal (Brassica napus, B. campestris)

Rape is a member of the genus *Brassica* in the family Cruciferae and it is grown for the oil in its seeds. There is a large world production of rapeseed, and varieties from two species are grown – *Brassica napus* and *B. campestris.* Western European production has increased rapidly owing to encouragement by the EU. A drawback to rapeseed in the past was the content of erucic acid in the residual oil

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and of glucosinolates in the meal. Glucosinolates, present in the unheated rapeseed, and widely distributed among the Cruciferae, are goitrogenic when hydrolysed during digestion by the enzyme myrosinase, present both in unheated rapeseed and in gut microorganisms. The active goitrogens released are isothiocyanates and oxazolidinethiones (goitrin) (see Goitrogens, this chapter). Although the protein quality of rapeseed meal is good and heat treatment decreases the hazard by destroying the myrosinase, the intestinal enzyme can still release quantities of the thyroactive substances and so only small amounts of meal of unknown origin are suitable for feeding to horses.

This problem led Canadian plant breeders to select varieties of *B. campestris* low in both erucic acid and glucosinolates (less than 3 g/kg seed), known as 'double-low' varieties and sold as canola meal. Comparable varieties are widely available in Europe, leading to their routine cultivation. The better varieties are a good source of protein, but use should be restricted to 200 g/kg concentrates. The protein content of the meal ranges from 340 to 390 g/kg and the protein contains 60–64 g lysine/kg; the meal therefore replaces soya on an equivalent protein basis. Several rapeseed varieties contain tannins, or polyphenols (averaging 30 g/kg seed) that reduce digestibility, contributing to a slightly lower protein value for the meal in comparison with that of soya (reviewed by Aherne & Kennelly 1983). A further discussion of these toxins is given later in this chapter (see Condensed tannins).

Lupin-seed meal (Lupinus albus, L. angustifolius, L. luteus)

Lupin-seed meal has disadvantages for horse feeding. There are a number of varieties within each of three species that have white, blue and yellow flowers. These are variably sweet or bitter. The bitter varieties contain significant amounts of toxic alkaloids and the ether extract content is variable (40–120 g/kg) and prone to rancidity. The CF content is high and variable (80–160 g/kg), but relatively well utilized by the horse, and the protein and lysine contents vary from 250 to 400 g/kg and from 14 to 23 g/kg, respectively. The seeds are small and must be ground, or crushed, for use. Only the sweet varieties should be used and limited to 50 g/kg concentrate.

Sesame-seed meal (Sesamum indicum, S. orientale)

The seed oil of sesame is a source of unstable polyunsaturated fatty acids and so only extracted, not expeller, meal should be used. This contains 40–55 g CP/kg, and is rich in methionine but deficient in lysine. The meal is also rich in P, mainly present as phytates. The husks contain oxalates so that only decorticated meal is suitable.

Peas (Pisum sativum, P. arvense), *white- and purple-flowered*

Peas contain less protein than do field beans, but the BV to horses is equivalent to that of soya protein. Legume seeds generally contain antinutritive factors, although the content in modern pea varieties is low. Some older varieties contain trypsin inhibitor and phytohaemagglutinins (lectins), which are partially inactivated by heating. Trypsin inhibitors are stable up to 80°C and soaking for 18 hours removes 65% of the haemagglutinin activity. However, autoclaving for 5 min at 121°C completely destroys pea trypsin inhibitors and haemagglutinins. The trypsin inhibitor content of peas is similar to that in field beans, but only 10% of that in soya-beans. Peas also contain chymotrypsin and amylase inhibitors, although the content of the latter is very low and destruction by heating is similar to that for trypsin inhibitor.

The oxalate content of peas is about 7 g/kg, phytic acid 5–8 g/kg and tannins (polyphenolic compounds) 0.2–0.4 g/kg. Tannins, mostly in the testa (seed coat), bind to enzymes and other proteins forming insoluble complexes and reducing digestibility. Extended cooking is a good insurance (see 'Natural and contaminant toxicants in feeds', this chapter). The extrusion and micronisation processes adopted for peas included in coarse mixtures and other compounded feeds are adequate to achieve a significant improvement in nutritive value. Peas are a very useful protein source.

Field beans (Vicia faba), *winter beans (horse) and spring beans (tick and horse)*

Within the bean family (Leguminosae or Fabaceae) two genera, *Vicia* and *Phaseolus*, grow throughout the world, and many are important food crops. Winter and spring varieties of field (horse) beans grown in the UK are members of the species *Vicia faba*, all safe for feeding to horses, especially after cooking. The winter field (horse) bean contains on average 230 g CP and 78 g CF/kg, whereas the spring bean contains 270 g CP and 68 g CF/kg. Amounts of lipid in both varieties are low – about 13 g/kg; like most other seeds they are rich in P but poor in Ca and Mn. Field-bean protein is of high quality as it is a valuable source of lysine. The bean is normally cracked, kibbled or coarsely ground, but is given whole to adult horses with sound teeth. The water-soluble polyphenolic condensed tannins in the hulls interact with both dietary and endogenous proteins in the intestines, increasing their

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faecal loss. High dietary concentrations of condensed tannins are said to depress appetite.

Phaseolus *and some other legume species*

There are importations of beans belonging to the genus *Phaseolus*, especially the lima (navy) bean (*P. lunatus*) and the kidney (haricot, pinto and yelloweye) bean, (*P. vulgaris*). Kidney beans are normally refused by horses unless cooked; if force-fed they will cause colic. These beans must all be cooked (wet heat) before feeding because they contain several toxic factors, including antiproteases and lectins, which will cause diarrhoea, and many beans of the genus *Phaseolus* also contain a cyanogenetic glycoside identical to that in linseed. Other beans available in parts of the world include the hyacinth bean or lablab (*Dolichos lablab*), horse gram (*D. biflorus*), green and black grams (*P. aureus, P. mungo*), chick pea (*Cicer arietinum*), which is widely used in Asia, and lentils (*Lens* spp.), of which India is the chief producer. The Indian or grass pea (*Lathyrus sativus*), at one time imported as an animal feed, and some other members of the same genus, cause lathyrism (see 'Lathyrism caused by β(γ-L-glutamyl) aminopropionitrile and L-α, γ-diamino butyric acid', this chapter). The horse is particularly, and characteristically, affected. If beans and peas of unknown origin are used to feed horses, extended cooking will provide a measure of safety from many of the toxins.

Lentils, split peas and red dahl (Lens esculenta, L. culinaris)

Lentils, split peas and red dahl are important and palatable legume crops, producing seeds containing protein of good quality (protein 260 g/kg, lysine 70 g/kg protein), for human consumption. They contain small amounts of trypsin inhibitors and haemagglutinins and, as they are in demand for human use, any batches available as livestock feed may be contaminated with moulds and mycotoxins. Lentil bran is obtained as a by-product of lentil preparation for human consumption.

Animal proteins

There are only two high-quality animal protein sources suitable as horse feeds – white fish-meal and milk-protein products. They are reserved almost entirely for foals, either in creep supplements or as milk replacers. Small amounts are occasionally given to adult horses in poor condition, but large amounts of dried skimmed milk cause diarrhoea, owing to the presence of lactose (see Chapter 1).

Fish-meals

Two types of fish-meal are recognized under British law, of which the first is a product from the drying and grinding of fish, or fish waste, of a variety of species. The second, marketed as white fish-meal, is a product containing not more than 4% salt and obtained by drying and grinding white fish or the waste of white fish to which no other matter has been added. This is a high-quality protein source containing abundant lysine, and it is suitable for, but not essential in, the diet of young foals. It is rich in minerals (about 80 g Ca and 35 g P/kg), trace elements (especially Mn, Fe and I) and several water-soluble vitamins, including vitamin B_{12}. This vitamin is found naturally only in animal products and bacteria. The dietary requirement of the weaned foal for the vitamin can be met by the inclusion of fish-meal, or synthetic sources, in its diet. The suckling foal should, however, receive ample in the dam's milk. About 5 or 10% of white fish-meal in a creep feed, or milk replacer, is quite satisfactory.

During processing the fish waste is dried by one of two procedures. The more desirable one is steam drying, either under reduced pressure or with no vacuum applied. The other procedure is based on flame drying, when the temperatures achieved decreases the digestibility of the protein and decreases lysine availability. Fish-meals of unknown origin can be contaminated with pathogenic organisms, in particular with *Salmonella* species or other enteric organisms that cause diarrhoea. Good quality white fish-meal, however, should be a safe feed. Meat meals, meat and bone meals and unsterilized bone flour should on no account be used for feeding to horses because many samples are contaminated with *Salmonella* and other transmissible pathogens, including bovine spongiform encephalopathy (BSE), or are shipped in contaminated bags. The problem of ridding stock of infection, once contracted, is considerable.

Cow's milk

Where liquid milk is used for feeding to orphan foals, it should be diluted with 15–20% of clean water and given in small amounts in as many meals as is practically convenient. Liquid cow's milk, on average, contains 125 g DM, 37 g fat, 33 g protein and 47 g lactose/kg. It contains little Mg and is deficient in Fe, a source of which should be provided for the young foal (at birth the foal's liver acts as a reservoir of Fe). Whole milk is rich in vitamin A and provides useful quantities of vitamin B_{12}, thiamin and riboflavin. Milk proteins contain abundant lysine.

Dried skimmed milk is widely available and sold commercially as a component of milk replacers and horse

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supplements. It contains very little fat and therefore practically none of the fat-soluble vitamins. However, the protein quality approaches that of the liquid product if drying has been carried out by the spray process. Roller drying subjects milk to higher temperatures which results in some loss of lysine availability, and in large quantities this product can cause diarrhoea. Any significant quantity of dried skimmed milk should not be fed to horses more than three years old, owing to their deficiency in the enzyme lactase (β-galactosidase).

Spray-dried skimmed milk is a useful supplement to feeds of young foals where the mother is providing inadequate quantities of milk to sustain normal growth. Concentrations of 10–15% in the dry diet have proved satisfactory; but its use in creep feed for foals approaching weaning is unnecessary if the objective is to encourage the development of a faculty for the digestion of post-weaning horse feeds.. A stud mix is a satisfactory creep feed.

Nucleotides

Nucleosides and nucleotides are structural units of ribonucleic acid (RNA) and deoxyribonucleic acid (DNA), which function in the maintenance and stimulation of bone marrow hematopoiesis and maintenance of intestinal mucosa where there is limited capacity for de novo synthesis of purine and pyrimidine bases. Supplementation of laboratory animals with these sources has been shown to stimulate haemopoiesis, to increase peripheral neutrophil counts, survival against methicillin-resistant Staphylococcus aureus (MRSA), and delayed-type cutaneous hypersensitivity, to reduce endotoxin-induced bacterial translocation and to improve injury to the gut in protein-deficiency.

Single cell cultures of yeast and bacteria as sources of protein

Yeasts (Saccharomyces cerevisiae, S. carlsbergensis)

Yeasts contain protein of good amino-acid balance. However, a greater proportion of the N in single-cell organisms (bacteria in particular and yeasts) compared with plant cells is composed of nucleic acids (50–120 g/kg DM in yeasts and 80–160 g/kg DM in bacteria). While some of the purine and pyrimidine bases in these acids can be used for nucleic acid biosynthesis, large amounts of uric acid, the end product of nucleic acid catabolism, are excreted in the urine (however, see Nucleotides, above). The CP and fat contents of yeasts range, respectively, from 400 to 450 g/kg and 25 to 55 g/kg. Yeasts are readily digested and are a rich source of B-group vitamins with the exception of vitamin B_{12}. This vitamin is synthesized almost exclusively by bacteria. Dietary inclusion rate of yeast up to 50–75 g/kg feed is economically feasible only for foals.

Bacteria

The CP and fat contents of bacterial cultures range, respectively, from 340–720 g/kg and 20–210 g/kg, but their use as a dietary protein source is not justified.

PRE- AND PRO-BIOTICS

Prebiotics

Substances added to the diet that preferentially stimulate the growth of beneficial microorganisms in the hind-gut have been termed *prebiotics*. They are a diverse group of complex polysaccharides containing sugar moieties resistant to hydrolysis by mammalian digestive enzymes, but which are fermentable by colonic bacteria. They include the rapidly fermented fibres, fructo-, galacto- and mannan-oligosaccharides (see also 'Laminitis control', Chapter 11). Fructo-oligosaccharides (FOS) are naturally occurring short to medium chains of fructose residues linked by β2–1 glycosidic bonds. They promote the growth of *Bifidobacteria* that are capable of hydrolysing these β-glycosidic bonds. Production of VFAs by benign bacteria is considered to lower the intestinal pH to the disadvantage of pathogens. Short-chain FOS given at rates of: 0, 8 and 24 g/day per horse (cf. amount consumed from pasture causing laminitis) caused a linear decrease in the pH of wet faeces and linear increases in its acetate, propionate, butyrate and lactate concentrations (Berg *et al.* 2005). Respondek *et al.* (2008) assessed the effectiveness of short-chain FOS in reducing hindgut microbial disturbances related to an abrupt incorporation of barley in the diet. Without the addition of short-chain FOS there was a substantial increase in colonic total anaerobes, lactobacilli, streptococci, and lactate-utilizing bacteria, whereas with the addition the *Lactobacillus* and *Streptococcus* populations did not increase cf. controls. Although the colonic D-lactate concentration increased after the meal with barley controls, it did not accumulate with short-chain FOS supplementation. Oligosaccharides of the sugar residue, mannose, are believed to reduce the adherence of pathogenic bacteria to enterocytes, by reaction with mannose-specific bacterial lectins (NRC 2007) and these oligosaccharides might prevent the absorption of mycotoxins in horses (Raymond *et al.* 2003, 2005). When 10 g/day of mannan-oligosaccharide was given to pregnant mares from 56 days before foaling, there was a marginal increase in colostrum IgG, IgM and IgA concentrations (Spearman *et al.* 2003). In accord with this, foals maintained on

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pasture and given a supplementary diet high in fat and fibre, cf. high sugar and starch, both before and after weaning, produced higher plasma IgA, IgG and α-tocopherol concentrations, a higher faecal pH and lower faecal butyric and valeric acid concentrations (Swanson *et al.* 2003). This observation is surprising as pasture grass is variably rich in fructo-oligosaccharides and, according to Benage *et al.* (2005), mannan oligosaccharides do not stimulate immune function, as measured by white cell counts or antibody titres in young, mature or aged horses.

Probiotics

Over the past two decades there has been increasing interest in the inclusion of probiotics in equine diets. Probiotics, or substances 'for life', are live microorganisms and their products that, when administered in adequate numbers, confer a benefit to the health of the host, principally through their influence on the microbial flora of the GI tract. Under EU legislation only registered live culture strains may be used for feeding to animals. The microorganism strain must be identified according to a recognized international code which includes: the deposit number of the strain; the number of cfus (expressed as cfu/kg, measured by an acceptable method); the period during which the cfus will remain present; and characteristics of microorganism that have arisen during manufacture. The organisms predominately used are certain species of Gram-positive lactic-acid-producing bacteria. In the UK, the Medicines Act (1986) will apply if medicinal claims are made.

Fungi

These are commonly strains of *Saccharomyces cerevisiae* (baker's and brewer's yeast), *Aspergillus oryzae* and *Torulopsis* spp. Studies by Glade & Sist (1990) showed that *A. oryzae* added to *in vitro* equine caecal cultures, did not alter fermentation products or pH. The consumption of live cultures of *S. cerevisiae* (Collection No. NCYC 1026) at a rate of 10–20 g/day per horse (10^9 CFU/g), stimulates hind-gut microbial growth, and improves dietary fibre fermentation and the apparent digestibility of pectins, hemicellulose, cellulose and CP, especially in young growing horses. Similar dosage rates for adult horses increase apparent digestion of NDF (Medina *et al.* 2001), CP and Mg (Switzer *et al.* 2003), and Ca and P, potentially through increased hind-gut phytase activity (Hill & Gutsell 1998). Jouany *et al.* (2009) gave horses 10 g of *S. cerevisiae* (CBS 493.94) containing 4.5×10^9 cfu/g and showed that the activity of most enzymes involved in plant cell wall digestion were increased. At these rates endogenous (metabolic) faecal N loss is reduced. N-retention and plasma concentrations of arginine, glutamine, glycine, isoleucine, leucine, methionine and valine in foals are increased, while concentrations of ammonia, hydroxyproline and 3-methyl-histidine are decreased (Glade & Biesik 1986; Glade & Sist 1990). Live *S. cerevisiae* culture (10 g/day) produced a viable yeast count of 4.4×10^6 cfu/mL in the caecum and of 5.6×10^4 cfu/mL in right-ventral colon with both high-starch and high-fibre diets (Jouany *et al.* 2009); with a starch overload hind-gut luminal pH, lactic acid and ammonia concentrations and molar percentages of acetate and butyrate were modified, so that the undesirable changes wrought by the overload were limited (Medina *et al.* 2002). Yet such changes have an unclear interpretation. Similar additions have decreased the caecal acetate content resulting from both a high-fibre and a high-starch diet, but increased the caecal lactic acid content only with the former (Krusic *et al.* 2001). Jouany *et al.* (2008) gave a live yeast (*S. cerevisiae*) preparation providing 4.5×10^{10} cfu/day per adult horse receiving a dehydrated alfalfa diet or a high-starch diet. The supplementation to both diets improved ADF digestibility and stimulated DM and NDF intakes, without influencing their mean retention time. Morgan *et al.* (2007) offered Russell Bermuda grass hay of either high (13.1% CP, 73.1% NDF and 35.3% ADF) or low (8.1% CP, 75.3% NDF and 37.6% ADF) quality to adult geldings at the rate of 1.35% of BW plus a grain mix (12.6% CP, 25.4% NDF and 12.1% ADF) at 0.45% of BW daily to which 56 g yeast culture/horse was added in the morning feed. The yeast had no effect on the high-quality hay, but it increased digestibilities of DM, CP, and NDF in the low-quality forage. Yocum & Alston-Mills (2002) found that either *Kluyveromyces marxianus* or *S. cerevisiae* given daily from 14 days before foaling until day 42 of lactation reduced the lactose content of the milk.

One question is whether killed cultures of fungi, or bacteria, retain some useful attributes of live cultures. Evidence from ruminant research shows that *S. cerevisiae* (Collection No. NCYC 1026) protects anaerobic rumen bacteria by increasing the rate of oxygen disappearance, indicating that viable cultures are necessary for this respiratory activity. Under EU legislation only registered culture strains may be used for live feeding to animals.

Bacteria

The bacterial species employed as probiotics are Gram-positive organisms which resist lysozyme. They ferment glucose and a variable number of other sugars with the formation of organic acids, principally lactic and acetic

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acids, and therefore withstand a relatively low ambient pH. The species are generally in the genera *Lactobacillus* and *Streptococcus*, although related species, including *Pediococcus* are used. The lactobacilli are probably all benign, whereas many *Streptococcus* spp. are parasitic in man and animals and some are highly pathogenic. Cultures must be pure, but a case for their use in horse feeding is as yet unproven. Attempts to achieve colonization of some *Lactobacillus* cultures have been unsuccessful. An equine-specific *L. pentosus* WE7, identified as having good inhibitory activity against enteric pathogens, will colonize the horse; but this organism induced clinical disease and diarrhoea when used as a probiotic in neonatal foals (Weese et al. 2004; Weese & Rouseau 2005). A culture of *L. acidophilus* did not increase the degradation rate of lactate in the caecum (Booth *et al.* 2001) (N.B. *L. acidophilus* ferments several sugars to form DL-lactic acid) and so it is questionable whether the use of this and related bacteria is justified for horses. Nevertheless, Ward *et al.* (2004) observed a marked reduction in *Salmonella* shedding by hospitalized horses without GI disease given a probiotic agent cf. controls.

The viability of Lactobacillus species varies enormously among strains. Cultures should be preserved first by freeze-drying, the preferred method, in which water is removed by sublimation. With the object of extending shelf-life, manufacturers frequently encapsulate organisms so that they withstand aggressive environments more readily. The strain type selected for the horse should have been demonstrated to survive and multiply in the equine GI tract. This assumes that viability is essential and, at this point, it is appropriate to remind ourselves of the characteristics that a useful culture should possess. It should:

- be incapable of causing disease or raising caecal lactic acid concentration;
- contain 10^8–10^9 viable organisms/g after 12 months' storage;
- have the correct host specificity, contain useful enzymes and certain antibacterial substances, and, if necessary, be capable of displacing lactic acid bacteria occurring naturally in the intestine;
- be Gram-positive and so resistant to gastric juice, i.e. both its low pH and its enzymes. Lactobacilli are generally resistant to the acidity encountered in the stomach, although the degree of resistance varies from strain to strain. They should be resistant to lysozyme. Gram-positive lactic-acid bacteria have a greater resistance to lysis by this enzyme than Gram-negative coliforms. and they are more resistant to the adverse effects of freezing and freeze-drying;
- be bile tolerant and tested on *Lactobacillus* selection (LBS) agar containing 0.15% oxgall. (Bile salts lower surface tension and probably emulsify the lipids in the bacterial cell membrane destroying them.)

Most strains of *L. bulgaricus* and *L. lactis* possess poor bile tolerance, despite their frequent inclusion in probiotic cultures. *Lactobacillus plantarum* and *Bifidobacterium bifidum* are two species frequently used, but most strains fail to meet the above criteria adequately.

The possible functions of probiotics are:

- to act as a source of useful enzymes in the GI tract. Yogurt can increase lactose digestion in humans, even though the bacteria might not survive and/or grow in the intestines. Lactobacilli contain an intracellular β-galactosidase, which is thus protected during its passage through the stomach, but bile salts alter the membrane permeability of the bacterial cell, allowing ingress of lactose and access to the enzyme. If the bacterium grows in the intestine it will produce large additional amounts of the enzyme;
- to inhibit the growth of intestinal pathogens. Many lactic acid bacteria have the ability to produce bacteriocins, antibiotic-like compounds that are active against closely related species. Enteric pathogens require their attachment to intestinal epithelial cells for pathogenicity. Precolonization and attachment to those cells by probiotic bacteria reduces this pathogenic potential; and
- to assimilate cholesterol.

How these possible functions can lead to increased performance of the horse is unknown. The objective selection of fungal and bacterial species and strains will depend on our knowledge of how probiotics work.

Enzymes (bacterial and fungal)

A Directive from the EU (Council Directives 93/113/EC 93/114/EC) deals with the roles of supplementary enzymes and microbial cultures and lists of all permitted and properly identified products for use within the EU including products for use in feed, in drinking water and those given as a drench. The recommendations of the Nomenclature Committee of the International Union of Biochemistry and Molecular Biology are applied to enzyme taxonomic identification. Enzymes are proteins and can therefore be denatured. Those contained within microbial cells are protected from destruction in storage, or, in the case of those that

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function at a neutral or alkaline pH, from destruction by the acid environment of the stomach. As enzymes possess pH optima for activity, it is essential that the optimum for a particular enzyme coincides with that in the gut location of its anticipated activity. Morris-Stoker *et al.* (2001) detected no effect of phytase supplementation on mineral availability when it was given to mature geldings, whereas response in the young of several species has been reported. However, van Doorn *et al.* (2004) found increased phytate-P digestibility in horses, without a change in total P apparent digestibility. Further evidence is required on this in diets of different types. Fibrolytic enzymes added to low-quality grass forage-based diets have failed to improve digestibility in horses. No improvement of the digestibility of timothy hay and of its NDF and ADF occurred in mature Arab geldings from the addition of cellulase providing 120 000 units/day (O'Connor *et al.* 2005; O'Connor-Robison *et al.* 2007).

Under EU legislation and in Schedule 4 of the UK Feeding Stuffs Regulations 2000, enzymes may be used that are not excluded from application of the Additives Directive by Article 22 of that Directive. Note: the addition of phytase to a P-deficient diet for turkey poults improves their bone characteristics. The Statutory Statement on animal feeding stuff product containers includes the following information:

- the names of the active constituents according to their enzymatic activities;
- the International Union of Biochemistry identification number, i.e. EC number;
- the activity units (expressed as activity units/kg or /L), if these can be measured by an acceptable method;
- an indication of the period during which the activity units will remain present;
- an indication of any significant characteristics of the enzyme arising during manufacture, as specified in the authorization concerned; and
- the EU registration number.

For a discussion of joint supplements, see: Glucosamine, methyl sulphonyl methane (MSM) and chondroitin sulphate supplementation, Chapter 8.

DIETARY VITAMIN AND MINERAL SUPPLEMENTS

A mixture of cereals, a concentrate protein source such as field beans or soya meal, and hay is adequate in terms of energy and amino acids to meet daily needs, but it is likely to be inadequate in certain minerals, trace elements and vitamins for optimum performance, particularly in the longer term. Some raw materials are relatively rich sources, especially of water-soluble vitamins, which makes supplementation of these unnecessary, but such supplementation is unlikely to be hazardous. The natural mineral and trace element contents of roughage and concentrates tend to be variable and to depend on source and quality. Optimum supplementation is therefore difficult to achieve without chemical analysis.

Where supplements are provided, the total daily intake of each nutrient should fall within the limits imposed for each in Chapters 3 and 4. Where supplements are given in addition to a compounded feed, the amounts from both sources should, of course, be summated. The risks from excess are real for the fat-soluble vitamins, with the exception of α-tocopherol, and for several of the trace elements. Those elements that have notoriously caused problems are Se and I. The reason for this is the fact that the minimum adequate levels and the toxic threshold concentrations are relatively close and some natural feeds are rich sources. Moreover, for iodine the clinical signs of excess are similar to those of deficiency (see Chapters 3 and 12 for diagnostic methods). Excessive consumption of the major minerals is rarely lethal, but inappropriate rates of intake can certainly contribute to poor performance.

Vitamin-like substances, metabolic enhancers and aids to GI health

Many supplements contain items that are not strictly nutrients, but that have some physiological value. Care should be taken, where necessary, that none of these contravenes the rules of competition and racing.

L-Carnitine (β-hydroxy-γ-trimethylaminobutyric acid)

A conditionally essential nutrient, L-carnitine is present in substantial amounts in diets composed of animal products, but is scarce in feeds derived from plants, and thus in horse feeds. It is synthesized from lysine and methionine. Carnitine facilitates the transport of long-chain fatty acids across inner mitochondrial membranes and probably regulates acetyl-CoA:CoA by buffering excess acetyl units during intense exercise. Supplements of 10 g L-carnitine given twice daily doubled the plasma carnitine concentration of intensely exercised TBs, and increased the carnitine content in the plasma of broodmare and their foals, without affecting muscle carnitine metabolism (Benamou & Harris 1993). A few human subjects with leg claudication and angina have been reported to show improved exercise tolerance following carnitine supplementation (Hiatt *et al.* 2001). The young foal, like the neonatal human, has a

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reduced capacity for carnitine biosynthesis, accounting for normal plasma concentrations of about a third those in adults. Healthy adult horses probably synthesize adequate amounts from lysine and S-adenosyl-methionine, yet the effect of dietary fat supplementation on carnitine need has not been examined. L-Carnitine supplementation of ponies is associated with a decrease in postprandial plasma glucose and insulin concentrations, indicating an enhanced glucose tolerance, but with an increase in plasma leptin concentration, not preceded by an increase in insulin concentration This indicated to Van Weyenberg *et al.* (2009) that other factors apart from plasma insulin concentration could influence plasma leptin concentration (see chapter 2).

Carnosine (β-alanyl-L-histidine)

Carnosine is found in high concentrations in equine skeletal and cardiac muscle and is a major physiochemical buffer. The carnosine content of muscle is positively correlated with the proportion of fast-twitch glycolytic fibres. It is a dipeptide of β-alanine and L-histidine. However, Kavazis *et al.* (2003) were unable to demonstrate any reduction in the rate of skeletal-muscle fatigue of rats, resulting from dietary supplementation with β-alanine and L-histidine.

Glucose

The horse has the ability to store large quantities of glycogen in muscle, and concentrations in well-trained, well-fed horses may be 600–700 mmol glucosyl units/kg muscle DM. Attempts have been made to accelerate glycogen repletion rates after extended exercise by use of i.v., or oral, glucose and glucose polymers, but without effect.

Creatine

Creatine was, perhaps, the most widely used supplement in human sport in 1999, as it can increase muscle creatine phosphate and improve performance for short-term high-intensity exercise (Maughan 1999). It had been proposed that supplementary creatine increases water retention and plasma volume of horses, but 25 g creatine monohydrate given twice daily had no effect on maximal treadmill performance of Standardbred trotters, as expressed by muscle metabolic activity and total blood volume (Schuback *et al.* 2000).

N,N-*Dimethylglycine*

N,N-Dimethylglycine (DMG) is an intermediate compound in the metabolism of betaine to sarcosine. In the process there is methyl group transfer to homocysteine with the formation of methionine. It has been suggested that DMG supplementation might delay the onset of fatigue induced by lactic acid. However, mature, conditioned, exercised horses supplemented with 1.6 mg DMG/kg BW failed to show typical increases in blood lactic acid concentration (see Chapter 9). Trimethylglycine also accelerates post-exercise lactate oxidation in untrained, but not in trained, horses.

L-Tryptophan

Supplements have been formulated to reduce nervousness in working horses, with some benefit being achieved (author's experience). L-Tryptophan, is a precursor of serotonin or 5-hydroxytryptophan (5-HT). Noble *et al.* (2008) gave adult TBs an oral dose of L-tryptophan (12.5 mg/kg BW) before a meal of lucerne hay or oats. Plasma tryptophan peaked 1.5–2.0 h after dosing and remained high for several hours in those given hay, but fell sharply in oat-fed horses. Tryptophan had no noticeable effect on behaviour. Recent work by Alberghina *et al.* (2010) indicates that an increased dietary fibre content increases both plasma 5-HT concentration and pH, without influencing plasma tryptophan level.

Dimethylsulphone or methylsulphonylmethane

Dimethylsulphone ($DMSO_2$), also known as methylsulphonylmethane (MSM), is a naturally occurring sulphur compound in both flora and fauna. Its supplementation has been shown to provide biologically available S in the metabolism of S-amino acids and it could be a component of the natural S cycle. MSM is the oxidation product of several natural and synthetic compounds and is an important odoriferous compound in milk. In laboratory animals with spontaneous chronic arthritis, MSM lessens destructive changes in joints when given in relatively large, but quite safe, doses. (For a discussion of glucosamine, MSM and chondroitin sulphate supplementation see Chapter 8.)

Nutraceuticals

A nutraceutical composed of mussel, shark cartilage, abalone, and *Biota orientalis* lipid extract could be helpful in preventing inflammation associated with arthritis and degenerative joint disease in horses (McIlwraith, 2004a; Pearson *et al.* 2009).

Hesperidine

A very large group of polyphenolic compounds (several thousand), distinct from carotenoids, exists in plants with red, blue or yellow pigments and are referred to as bioflavonoids. They include catechins, proanthocyanidins

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(condensed tannins) and flav*a*nones (reduced flav*o*nones). Undoubtedly, more will be learnt about the significance of some of these in future. Some are known to potentiate the antiscorbutic activity of ascorbic acid. Hesperidine is an aglycone flavonoid that has been reported to reduce capillary fragility and/or permeability. It might 'spare' vitamin C, possibly by chelating divalent metal cations (Cu^{++}, Fe^{++}), thus serving an antioxidant function. However, the function is speculative.

Herbs

Many herbs are generally regarded as safe (GRAS) and are used in some horse feed products as flavouring agents, although several contain alkaloids and other phytochemicals such as salicylates, digitalis, heroin, cocaine and marijuana, and are unsafe when provided in large amounts. Some herbs have value in controlling insulin resistance (Tinworth *et al.* 2009).

Williams & Lamprecht (2008) reviewed the known effects of ginseng, ginger, yucca, garlic and Echinacea that are not regulated. Echinacea (a genus of nine species) has been reported to have anti-inflammatory and antioxidant properties. O'Neill *et al.* (2002) gave healthy horses a powdered root extract of echinacoside from *Echinacea angustifolia* (Compositae, common name, Coneflower, found in the eastern US), at a dose of 1.0 g twice daily. This increased the phagocytic ability of neutrophils, stimulated their migration from peripheral circulation and boosted peripheral lymphocyte counts. It also increased the size and concentration of peripheral red cells, haemoglobin concentration and packed cell volume (PCV). The authors concluded that *Echinacea* stimulated immunocompetence and is also a haematinic agent. Ginseng has been shown to exert an inhibitory effect on pro-inflammatory cytokines and cyclooxygenase-2 expression. The anti-inflammatory effects of a single dose of ginger, given post-exercise, have been examined. Yucca contains steroid-like saponins, which produce anti-inflammatory, anti-oxidant, and anti-spasmodic effects. Garlic is widely offered to horses. Nevertheless, when given freeze-dried garlic at >0.2 g/kg BW in each of two daily meals there was oxidative damage to red blood cells, indicated by **Heinz body anaemia**, increases in mean cell volume (MCV), platelet count, and serum unconjugated and total bilirubin concentrations and decreases in RBC count over the time of exposure (Pearson *et al.* 2005). The horses voluntarily consumed a toxic dose of 0.25 g/kg BW twice daily for 71 days, but showed improved haematology 4–5 weeks following removal of the supplement.

Grape seed extract

A grape seed extract given at the rate of 150 mg/kg BW daily to TB race horses decreased blood glucose from 5.77 to 5.32 mmol/L and increased faecal pH from 6.6 to 7.0 (Davies *et al.* 2009). The authors considered it might play a role in prevention of acidosis.

Amino acids and health

There is interest in the oral supplementation of patients following trauma and exhausted long-distance athletes with BCAAs, arginine or glutamine, to overcome leucopenia and immunodepression, associated with stress. Glutamine is a fuel for some cells of the immune system and supplementation enhances cell-mediated immunity and mucosal immunity by modulation of the inflammatory response and preservation of respiratory and intestinal IgA levels. Evidence in man indicates that glutamine could play a role in immunodepression related to trauma and burns, but it might not play a mechanistic role in exercise-induced immunodepression. Arginine and glutamine consumption stimulate radical oxygen species production by neutrophils in stressed rats via $NO^{\cdot}$ and polyamine generation (Moinard *et al.* 2003). The mechanism by which they enhance immunity could be related to this stimulation. No equine evidence is presently available (see also Ammonia and the alanine vehicle, and Protein assimilation, Chapter 9).

Fuller's earth (sodium montmorillonite, sodium bentonite, see also Chapter 3)

Fuller's earth, or bentonite, is a fine greyish-white powder that consists mainly of montmorillonite, a native hydrated aluminium silicate, with which finely divided calcite (calcium carbonate), Mg and Fe are associated. Fuller's earth can adsorb gases produced in the GI tract. It adsorbs water, swelling to about 12 times the volume of the dry powder. A mixture of 20 g/L water has a pH of 9–10 and so has buffering capacity in the GI tract, reducing a rapid decrease in pH during starch overload (see Chapter 11). It is used as a pellet binder and is available as sodium bentonite.

Ergonomic aids

Sodium bicarbonate

Lactic acid accumulates in skeletal muscles and body fluids during and following high-intensity exercise. At the pH of normal muscle tissue it is highly dissociated into lactate and H^+ ions. These ions accumulate, lowering the pH and reducing the activity of glycolytic enzymes, probably impairing the contraction process of working

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muscles, expressed as fatigue. H^+ ions are buffered by the bicarbonate buffering system:

$$H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$$

and the CO_2 is exhaled, disposing of the H^+ ions. Supplementation of horses with sodium bicarbonate at the rate of about 0.4 g/kg BW in 1 L water 3 h before a race of more than two min duration (<1500 m) raises venous blood pH and lactate, improving buffering during metabolic acidosis. (See also Chapter 9.)

FEED STORAGE

Some organic nutrients and non-nutrients in forages, cereals and compounded feeds deteriorate during storage. Labile, readily oxidized pigments, unsaturated fats and fat-soluble substances are destroyed at differing rates depending on their degree of protection, the environmental conditions, their propensity to oxidation and the presence or absence of accelerating substances. The immediate effects include a reduction in feed acceptability to the horse, which is perhaps one of the more discerning and perspicacious of domestic animals when it comes to feed selection.

All the fat-soluble vitamins present naturally in feed – that is, vitamins A, D, E and K – are subject to oxidation, together with the unsaturated and polyunsaturated fatty acids. Rancidity of the latter depresses acceptance, although some stability is imparted by natural and permitted synthetic antioxidants, which are present, or used, in mixed feed. Added synthetic sources of vitamins A and E are much more stable than are their natural counterparts, but they contribute very little antioxidant activity. The critical water-soluble B vitamins are fairly resistant to destruction during storage, although riboflavin in feed will be lost where it is exposed to light. Advice given on labels attached to proprietary supplements and feeds should be followed.

Several factors are essential attributes of good feed stores and grain silos. These are a low and uniform ambient temperature, low humidity, good ventilation, absence of direct sunlight, and freedom from rodent, bird, insect and mite infestation. These characteristics imply that feed and grain stores should be insulated and windowless, but should be well ventilated and both clean and cleanable. Construction materials should be rodent-proof, stacked feed should be raised from the floor and accessible from all sides, and roofs should be free from leaks. Galvanized bins are generally preferred to plastic bins, which can be gnawed by rodents, but metal bins are subject to moisture condensation on the inner surface if the feeds have excessive moisture contents (a maximum of 120 g moisture/kg feed should be achieved). Uniformity of temperature reduces the risk of it reaching dew point.

An Irish study showed that 14% of both Irish and Canadian oat samples were badly contaminated with fungi, although the Irish samples contained slightly more moisture (MacCarthy *et al.* 1976). These fungi grow during the maturation of the crop in the field and normally have a minor role in feed stability. However, high concentrations can affect acceptability to the horse and fungal invasion does detract from the stability of cereals during storage. *Fusarium* species produce toxins that subsequently affect fertility or other aspects of animal health.

Storage fungi are another matter; these species can grow in environments with relatively low moisture and high osmotic pressure, known as conditions of low water activity. Even in these environments, heating, mustiness, caking, and eventually the decay of stored grains, oilseeds and mixed feeds can occur, feed stability and nutritional value can decline, and fungal toxins may be produced. Species of *Aspergillus* and *Penicillium* are the main culprits and all stored feed can be invaded by *A. glaucus*, where there are moisture contents that yield water activities of 0.73–0.78 at temperatures of 5–40°C . However, *Chrysosporium inops* will spread at moisture contents as low as 150–160 g/kg. Most storage fungi have minimum temperatures for growth of 0–5°C, grow optimally at 25–30°C and do not grow at temperatures above 40–45°C. However, *A. candidus* and *A. flavus* (producing aflatoxin) grow vigorously at 50–55°C and *Penicillium* grows slowly at temperatures down to −2°C.

Uninsulated bins and waterproof sacks subject to variations in environmental temperature are particularly prone to moisture condensation on their inner surfaces, even when the average moisture content of the product is low. Of course, the probability of this occurring increases with greater average moisture levels. Once mould growth is initiated, this generates metabolic water and a vicious cycle is established.

Insects, e.g. grain weevils, beetles and flour mites, not only accelerate deterioration of feed and grain, but also generate both heat and metabolic water, and are vectors of fungal spores. Dirty, badly stored grain will be ripe for the hatching of eggs and the multiplication of these insects and mites. Mites will multiply at moisture levels as low as 125 g/kg and a temperature of 4°C. Insects require slightly higher combinations of temperature and moisture. Cleaning and fumigation of long-stored feed is desirable. Hygiene and the storage of feed at low temperatures and in a dry

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condition, without pockets of high moisture, are the greatest assurance for the maintenance of feed quality. Weevils and beetles can be seen with the naked eye. Mites are extremely small but their presence can be detected in meals by observation for a minute, during which time movement of fine feed particles should be apparent. There is a characteristic sour smell from mite infestation, whereas with moulding, discolouration of the grain, dust, and a fungal smell can be detected.

Rodent infestation not only causes feed loss, but rat and mouse droppings introduce the risk of enteric disease, principally salmonellosis, but also other pathogens, including Tyzzer's bacillus (*Clostridium piliformis*). This bacterium is carried in the GI tract of many wild species, e.g. rodents and lagomorphs (see Chapter 11).

Mould inhibitors, such as calcium propionate, propionic acid, sorbic acid and hydroxyquinoline, are used, but they are effective only for coating grain and are relatively ineffectual when included in mixed feed, according to the author's experience. Propionic acid is more effective than the calcium salt.

NATURAL AND CONTAMINANT TOXICANTS IN FEEDS

Feed is sometimes naïvely considered to be a parcel of nutrients, both essential and nonessential. A consideration closer to the truth accepts that natural feeds also contain materials that are either inert, or that influence the metabolism of other dietary constituents, and substances with or without nutritional value, but which are present in toxic concentrations. Many of these potentially hazardous substances are produced, either by the plants themselves or by organisms infecting them and their products. Finally, there are contaminants that result from human intervention and activity. Table 5.10 gives an arbitrary classification to indicate the extent of the problem; the groupings and distinctions drawn are by no means absolute. (See also Chapter 10.)

Table 5.10 Classification of naturally occurring toxicants and toxic contaminants of horse feed.

(1) *Naturally occurring toxicants*
- (a) Protein or amino acid derivatives
  - Lectin (haemagglutinins)
  - Trypsin inhibitors
  - Lathyrogens
  - Nitrates/nitrites
- (b) Glycosides
  - Goitrogens
  - Cyanogens
- (c) Miscellaneous
  - Tannins (polyphenols), saponins
  - Gossypol
  - Phytin, oxalic acid
  - Several antivitamin factors
- (d) Poisonous shrubs and weeds (principally alkaloids)

(2) *Moulds and pathogenic bacteria developing owing to bad harvesting, handling and storage*
- (a) Spores causing respiratory allergies
  - *Aspergillus fumigatus*
  - *Micropolyspora faeni*
- (b) Mycotoxins produced by moulds
  - Hepatotoxins, fumonisins
  - Hormonal
  - Other
- (c) Pathogenic bacteria, or their toxins
  - Pathogenic *Salmonella* spp.
  - *Clostridium botulinum* toxin
- (d) Endophytic fungi in grasses
  - *Acremonium coenophialum*
  - *A. lolii*

(3) *Dietary allergens absorbed from intestines*

(4) *Dietary contamination during manufacture*
- Toxic antibiotics
- Dopes
- Pesticide and herbicide residues
- Nutrients with narrow margin of safety
- Heavy metals unlikely to have nutritional value
- Breakdown products of feed constituents

Toxicants produced by plants

Toxicants likely to be consumed by browsing horses will be discussed in Chapter 10. Here our concern is with substances present in seeds used as feeds.

Condensed tannins

Condensed tannins comprise a diverse group of water-soluble polyphenolic compounds. They are contained in sorghum grains, lentils and many other legume seeds. Peas and field beans, for example, contain significant amounts of condensed tannins, especially where the seed coat is black (not always a reliable guide) or where field beans have coloured flowers. They are not toxic but depress appetite and react with, and lower the digestibility of, proteins and carbohydrates in a pH-dependent manner if they are present in a sufficient concentration. Autoclaving

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or pressure-cooking destroys these tannins, but prolonged treatment is required at lower temperatures. Tannins react intestinally with both dietary and endogenous proteins (for example, slightly lowering trypsin activity in the ileum), increasing their faecal loss. The soaking of some beans before cooking lowers digestibility further, as it seems the tannins diffuse from the seed coat into the kernel where they react with the proteins. If these polyphenols are absorbed, their subsequent detoxification involves methylation. This could lower the protein value of a foal diet, if it is limiting in methionine content.

This enormous group of compounds is under intense scrutiny as the spatial configuration of the phenolic hydroxyl groups in some isoflavonoids confers oestrogenic activity, affecting the fertility of domestic animals and man. Moreover, the antioxidant activity of the polyphenols in green tea, black grapes and many other natural plant products is helpful in reducing atherosclerotic development.

Lipoxygenase activity

Ground lentils and other ground pulses rapidly become rancid following disruption. The polyunsaturated fatty acids are particularly susceptible to lipoxygenase activity. Heat treatment prior to grinding destroys the enzyme.

Antiproteases (trypsin, or protease, inhibitors) and lectins

Two widely distributed groups of compounds are digestive enzyme inhibitors and lectins (previously known as haemagglutinins). The specific compound, its toxicity and susceptibility to destruction by heat, varies among the species of plant within which it is found. Plants producing trypsin inhibitors and lectins include field or horse beans, black grams and kidney, haricot or navy beans. Horse grams, moth bean (*Phaseolus aconitifolius*), certain pulses (also containing amylase inhibitors), groundnuts or peanuts (*Arachis hypogaea*), soya beans and rice germ also contain these substances. Most rice bran fed to horses has had the germ removed, although some residual activity is normally found.

Trypsin inhibitors depress protein digestion, but lectins in commonly consumed legume seeds are more harmful because they disrupt the brush borders of the small intestinal villi, hampering absorption of nutrients, but allowing the absorption of certain toxins. They stimulate hypertrophy and hyperplasia of the pancreas and, according to the evidence of McGuinness *et al.* (1980), may eventually cause adenomatous nodules and pancreatic cancer of exocrine glands. At high concentrations they induce rapid depletion of muscle lipid and glycogen in laboratory animals. Tissue catabolism and urinary nitrogen are increased, and thus growth in young stock is depressed.

The activity of both these groups of substance is destroyed by steam-heating. The trypsin inhibitor activity of field beans is reduced by 80–85% during steam heating at 100°C for 2 min and by about 90% during treatment for 5 min. However, the trypsin inhibitor and lectin activities of kidney beans are more stable and require treatment for two hours at 93°C for adequate destruction. Kidney beans are therefore generally unsuitable for non-industrial processing.

Lathyrism caused by β(γ-L-glutamyl) aminopropionitrile and L-α,γ-diaminobutyric acid

β(γ-L-Glutamyl)aminopropionitrile and L-α,γ-diaminobutyric acid are present in various species of *Lathyrus*. The horse is particularly subject to intoxication bought on by these substances. After long periods of feeding, the Indian, or grass, pea causes a condition known as lathyrism, which is exemplified in the horse as a sudden and transient paralysis of the larynx with near suffocation brought on by exercise. This is caused by degenerative changes in the nerves and muscles of the region and profound inflammation of the liver and spleen. Other closely related species, including sweet pea (*L. odoratus*), wild winter pea (*L. hirsutus*), singletary pea (*L. pusillus*) and everlasting pea (*L. sylvestris*), also cause lathyrism. Although the whole plant contains the toxin, the seeds are the most potent source and it is only partially destroyed by heat.

Goitrogens

Goitrogenic activity is caused by goitrins, which are derived from glucosinolates found in many members of the Cruciferae, including cabbages, rape and mustard. Goitrins are released by enzymes contained within the plant. The destruction of these enzymes by heat treatment largely eliminates the hazard. The effect of goitrins is not counteracted by additional dietary iodine, but further enzymatic metabolism of certain goitrins can release isothiocyanates and thiocyanates. Their antithyroid effect on young horses, in particular, can be overcome by dietary iodine, e.g. in uncooked soyabeans (see Chapter 3). The enzymes are destroyed by heat treatment.

Cyanogenic glycosides and hydrocyanic acid

Cyanogenic glycosides release HCN on soaking the seed. Feeds containing HCN glycosides should be given dry and in small amounts. Larger amounts require preheating to a

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sufficient temperature (adding to boiling water) to destroy the enzyme that releases the HCN. The glucoside in *Phaseolus lunatus* remains stable on cooking. Vicine is the pyrimidine glucoside in *Vicia faba* responsible for the haemolytic anaemia of favism in human subjects deficient in glucose-6-phosphate dehydrogenase, but favism is generally unrecognized in horses. Glycosides, derivatives of α-hydroxynitriles, present in lima beans, sorghum leaves, linseed and cassava (tapioca, *Manihot esculenta*), generate HCN when acted on by the plant enzymes. HCN can cause respiratory failure by inhibiting cytochrome-c oxidase (EC 1.9.3.1). Heat treatment ensures safety, as the poison is released by enzyme activity, so long as the prolonged storage of, foe example, moist seeds or cassava roots, has not led to accumulation of HCN. HCN can react with any thiosulphate present, producing thiocyanate, which is itself responsible for thyroid enlargement after prolonged feeding. The dry storage of many uncooked feedstuffs is essential for the suppression of their enzymatic activity.

Gossypol

Gossypol, occurring in many strains of cottonseed, is the reason the meal is not widely used in horse feeds. In both its bound and free forms, the pigment gossypol reacts, incompletely, with cottonseed protein to depress appetite and protein digestibility and so reduce the efficiency of amino acid utilization; its toxicity can cause circulatory failure and death. The pigment also reacts with dietary iron, precipitating it within the intestines. Fairly large additions of supplementary iron to the diet will then promote further precipitation, which partially suppresses the adverse effects.

Antivitamins

Several antivitamin factors are present in animal and vegetable feeds, but most are of little significance to horses. A thiaminase present in the bracken fern (*Pteridium aquilinum*) is a cause of bracken poisoning, which can be counteracted with large doses of thiamin, and the antivitamin E factor in raw kidney beans is partly destroyed by cooking. Sulphonamides have an antimicrobial action, depressing intestinal vitamin K synthesis.

Phytins and oxalates

High levels of phytic acid, or its salts, in many seed products, when consumed in large quantities will interfere with the availability of Ca and several trace metals, particularly Zn. Oxalates detectable in many plants and present in high concentrations in certain tropical species of grass, kill cattle and cause lameness in horses, owing to precipitation of calcium (see Chapter 10).

Nitrates

Green fodder containing more than 5 g nitrate/kg DM fed alone can cause digestive disturbances and respiratory and circulatory abnormalities. The rapid growth of pasture after high rainfall and excessive use of nitrogen fertilizers leads to high concentrations of nitrates in herbage and, through leaching, contamination of drainage water. Although nitrates are only slightly toxic, they can be reduced to nitrites before or after consumption. High levels of nitrites accumulate in plants after herbicide treatment and during the making of oat hay, owing to nitrate reduction encouraged by inclement weather. In the body, nitrites convert blood haemoglobin to methaemoglobin, which is unable to act as an oxygen carrier. Large intakes therefore cause death. Pigs are more susceptible than are horses, which appear to react similarly to ruminants.

Alkaloids

In South Africa, America and Australia numerous species of the legume *Crotalaria* (one species of which causes Kimberley horse disease) have proved very poisonous in horses. Lesions are induced in the liver by pyrrolizidine alkaloids similar to those found in ragwort (*Senecio*). Alkaloids are considered in Chapter 10 under Grazing management. Other details are found in Table 10.17. Green potatoes (*Solanum tuberosum*) contain the alkaloid solanine. Whereas ruminants would remain healthy, horses are killed by eating quantities of the tubers that are not apparently green.

Moulds

The effects of moulds are of two types:

(1) Certain mould spores, when found in large numbers in badly harvested and stored roughages and cereals, can cause a respiratory allergy when inhaled by sensitive horses (see Chapter 11).
(2) Many mould species, under appropriate conditions of temperature and humidity, produce toxins which have a variety of metabolic effects.

Mycotoxicosis

Fumonisins

Fusarium moniliforme (mating type A, of *F. moniliforme*), or *F. proliferatum*, generally growing before harvest on maize, produce water-soluble fumonisins, and possibly other toxins. These toxins cause small, firm, yellow hepatic lesions, with centrilobular necrosis and fibrosis (Thiel *et al.* 1992). The disease in horses, donkeys and mules

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(equine leukoencephalomalacia, LEM)is also characterized by oedema and bilaterally symmetrical focal necrosis in the medulla oblongata (Marasas *et al.* 1988b), causing blind staggers. In man, oesophageal cancer occurs (Bryden 1995,1998). The mating type of *F. moniliforme* found on sorghum does not produce fumonisins (Bryden 1998, personal communication). Fumonisins bear a structural relationship to the sphingoid base, sphingosine, and so interrupt sphingolipid metabolism in the central nervous system with a rise in serum sphinganine. Of six fumonisins, FB_{1a} and FB_2 are the only significant equine toxins. Depending on dose, clinical signs can develop within eight days and include apathy, somnolent appearance with protruding tongue, reluctance to move backwards, aimless circling and ataxia (Nelson *et al.* 1993).

F. moniliforme is an aggressive colonizer of maize pre- or post-maturity (Bryden *et al.* 1998) and especially that infested by weevils (Smalley 1992). FB_1 is the major phytotoxin (Lamprecht *et al.* 1994), but is not acutely toxic (Gilchrist *et al.* 1992). The fungus can colonize, reproduce and produce toxins on a wide range of mature plant products, plant debris and on autoclaved maize seed when incubated at 20–22°C (Marijanovic *et al.* 1991; Nelson *et al.* 1993), or when grown on autoclaved rice for 28 days at 22–26°C (Mirocha *et al.* 1992). It is a facultative parasite (Gilchrist *et al.* 1995) and an endophyte (Bryden, personal communication; Marasas *et al.* 1988a). In Australian maize samples drawn from both field and storage, 84% were infected with *F. moniliforme* (Pitt & Hocking 1996), and of 675 visually normal maize kernels, 60% were infected with *F. moniliforme*, while only seven of 100 samples of Australian maize for human and animal food were negative for FB_1 (Bryden *et al.* 1998). Almost half these animal feed samples contained FB levels of 5 mg/kg and above (mean 7.8 mg/kg). The highest concentration recorded by Bryden, 40.6 mg/kg, was detected in maize causing LEM in horses. Fumonisin B_1 has worldwide distribution. The range of content in maize grain samples from Iowa was 2.5–3.5 mg/kg and in South Africa 0.4–1.8 mg/kg.

Concentrations of as little as 1.3 and 0.1 mg Fumonisin B_1 and B_2 /kg of feed, respectively, cause LEM (Thiel *et al.* 1992). Fumonisin intakes of as little as 0.6 mg/kg BW/day (approximately 20 mg/kg air-dry feed; Thiel *et al.* 1992) and oral doses of 1.25–4.0 mg FB_1 per kg BW (approximately equivalent to 40–130 mg/kg feed; Kellerman *et al.* 1990) caused pathological effects in horses. A daily intravenous dose for of 0.125 mg fumonisin B_1 per kg BW for seven days caused neurotoxic signs in a horse on day 8 (Kellerman *et al.* 1990). However, most cases of LEM have been associated with concentrations of >500 mg/kg diet.

Colic and mycotoxins

At the concentrations found in contaminated feedstuffs only T_2 toxin, at concentrations >50 µg/kg feed, and zearalenone, at concentrations >70 µg/kg feed, have caused colic in horses. The author has witnessed several cases of impaction colic and death amongst Arabian horses consuming slightly mouldy timothy hay. No colic has been observed with aflatoxin, deoxynivalenol or with fumonisin B_1.

Ergotamine, ergometrine

There are numerous historical references to the consequences of ergot of rye consumption, namely abortion and other effects of blood-vessel constriction. The mould, *Claviceps purpurea*, also infects rye- and other grasses, and is therefore a hazard in pasture and hay at high concentrations.

Aflatoxin

Reports, especially from Thailand and the US, have recorded deaths or brain, heart and particularly liver lesions, and haemolytic enteritis in horses receiving aflatoxin B_1 produced by *Aspergillus flavus* at levels of less than 1 mg/kg in cereals, peanuts and even hay. Horses and ponies are more susceptible to acute aflatoxicosis than are pigs, sheep and calves, as daily intakes of 0.075 and 0.15 mg aflatoxin B_1/kg BW are lethal for ponies in 36–39 and 25–32 days, respectively (Cysewski *et al.* 1982).

Zearalenone

The toxin zearalenone, produced by Fusarium species, causes vulvovaginitis and reproductive failure in females of several domestic species. Whereas aflatoxin usually develops during storage, this toxin can develop preharvest. Raymond *et al.* (2005) measured the effects of feeding Fusarium mycotoxin (deoxynivalenol, 15-acetyldeoxynivalenol and zearalenone)-contaminated grains to exercising horses. Feed intake and body weight were depressed, without affecting treadmill performance. No benefit on feed intake occurred from giving a polymeric glucomannan mycotoxin adsorbent (see also Zearalenone, Chapter 10).

Dietary allergens (other than mould spores)

Dietary allergens are not contaminants, but certain horses can react to normal protein constituents of feed (Plate 5.4). The vascular reaction of the skin is raised oedematous

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Plate 5.4 A two-year-old TB gelding with widespread 'bumps' on the head, neck, shoulders, ribs and flanks. An allergic reaction to bran and oats was detected in the blood serum. The horse recovered over several months when its diet consisted of a high-fibre cubed diet, low in cereal and with water *ad libitum*.

plaques of varying size (0.5–10 cm diameter), known as urticaria, to which the TB is particularly prone. The effects are normally overcome, according to the author's experience, by removal of the offending source from the diet, or administration of dexamethasone i.v. 0.04 mg/kg BW (Pilsworth & Knottenbelt 2007). Problems of cross-reactivity in which related sources of proteins yield similar reactions can pose problems of interpretation. Foods containing gluten are frequently implicated, but a single insect bite can cause generalised urticaria in a sensitised horse.

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Hypersensitivity to drugs: penicillin, clenbuterol and especially phenylbutazone, are frequent, either to the drug, the carrier, or preservative.

Human intestinal epithelial cells take up and process food antigens (Heyman 2001). Among the peptidic metabolites generated during transepithelial transport of luminal antigens, some have a molecular mass compatible with a binding to restriction (major histocompatibility complex) molecules. These can be up-regulated on enterocytes, especially in inflammatory conditions, such as gastroenteritis. Nutritional factors can influence the properties of this epithelial barrier and the immune response to luminal antigens. In particular, Sanderson (2001) indicated that changes in diet can alter the expression of genes in the gut epithelium encoding for proteins that signal to the mucosal immune system. The epithelium thus acts as a transducing monolayer signalling between the contents of the intestine and the mucosal immune system.

Heavy metal and mineral contamination from pastures

The approximate maximum tolerable dietary concentrations of heavy metals and some other elements (influenced by the concentrations of essential minerals and trace elements) are shown in Table 5.11 (see also Frape 1996, 2002b).

Table 5.11 Approximate maximum tolerable dietary concentrations of heavy metals.

	mg/kg feed DM
Arsenic (As)	2
Cadmium (Cd)	10
Fluorine (F)	50
Iodine (I)	1
Lead (Pb)	20
Mercury (Hg)	0.2
Molybdenum (Mo)	200 (excluding any possible interaction with Cu availability)
Selenium (Se)	2
Aluminium	950

Dietary Al in excess of 1500 mg/kg causes a reduction in P absorption and an increased P requirement in ruminants and horses, whereas, over four weeks, diets containing 930 mg Al/kg had a negligible effect on nutrient digestibility and mineral metabolism in adult TBs (Roose *et al.* 2001).

Pb is one of the commonest causes of poisoning in cattle, sheep and horses. Signs of toxicity are more frequent in young horses and include lack of appetite, muscular stiffness and weakness, diarrhoea and, in an acute form, pharyngeal paralysis and regurgitation of food and water. Pb accumulates in the bones and as little as 80 mg/kg diet eventually causes toxic signs, sometimes precipitated by other stresses. Natural feeds with 1–5 mg Pb/kg cause no problems. The acute lethal dose is 1–1.8 g/kg BW as lead acetate or carbonate. The chronic lethal dose depends on many factors but is said, on average, to be about 12 mg Pb/kg BW daily for 300 days.

The contamination of pasture with Pb, Cd and As (derived from mine workings, dumping or sewage sludge, aerial dust and water erosion, even from car batteries and lead shot) are local risks. Where the pasture is dense, undoubtedly the greater problem arises from surface contamination of plants, but where it is sparse, soil, either rich in these heavy metals or contaminated by them, can be consumed in sufficient amounts to cause problems. Lead shot is somewhat hazardous to horses but, because they graze closely, consumption can be greater than might be generally appreciated, followed by some solution in the stomach. Where grass is ensiled, entrapped shot partly dissolves during fermentation and highly toxic levels of 3800 mg soluble Pb/kg DM have been detected in England by the author (Frape & Pringle 1984). Whereas Pb mainly contaminates the surfaces of plants, Cd is readily absorbed and is accumulated from soils rich in this element. Of common pasture species, the daisy (*Bellis*) accumulates 60–80 mg Cd/kg (30 times as much as in grasses) from contaminated soils (Matthews & Thornton 1982).

Pastures to the leeward of steel and brick works amass abnormally high concentrations of F. The horse is less subject to fluorosis than cattle and sheep, but damage to its bones and teeth has been induced by this element. Horses will tolerate 50 mg/kg feed for extended periods. Hg poisoning, shown by colic and diarrhoea, has arisen in horses as a consequence of the mistaken use of dressed seed as a feed. Chance exposure from other sources is unlikely.

The required trace elements, I, Se and Mo, can be consumed in toxic quantities following natural accumulation in vegetable materials (see also Chapter 3). Seaweed can be a source of excessive I, and certain accumulator plants store large quantities of Se from Se-rich soils. When these plants die it is said that they in turn deposit Se in a form

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readily absorbed by neighbouring plants. Many Se-rich areas are sparsely covered and the consumption of soil rich in the element is another source of risk. Mo is readily absorbed by most plants from soils containing excessive amounts. The ingestion of soil rich in Fe and S is known to reduce Cu absorption in grazing animals. Season and the extent of soil drainage can influence the accumulation of several metals in herbage. Concentrations tend to be higher during the winter months (see also Chapters 3 and 10).

Pesticide residues

Many normal feeds contain trace amounts of pesticide residues but, apart from gross contamination through negligence, the amounts normally detected are insufficient to cause any problem to horses (after herbicide treatment, pastures should be rested for two weeks before grazing is permitted). The rodenticide zinc phosphide (now rarely used) has been consumed in lethal amounts by horses; it releases the poisonous gas phosphine (PH_3) and ejects it from the stomach. If zinc phosphide poisoning is suspected, a nasogastric tube should only be used in the open air. The acute lethal dose in horses is 20–40 mg/kg BW, or about 15 g for a horse (about 5–10 g for a pony).

Ethylene glycol (EG)

Swor *et al.* (2002) successfully treated a gelding that presented with tachycardia and discomfort after consuming ethylene glycol (EG). Toxicity is attributable to EG oxidation products, through the action of alcohol dehydrogenase (ADH, EC 1.1.1.1). ADH has greater affinity for ethanol than for EG and the metabolism of EG may be arrested by early i.v. administration of a 20% solution of ethanol at 2.5 mL/kg BW. In this gelding, intermittent infusions were continued for 32 h to maintain a therapeutic blood level of ethanol, with a return to full health. An alternative therapy in man is administration of fomepizole, or 4-methylpyrazole (Antizol®) which inhibits alcohol dehydrogenase (Brent 2001) without serious adverse effects, although no evidence exists of its application to equine toxicosis.

FEED ADDITIVES

Synthetic antioxidants

A number of natural and synthetic antioxidants are permitted to be added to animal feeds in order to slow the rate of feed oxidation during storage. In most countries the list includes various forms of ascorbic acid, natural tocopherol-rich extracts, α-, δ-, and γ-tocopherols, propyl- octyl- and dodecyl-gallates, butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and ethoxyquin. The maximum content in a complete feedstuff is strictly controlled for each substance, singly or in combination. These maxima differ slightly between countries. For example, the last three listed here are permitted in the UK to a maximum, singly or together, of 150 mg/kg total feed (Anon 2000b), while in the US they are permitted, singly or together, to a maximum of 200 mg/kg total fat or oil (including volatile oils) content. Tertiary butyl hydroquinone (TBHQ) is permitted to a maximum of 200 mg/kg total fat or oil, singly or in combination with other preservatives (AAFCO 2005) in the US, but it does not yet appear in UK Feeding Stuff Regulations. All these additives are GRAS (NRC 2007). (See also Chapter 6, 'Shelf-life of Feeds'.)

Anti-moulding agents

A large number of other GRAS chemicals are added as preservatives, to maintain feed value and inhibit microbial colonization and growth (Anon 2000b; AAFCO 2005). Most of these are organic acids and their salts, including propionic acid and its salts. Propionic acid may be added without conditions, and is effective at 0.3–1.0% by weight to control mould growth of grains. In the author's experience this is effective when the acid is added to whole grains, but not so when it is added to ground, or crushed, grain or to mixed feed in a humid environment.

Flavours

A wide variety of spices, seasonings, flavourings, and natural oils are considered GRAS. Foals discriminate against salty, sour and bitter solutions compared with water (NRC 2007). Flavour preferences have been reviewed by Goodwin *et al.* (2005a,b), who found the following order of preference for the top eight flavours added to a cereal by-product: fenugreek > banana > cherry > rosemary > cumin > carrot > peppermint > oregano. Mint was preferred to garlic as a flavour in pelleted grain diets (Cairns *et al.* 2002). When tart cherry juice (rich in malic acid) was given to horses for 2 weeks before hard exercise it reduced post-exercise serum AST activity (Ducharme *et al.* 2009).

Domestic animal feed-additives

Several drugs are used in the feed of farm animals to promote growth, counteract diarrhoea and parasitic infection, and influence the carcass. Most of these drugs have little, if any, ill effect on horses when present in the diet at normal feed levels, or when horses are mistakenly given feed containing drugs intended for other species. Higher

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Table 5.12 Ionophore antibiotic toxicity.

Antibiotic	Active chemical usage rate (mg/kg feed DM)	Equine lethal oral dose (LD_{50}) (mg/kg BW)[1]
Salinomycin	60	approx. 0.6
Narasin	70	approx. 0.7
Monensin	100–120	1.38–3[2]
Lasalocid	30–50	21.5

[1]Lethal dose in mice is about ten times higher.

[2]Matsuoka *et al.* (1996) found an LD_{50} of 1.38 mg/kg BW for a single dose by gavage using mycelial monensin.

dosages are a different matter. Although framycetin sulphate is given in cases of flatulence, or fermentative colic, the persistent use of some antibiotics, especially oxytetracycline, causes a severe upset to the intestinal flora, including a fungal overgrowth, precipitating acute and intractable diarrhoea, lethargy and lack of appetite. Two other drugs, monensin and lincomycin, can have severe toxic effects in horses when given at normal feed rates.

Ionophore antibiotics

Ionophores are polyether carboxylic antibiotics given to poultry for the control of coccidiosis and to ruminants to improve feed utilization. There are presently six of these carboxylic ionophores: monensin, lasalocid, salinomycin, narasin, maduramicin and laidlomycin. Dietary threshold concentrations exist for these, below which no adverse effect has been observed, but at higher rates of intake, severe colic, sweating, trembling and occasionally haematuria can occur. Reliable and quantitative equine data are not available for any of them (Table 5.12).

Monensin sodium

Monensin is fed to beef cattle to promote growth and to poultry as a coccidiostat. Poultry feed containing 100 mg monensin/kg, the normal feed level, has severe toxic effects in horses. At a level of 30 mg/kg in the feed, horses experience a reduced appetite and uneasiness, although Matsuoka *et al.* (1996) indicate that horses can tolerate the highest usage rate for cattle of 33 mg/kg feed. Continuous dosing at a level of 100 mg/kg (about 2.5 mg/kg BW) is lethal in a matter of 2–4 days to about half the horses. In the author's experience, horses present signs anorexia, posterior weakness, profuse sweating, tachycardia, occasionally muscular tremors, polyuria, myoglobinuria (dark-brown urine), elevated urinary potassium, elevated serum levels of muscle enzymes, progressive ataxia and recumbency. Post-mortem examination shows myocardial degeneration and monensin is confirmed by analysis of stomach contents. In the early stages of toxicity, recovery is frequently achieved by removing the feed and dosing the horse with mineral oil, although it can suffer permanent heart damage, with increased risk during hard physical exertion. Hughes *et al.* (2009) observed clinical effects in 29 horses and eight ponies that had received <1–2.5 kg feed daily, contaminated with monensin sodium (52.1–179 mg/kg feed) for 1–9 days. Eleven animals showed abnormal echocardiographic effects, although in 21 animals these changes had returned to a normal range 11 months later and normal work was resumed.

Lincomycin

Lincomycin is an antibacterial drug sometimes included in pig feed. It is less toxic than monensin in horses, but above dose levels of 80 µg/kg BW daily (5 mg/kg total diet fed continuously) metabolic signs of toxicity and evidence of liver damage have been observed by the author.

Growth hormone

There is increasing use of exogenous bovine growth hormone in TB studs. This increases the extent of 'double muscling' and rate of growth with little effect on ultimate height. The author's evidence indicates that it also increases the risk of DOD.

PROHIBITED SUBSTANCES

The list of proscribed drugs or prohibited substances embraces a very high proportion of the drugs permitted in livestock feeds by EU legislation, and the detection of any of them, or their recognizable metabolites, in urine, blood, saliva or sweat will lead to the disqualification of a horse subject to the Rules of Racing. Apart from drugs that might be used directly in horses, any antibiotic, growth promoter or other drug given to poultry, pigs or ruminants must not be detected in the above fluids. Some drugs acting on the cardiovascular system, some antibiotics and one or two anabolic agents have been detected in contaminated feed ingredients – oats, soya-bean meal, bran – and in feed additives, and they can occur in feeds for other classes of stock mistakenly fed to horses. In practice, those of chief concern are the xanthine alkaloids – theobromine and caffeine, and its metabolite theophylline.

Caffeine is present in tea, coffee, coffee by-product, cola nuts, cacao and its hull (available as a by-product),

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and in maté leaves. Tea dust contains as much as 1.5–3.5% caffeine, whereas coffee by-product, contains only some 200 mg caffeine/kg. Small amounts of theophylline are found in tea, but as much as 1.5–3% theobromine is typically present in cacao beans, and its waste-product, the hull, contains as much as 0.7–1.2%. Theobromine also occurs in cola nuts and tea. The widespread international traffic in coffee and cocoa beans, and in their by-products, constitutes a formidable risk through their contamination of the means of transport, from ships at one extreme to hemp sacks at the other. These, in their turn, put in jeopardy cereals, pulses and other raw materials moved from one place to another, leading to infringements of the Rules of Racing through the inadvertent consumption of contaminated batches of these feeds. Gross contamination and its control in animal feedstuffs has been discussed in a code of practice published by the United Kingdom Agricultural Supply Trade Association (UKASTA, 1984).

After an oral dose of caffeine, about 1% appears unchanged in the urine, the excretion of which is almost complete after three days. About 60% of the caffeine is excreted in the urine as metabolites, including theophylline and theobromine. Traces of the latter continue to be excreted for up to 10 days, whereas theophylline excretion is virtually complete after 4–5 days. Thus, the unintentional use of these alkaloids, or of coffee or cocoa wastes containing them, can result in their detection in the urine within 1 h of an oral dose and up to 10 days thereafter. Experiments have shown that theobromine can be detected in the urine of TB geldings when they receive 7 kg/day of cubes containing 1 mg theobromine (in the form of cocoa husk)/kg. The extent of the excretion curves in urine of some, but not all, other drugs has been determined. Both caffeine and theobromine are readily absorbed from the intestinal tract and they soon impart their effects on cardiac and respiratory stimulation and on diuresis. They affect horse speed only when they are used in high doses. The threshold level for theobromine in the urine, accepted by the Stewards of the Jockey Club, is 2 µg/mL urine, by HPLC determination (Haywood *et al.* 1990).

Three other proscribed compounds occurring naturally in plant tissue are: hyoscine, present in the Jimson weed (found in soya bean fields); hordeine, present in germinating barley, alfalfa and canary grass (*Phalaris canariensis*); and morphine, present in poppy (*Papaver somniferum*) seeds and seed capsules.

EU Regulations on pharmaceutical preparations

As horses may enter the human food chain in certain European countries, there is a restriction on the use of some pharmaceutical preparations to avoid residues from entering the food chain. The following is the position at the time of writing this Edition (summer 2009).

The Essentials List (1950/2006) extended the range of substances that could be used to treat horses intended for human consumption (with a 6 months exclusion from the food chain) beyond those listed in Annexes I-III of the residues regulations (2377/90). Substances listed in Annex IV (such as metronidazole) are excluded from consideration.

A wide-ranging review of the residues regulations (2377/90) has been completed and a series of amendments has been agreed by the European Parliament. One of these relates specifically to equine veterinary medicines:

> *3. By way of derogation from Article 11, and in accordance with the procedure referred to in Article 89(2), the Commission shall establish a list of substances:*
>
> ** which are essential for the treatment of equidae, or * which bring added clinical benefit compared to other treatment options available for equidae, and for which the withdrawal period shall not be less than six months according to the control mechanisms laid down in Commission Decisions 93/623/EEC and 2000/68/EC.*

STUDY QUESTIONS

(1) How would you set about changing an existing stable over from hay to haylage?
(2) What factors should be considered when it is proposed to introduce fat into a rationing system?
(3) If probiotics are to be introduced, what checks should be made concerning: (a) the product and (b) the feed system for their use?

FURTHER READING

Jansen, W.L., Sloet van Oldruitenborgh-Oosterbaan, M.M., Cone, J.W., *et al.* (2007) Studies on the mechanism by which a high intake of soybean oil depresses the apparent digestibility of fibre in horses. *Animal Feed Science and Technology*, **138**, 298–308.

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6
Estimating Nutrient Requirements

What good receipt have you for a horse, that hath taken a surfeit of provender. This comes commonly to such horses as are insatiable feeders and therefore it is requisite that they be dieted, especially if they have too much rest, and too little exercise.

T. De Gray 1639

The formulation of adequate diets requires knowledge of three types of information:

(1) the requirement of the horse for each of the nutrients and energy, affected principally by the horse's size and function;
(2) the nutrient composition of each of the appropriate feeds available; and
(3) the capacity of the horse for feed.

Tables have been derived to provide the information necessary for (1) and (2), and some discussion is appropriate first concerning (3).

RELATIONSHIP OF CAPACITY FOR FEED TO BODY WEIGHT

The daily requirements of horses and ponies have been estimated in terms of the amounts of each nutrient – minerals, trace elements, vitamins and amino acids (or, more realistically, protein) – required per day for the various functions of maintenance, growth, lactation and so on. The normal vehicle for these nutrients is the daily feed, and if a particular horse were to consume twice as much feed as another horse fulfilling the same tasks then it might be reasonable to suppose that the nutrient concentration in the diet of the first horse need be only half that in the diet of the second. Thus, in order to make useful statements about dietary composition and to facilitate calculation of adequate diets, it is necessary to predict reliably the appetite of a horse, or group of horses, for feed DM. The appetite and capacity of horses for acceptable feed are regulated by five dominant and related factors:

(1) the volume of different parts of the intestinal tract;
(2) the rate of passage of the digesta;
(3) the concentration of certain digestion products in the intestine;
(4) the energy demands of the horse; and
(5) the energy density and its chemical form in feed.

The concentration of certain digestion products in the intestine seems to control meal size and the rate of passage of the digesta will be modulated by the physical form of the feed. The volume of different parts of the intestinal tract is controlled by the body size of the animal, but to some extent is modified by breed and adaptation. Edouard *et al.* (2008) showed that DM digestibility declined with declining forage quality, the feed intake of some horses decreasing, above maintenance needs, with decreasing quality; but on average intake increased as protein content, and digestibility declined and as fibre content increased. Smith *et al.* (2007) employed the alkane pair technique in estimating DM intake. The method involved dosing two or three times daily with even-chain alkanes, using dotriacontane (C32) and hexatriacontane (C36) as external markers. C31 provided a more accurate estimate of intake than C33 as internal marker. The accuracy of estimated intake improved progressively as the number of faecal sampling days was increased from one to six.

The most common, easily corrected, deficiencies in home-prepared feed mixtures are those of Ca, P, protein, NaCl and possibly vitamin A. However, in addition

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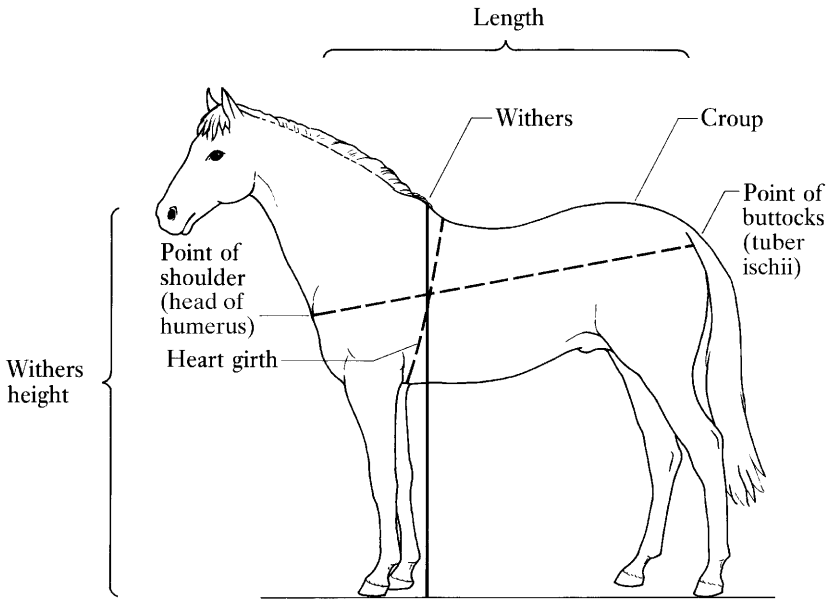


Figure 6.1 Linear body measurements used in estimating body weight of horses and ponies.

to water, the fundamental, and long-term need of the horse is for a digestible source of dietary energy. Ideally, in situations of moderate work or productivity, the energy demands should just be met by the appetite and capacity of the horse for feed. Not only capacity, but also energy requirement for a variety of functions, is closely allied to body weight, varying with the extent of gut fill. A means of estimating weight is fundamental to any rationing system.

In the absence of a weighing machine, the most reliable predictions include a measurement of the heart girth following respiratory expiration (Figure 6.1). As conformation changes with age and differs among breeds, the measurement of girth alone is bound to yield only an approximate estimate. Improvement on this is achieved by inclusion of the length from the point of the shoulder to the point of the buttocks (Figure 6.1), and the equation given under 'Measurement of energy expenditure in maintenance and exercise', below, provides the appropriate relationship.

For many, withers height will be a more familiar index of size. Figure 6.2 shows its approximate relationship to body weight for several types of horse and pony, and Figure 6.3 delineates the change in withers height with age during growth. Suggested daily allowances for, and capacities of, horses of different liveweights are indicated in Figure 6.4. The capacities are approximately 10% higher than the voluntary intake values of 28–29 g/kg BW for a mixed diet in Arabian and Quarter Horse foals 5–8 months old (Turcott *et al.* 2003). The allowances given to idle horses would be lower than those shown, whereas lactating mares, or working horses, will consume more feed. Among seven racing stables in the US the average intake of concentrate by 3–4 year olds was 6.16 kg/day (4.9–7.5 kg) and that of hay 9.37 kg/day (6.4–11.9 kg) (Glade 1983a). Among 2–4 year olds in Newmarket the concentrate intake averaged 8.15 kg/day and that of roughage 5.5 kg/day per horse (author's unpublished observations). The lower intake of roughage in the UK could reflect the generally poorer nutritional quality of this feed as supplied to horses. The US horses averaged 496 kg BW whereas those in the UK were about 480 kg BW.

Measurement of energy expenditure in maintenance and exercise

Maintenance energy expenditure is eventually expressed as heat evolved at constant body temperature, but the expenditure for production furnishes either work done or useful derivatives, such as growth, milk, products of conception etc. Heat evolved is measured by direct calorimetry, but energy expenditure for maintenance and for work are both more simply measured by indirect calorimetry. This method has been widely applied in equine nutrition research and is summarized here.

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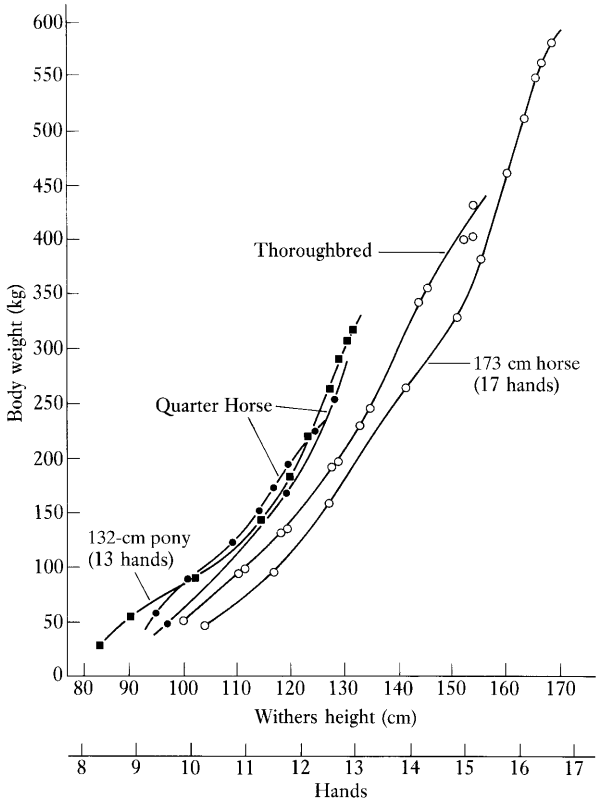


Figure 6.2 Relationship between body weight and withers height in normally growing horses and ponies (1 hand = 4 in = 10.16 cm). Note that horses and ponies achieve mature height before mature weight so the curves must be concave upwards as maturity is approached (*upper end of curve*). Furthermore, as the curves are not coincident, withers height is generally not a good predictor of BW (data from Green 1961, 1969; Hintz 1980a; Knight & Tyznik 1985; R.W.W. Ellis & R.A. Jones 1984, personal communications).

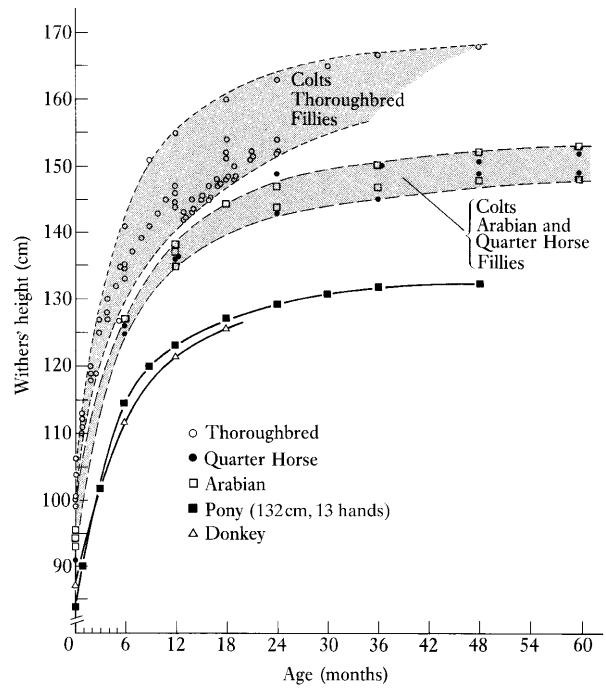


Figure 6.3 Expected withers height of normally growing horses and ponies at various ages (1 hand = 4 in = 10.16 cm) (data from Green 1961, 1969; Hintz 1980a; Knight & Tyznik 1985; R.W.W. Ellis & R.A. Jones 1984, personal communications).

How is the energy requirement of maintenance determined?

The measurement of the inherent activity of the horse's organs and tissues, independent of exercise and other extraneous factors, is the measurement of basal metabolic rate (BMR) under basal conditions when the body is at absolute rest. The specific dynamic action of digestive processes is absent and air temperature and air movement are such that sweating is minimal and the sympathetic nervous system is not activated to help maintain body temperature. For practical purposes it is more appropriate to measure maintenance energy expenditure when feeding allows the maintenance of body weight, when movement is that of standing and walking slowly without a rider.

Maintenance requirement is a function of body weight, obtained either by weighing the horse, or by estimation from linear measurements, as follows (Carroll & Huntington 1988):

$$\text{Body weight (kg)} = \frac{\text{heart girth (cm)}^2 \times \text{length (cm)}}{11\,877}$$

Carroll & Huntington examined the effect of condition score (maximum 5) on the accuracy of weight prediction and found a difference in the best constant (denominator value) of the equation above between animals with a condition score of 2.5 or less, for which the denominator is 12 265 cm^3/kg, and those for which the score is ≥3, for which the denominator is 11 706 cm^3/kg. These values are used to replace '11 877' in the above equation and so to improve the estimate. Age affects conformation and so also the preferred denominator. Wilson *et al.* (2003b)

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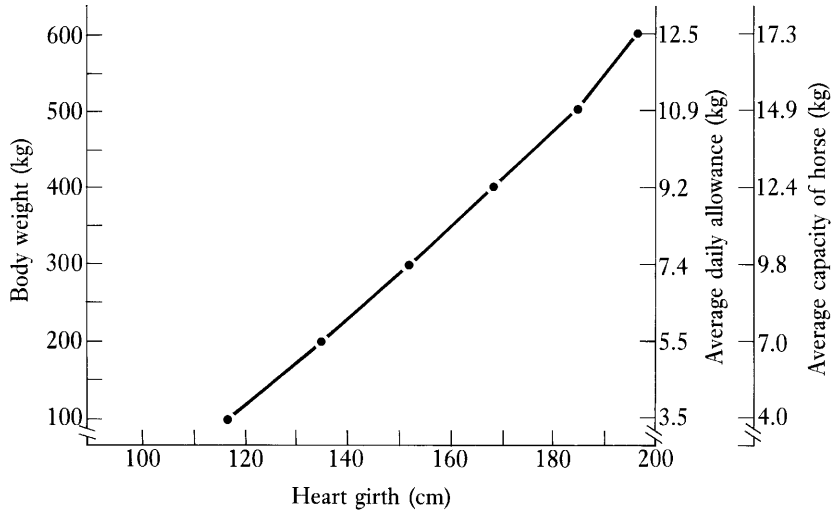


Figure 6.4 Relationship for horses and ponies between heart girth and average live weight, average daily capacity for concentrates and forage, and average allowance of concentrates and forage (12% moisture).

derived a constant of 10 080 cm^3 for weanling horses. Staniar *et al.* (2003), with a carefully conducted study of TBs from birth to 17 months, fitted a slightly cumbersome broken-line model with different coefficients. This derived body volumes V_{t+1} from girth (G), carpus circumference (C), length of front leg (F) and body length (B):

$$V_{t+1} = \left[\left(G^2B\right) + \left(C^2F\right)\right]/4\pi$$

$$\text{Where } V_{t+1} < 0.27 \text{ m}^3, \text{ BW (kg)} = 1093V_{t+1},$$

$$\text{Where } V_{t+1} \geq 0.27 \text{ m}^3, \text{ BW (kg)} = 984V_{t+1} + 24.$$

Early estimates of energy needs

During the 1880s, Müntz & Grandeau in Paris, Wolff and colleagues in Hohenheim and, during the 1930s, Ehrenberg and his colleagues in Breslau, all measured the energy requirements for maintenance and work by feeding defined rations and recording changes in body weight. In 1911 these data were supplemented by respiratory experiments in Berlin by Zuntz, in Bonn by Hagemann, and subsequently in 1931 by Fingerling in Mökkern (Klingeberg-Kraus 2001) and by Brody (1945) in Missouri. The latter recorded the results in his classic text *Bioenergetics and Growth*.

Two general methods of estimating heat evolved in maintenance

In the absence of work achieved or other useful output, e.g. milk or weight gain, the metabolic activity of maintenance results in heat evolved. This is measured through direct calorimetry by the absorption of heat in a water jacket and by collecting expired moisture. Alternatively, indirect calorimetry requires measurements of respiratory exchange and of urinary N excretion, when the reactions are neither endothermic, nor partially anaerobic, and the energy equivalent of O_2 is known.

Indirect calorimetry

Indirect calorimetry was first used by Lavoisier and Laplace. It is based on the fact that O_2 consumption and CO_2 production are normally closely correlated to heat production, as illustrated by the oxidation equation for one gram-molecule (mole) of carbohydrate:

$$C_6H_{12}O_6 + 6O_2 \quad = \quad 6CO_2 + 6H_2O + 2837 \text{ kJ}$$

$$180 \text{ g} \qquad 134.4 \text{ L} \qquad 134.4 \text{ L}^*$$

where 1 L = 1 dm^3 or 10^{-3} m^3

under standard conditions of temperature and pressure (STP*). Thus, the consumption of 6 mols (6 × 22.4 = 134.4 L) O_2 in the oxidation of 1 mol hexose yields 2837 kJ (or 1 L O_2 yields from carbohydrate oxidation 21.1 kJ). Similarly, from the oxidation of mixed fat, 19.6 kJ are generated per litre O_2 and from the oxidation of mixed

*Standard temperature and pressure are the conditions the volume of dry gas would assume at 0°C and 760 mm pressure.

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Table 6.1 Constants at STP for protein, fat and carbohydrate when oxidized in the animal body (Brouwer 1965a,b).

		Consumed on oxidation of 1 g		Set free on oxidation of 1 g			
	Carbon %	O_2 g	O_2 L	CO_2 g	CO_2 L	Heat kJ	Respiratory quotient (RQ)
Protein*	52.00	1.366	0.957	1.520	0.774	18.4	0.809
Fats**	76.70	2.875	2.013	2.810	1.431	39.7	0.711
Starch	44.45	1.184	0.829	1.629	0.829	17.6	1.00
Saccharose	42.11	1.122	0.786	1.543	0.786	16.6	1.00
Glucose	40.00	1.066	0.746	1.466	0.746	15.6	1.00

*The approximate composition of protein is: N: 16%; C: 52%; 23.8 kJ/g.
**It is assumed that fats contain only a small proportion of short-chain fatty acids.

protein 20.2 kJ per litre O_2 consumed. As the heat equivalent of O_2 consumed varies with the nature of the substrate oxidized, it is necessary to know the composition of the fuel mix (carbohydrate, fat, protein) oxidized. The amount of protein oxidized is calculated from urinary N excreted, assuming that protein contains 16% N and that all urinary N is derived from protein (i.e. protein is therefore approximately = N × 6.25).

The relative amounts of fat and carbohydrate oxidized are determined from the non-protein respiratory quotient (RQ), i.e. mols of CO_2 produced to mols of O_2 consumed. For carbohydrate this is:

$$6CO_2/6O_2 = 1.00$$

The RQ for mixed fat is 0.71 (for short-chain fats the RQ is nearer 0.8 and for long-chain fats it is nearer 0.7) and for mixed protein it is approximately 0.81.

The RQ does not have the rigorous significance indicated above, as huge amounts of CO_2 are produced in the GI tract by anaerobic bacterial fermentation and by liberation of CO_2 from bicarbonates. (It will be noted that the heat of bacterial fermentation, as in ruminants, can help compensate for heat lost in cold climates and thus the horse avoids a shivering response in maintenance.) Excess CO_2 is liberated under conditions of acidosis and over-ventilation, or stored during alkalosis. So the rate of O_2 consumption is the better measure of heat production. Furthermore, the energy equivalent of CO_2 has a relatively wide range, whereas the range for O_2 is relatively narrow over the possible range of RQ. Finally, the RQ of protein corresponds to the average energy value of O_2, 20.2 kJ/L. From these arguments it is apparent that the measurement of energy metabolism requires knowledge only of the rate of O_2 consumption, corrected to standard conditions. Heat production is calculated by assuming an equivalent energy value for O_2 of 20.2 kJ/L. This corresponds to the RQ of 0.82 in accord with the evidence in Table 6.1.

Supplementary information:

- molar volumes O_2, N_2, CO_2, CH_4, H_2 = 22.41 L
- heats of combustion: CH_4 = 39.54 kJ/L; H_2 = 12.76 kJ/L

The values below follow from Table 6.1:

- Protein (g) = 6.25 × N (g)
- C in protein (g) = 3.25 × N (g); and
- Fat (g) = 1.304 × C (g)

Using the constants given in Table 6.1, the energy balance (E) can be calculated from the C balance (in g) and N balance (in g), as follows:

$$E\,(kJ) = 51.831\,C - 19.40\,N$$

Heat production (H) determined from measurement of gas (O_2, CO_2 and CH_4) exchange

Heat production (kJ) is calculated from the net volume of oxygen consumed at STP, the volumes of carbon dioxide and methane produced at STP, and the mass of urine nitrogen (g). It is expressed as:

$$H\,(kJ) = 16.18\,O + 5.02\,C - 2.17\,M - 5.99\,N \qquad (1)$$

N.B. Including urinary CO_2 in free and carbonate forms.

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The flow and pressure of CO_2 and O_2 are determined in the incoming air (approx. 21% O_2, 0.039% CO_2, by volume) and outgoing air (say 17% and 3.9%, respectively, by volume),

$$\text{e.g. } V_{(e)}(0.039 - 0.00039)/V_{(e)}(0.21 - 0.17) = \text{RQ } 0.97$$

where $V_{(e)}$ = ventilation rate. The carbon content of feed, faeces and urine is determined. The contribution of urine to the C balance is only about 1/10 of that from faeces, but the coefficient of variation (%) for urine is 10 times larger than that for faeces (Thorbeck *et al.* 1965). [For the determination of the heat of combustion of urine it is necessary to use urine acidified with H_2SO_4, not in large excess, in order to avoid loss of carbon, that must be accounted for in the calculation of the total production of CO_2 (Nijkamp 1965). HCl should not be used.]

Heat production determined from measurement of O_2 exchange only

In experiments in which O_2 consumption only is determined, heat production can be estimated with sufficient accuracy on the assumption that the heat equivalent of O_2 is 20 kJ/L. This heat equivalent implies that the RQ is 0.75 on the Brouwer (1965a,b) scale. The value for heat equivalent when the RQ is 0.81 is 20.24, but rounding to 20 is considered admissible.

$$\text{H (kJ)} = 20\ \text{O (L)} \qquad (2)$$

Relevance of oxygen consumption to energy expenditure

O_2 consumption has been widely used to estimate energy requirements during exercise and a curvilinear relationship exists between it and speed (Pagan & Hintz 1986b; Eaton *et al.* 1995a). A strong relationship exists between O_2 consumption and heart rate (HR), which is more easily measured than O_2 consumption:

$$O_2 \text{ utilization (mL } O_2/\text{kg BW.min)} = 0.0019\ HR^{2.0653} \quad (R^2 = 0.9)$$

where HR is given in beats per min, although a closer relationship exists with per cent of maximum HR (Coenen 2005b). Measures of HR are more widely applicable than speed, as for some activities speed is irrelevant. Treadmills are frequently used to estimate energy consumption, but they do not reflect the effects of weight carried (Thornton *et al.* 1987), the track or turf, ground reaction forces, wind resistance, level of training, type of exercise, weather, and possibly psychological factors. O_2 utilization is increased 25–30% during exercise on an inclined treadmill. A slope of 10% surprisingly yielded consumption values 100–150% greater than those on a flat surface (Eaton *et al.* 1995a), as distinct from the effects of rising, or falling, ground, which has a major impact. Moreover, O_2 use excludes the anaerobic component (Eaton *et al.* 1995b).

CONCENTRATES AND ROUGHAGES

Horses should receive at least some long roughage, such as fresh grass, hay or silage. Although a proportion of this can be replaced by succulent green or root vegetables and soaked sugar-beet pulp, hay or grass can comprise the total ration and usually forms at least half of it. The concentrate portion of feed therefore varies from nothing to 50%, and only in exceptional circumstances, or in the hands of experienced feeders, should it rise to form 75% of the daily dry feed.

Concentrated feeds, such as cereals, cereal by-products and oilseed meals, are traditionally fed by the bowl, i.e. by volume, whereas the energy and nutrients provided are much more closely associated with their weight. Feeding containers should be calibrated to show the volume occupied by a unit weight of each type of feed. Table 5.5 gives average conversion values for cereals, although their bushel weight may vary from season to season. The energy content per kg of each type of concentrate also differs. Ideally, feed bowls should be calibrated to indicate the volume, giving multiples of 2 MJ DE (or 1 MJ NE), for each type in use.

FEED ENERGY

The gross energy (GE) of a feed is the heat evolved when it is subjected to complete combustion in an atmosphere of O_2. Obviously, all GE, measured as heat, is not available to the animal because a portion of the feed remains undigested and is voided in the faeces. In addition, a relatively unknown quantity is lost from the horse as the gases CH_4 and H_2, through the anus, but also by absorption and exhalation. Of the products of digestion and fermentation that are absorbed, a proportion of the amino acids is deaminated and the nitrogen incorporated in urea. Much of this is excreted in the urine.

The GE of a feed, less the energy content of the faeces attributable to it, is the digestible energy (DE) (Sutton *et al.* 1977a). Subtracting the energy content of combustible waste gases, and urine excreted, leaves the metabolizable energy (ME). This is the residue of feed energy that is available to the body for its various processes of tissue repair, the functioning of organs, the physical work of

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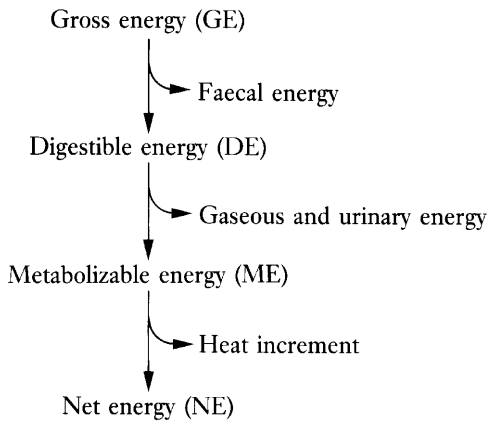


Figure 6.5 Partition of dietary energy.

skeletal muscles, growth and milk production. The efficiency of ME utilization depends not only on the chemical form of the nutrients derived from the diet but also on which of these functions is being performed. The efficiency is measured either as the amount of useful product or the quantity of waste heat dissipated. The ME less this heat increment attributable to the feed is the net energy (NE). (Heat increment is the heat loss of a nourished animal in excess of that lost by a fasting animal.) The scheme is summarized in Figure 6.5. NE is used for maintenance, growth, work, reproduction, etc.

When energy demands are great, concentrated feeds, such as cereal grains, must form part of the diet, simply because the horse can consume larger quantities of DM daily when cereals are included and they contain more ME per kg DM. Conversely, an idle horse has relatively low energy requirements, yet its appetite should be satisfied. As the horse can consume daily lesser quantities by weight of bulky fibrous feeds than of concentrates, its appetite is more likely to be satisfied when idle by the lower intakes of energy provided by bulky fibrous feeds. The idle horse is a stabled, voluntarily active, non-working animal, described as having energy requirements for maintenance only, i.e. a zero change in BW, or, more accurately, a zero change in body energy content.

NRC (2007) assessment of maintenance needs of adult horses

The NRC (2007) has estimated that energy requirements for maintenance are linearly related to BW between weights of 200 and 800 kg (see Table 6.6 below). Incorporating the efficiency of DE use for ME, the minimum daily DE requirement for maintenance (DEm) ranged from 25.7 to 35.1 kcal/kg BW (108–147 kJ/kg), compared with a mean value for confined horses of 30.3 kcal DE/kg BW (127 kJ), and for horses turned out for several hours daily, 33.3 kcal DE/kg BW (139 kJ)). For non-breeding stallions, young adult horses, or adult horses of a nervous temperament it is 36.3 kcal DE/kg BW (152 kJ) daily, but it is higher for draught horses, at 30.3 kcal/kg BW (127 kJ), while for breeding stallions in heavy use the daily DE requirement is estimated to be 20% higher than maintenance, i.e. 43.5 kcal/kg BW (182 kJ). The range is caused by several factors: greater heat production with a hay diet than with one based on concentrates, a lower requirement for older than for younger horses (Martin-Rosset & Vermorel 1991), a lower requirement for ponies than for horses per unit of metabolic size (BW 0.75) and variation with body composition (lean tissue having a greater O_2 consumption rate than fat tissue). The NRC has therefore categorized the needs for minimum, average and elevated daily requirements as: 127, 139 and 152 kJ/kg BW, respectively. Moreover, environmental temperature outside the thermoneutral zone (TNZ) increases metabolic rate. Where the environmental temperature is below the lower critical temperature (LCT) the DE intake of adult acclimatized horses should be raised by about 2% for each degree Celsius below the LCT (McBride *et al.* 1985). Cymbaluk & Christison (1990) suggested that the maintenance requirement of growing horses was increased by 33% in housing at −15°C and by more than 50% at −25°C. Precision is impossible, because climatic conditions, such as precipitation and wind, also affect metabolic rate. Effects on maintenance needs caused by ambient conditions above the upper critical temperature have not been quantified. The relationship between BW and the DE requirements for maintenance is depicted in Figure 6.6.

Texas workers (Potter *et al.* 1987) found that the energy requirement of heavy (675–839 kg) Belgian and Percheron horses was 10–20% lower than predicted by Pagan & Hintz (1986a). The difference was attributed to the lower activity of heavy horses and the slower rates of acceleration and deceleration during voluntary work. This situation can now be accommodated within the 'minimum' maintenance needs (NRC 2007, Figure 6.6).

Energy needs for working horses

In order to estimate the dietary energy needs for exercise, independent of maintenance, in terms of DE, the NRC (2007) has assumed a mean efficiency of DE conversion to NE of 30–40%, with a wider range of 20–50% (Figure 6.7). This extended range assumes a constant maintenance requirement. However, maintenance requirements increase

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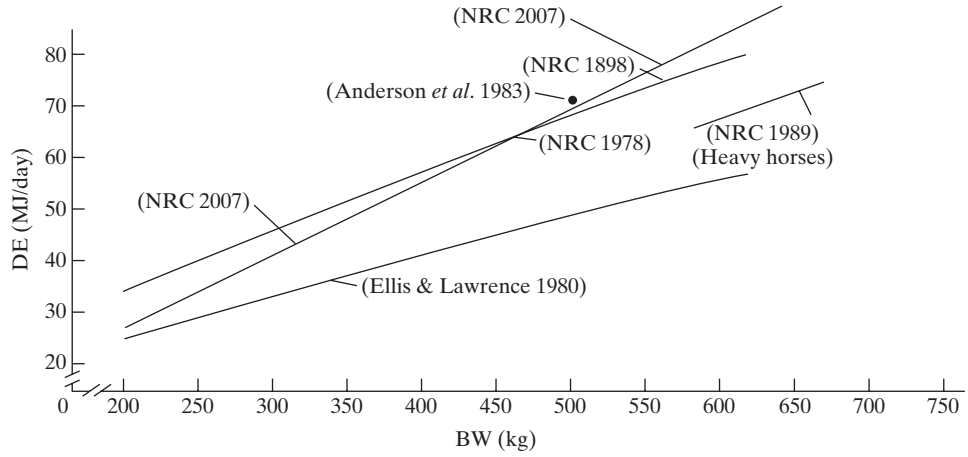


Figure 6.6 Approximate relationship of kg BW to daily DE requirements for maintenance according to five sources: for weights 600 kg BW or less, DE (MJ/day) = 5.9 + 0.13 BW (NRC 1989), for weight >600 kg BW, DE (MJ/day) = 7.61 + 0.1602 BW − 0.000063 $(BW)^2$ (NRC 1989); DE (kJ/day) = 649 $BW^{0.75} \equiv 0.649\ BW^{0.75}$ MJ/day (NRC 1978); DE (kJ/day) = 465 $BW^{0.75} \equiv 0.465\ BW^{0.75}$ MJ/day (Ellis & Lawrence 1980); for average voluntary activity, BW, 200–800 kg, DE (MJ/day) = 139 kJ.BW (NRC 2007).

with increasing activity. When these increases are incorporated the narrower range of DE to NE conversion (30–40%) is appropriate and maintenance needs are: 139 kJ/kg BW (33.3 kcal/kg BW) for light to moderate work and 152 kJ/ kg BW (36.3 kcal/kg BW) for heavy and very intense work (NRC 2007). For example, for 1 h after short-term, moderately intense exercise, O_2 utilization was elevated 11% above the pre-exercise value (Dunn *et al.* 1991). Cutting horses, working in a hot environment, expend 10–20% more energy than would be predicted (Webb *et al.* 1990), when high fat diets have a role (Potter *et al.* 1989).

The energy costs of exercise were measured as the amount of CO_2 produced during exercise in several breeds of 433–520 kg BW (Pagan & Hintz 1986a,b):

$$\text{NE kg}^{-1}\text{.BW h}^{-1} = e^{(3.02+0.0065Y)},\ \text{s.d.} = 0.105;\ R^2 = 0.92$$

where Y = activity (speed) in m/min, e = exponential (2.7183).

The equation corrected for basal maintenance and converted to DE (NE = 0.57 DE) is then

$$\text{DE, in kJ/kg (BW + rider + tack) per h} = 7.34\left(e^{(3.02+0.0065Y)} - 13.920\right) \times 0.06^{*} \quad (3)$$

*if the measurement is per min then this multiplier is 0.001.

This method ignores the anaerobic component and so underestimates the true energy utilization. Moreover, maintenance needs are elevated during full training when horses tend to engage in compulsive behaviour, such as cribbing, weaving, or stall kicking (Luescher *et al.* 1998) and during transportation over long distances (Doherty *et al.* 1997). The mean daily DM intake of race horses in training, in consequence, greatly exceeds 2.2% BW, according to the author's observations and those of others (Gallagher *et al.* 1992a.b).

Although energy consumption during exercise is related to O_2 consumption which in turn is related to percentage of maximum heart rate, anaerobic metabolism is unaccounted for. Moreover, speed is only one factor in energy needs of exercise and factors such as surface conditions, rider weight, differences between horses, and the elevated metabolic rate of post-exercise recovery, influence requirements. With a NE/DE ranging from 0.3–0.4 for exercise, the NRC (2007) has therefore averaged dietary requirements of working horses over time in four categories of daily DE requirements (see Tables 2.2, 6.6), derived from the following equations for maintenance plus work:

Light work:

$$\text{DE (Mcal/day)} = 1.2\ (0.0333\ \text{BW}) = 0.03996\ \text{BW}$$
$$\text{DE (MJ/day)} = 5.0\ (0.0333\ \text{BW}) = 0.16719\ \text{BW} \quad (4a–d)$$

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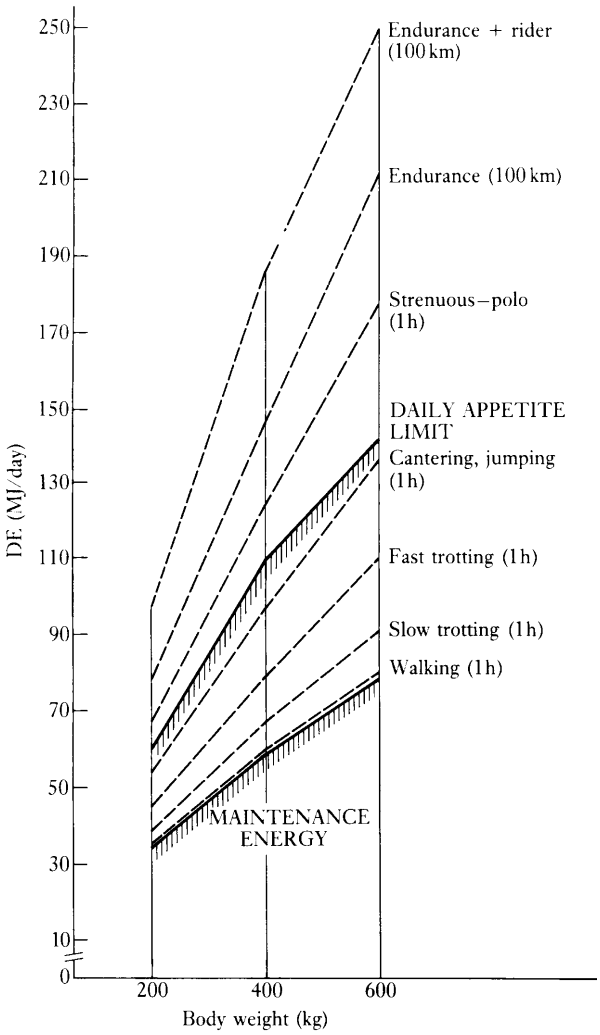


Figure 6.7 DE demands of daily maintenance and work at a constant elevation in relation to appetite of horses of three body weights. (Effect of a 67 kg rider on 400 and 600 kg horses and 33 kg rider on 200 kg horse given for endurance rides only.)

Moderate work:

$$\text{DE (Mcal/day)} = 1.4\,(0.0333\ \text{BW}) = 0.04662\ \text{BW}$$
$$\text{DE (MJ/day)} = 5.9\,(0.0333\ \text{BW}) = 0.19506\ \text{BW}$$

Heavy work:

$$\text{DE (Mcal/day)} = 1.6\,(0.0333\ \text{BW}) = 0.05328\ \text{BW}$$
$$\text{DE (MJ/day)} = 6.7\,(0.0333\ \text{BW}) = 0.22292\ \text{BW}$$

Very heavy work:

$$\text{DE (Mcal/day)} = 1.9\,(0.0363\ \text{BW}) = 0.06897\ \text{BW}$$
$$\text{DE (MJ/day)} = 7.9\,(0.0363\ \text{BW}) = 0.28857\ \text{BW}.$$

NRC (2007) estimates of the maintenance needs of growing horses

The daily maintenance requirements of growing horses, determined by extrapolation of growth data to zero gain, were found to be 158 kJ DE/kg BW and 148 kJ DE/kg BW for limit- and for *ad libitum*-fed horses, respectively (Cymbaluk *et al.* 1989a). (In the same study 24–83 g BW gain/MJ DE was achieved above maintenance.) This is about one-quarter the efficiency of poultry and pigs. Individual horses differ and some will eventually become fat when subjected to a regime under which others will lose condition.

More recently, the NRC (2007) concluded that the DE_m for growing horses is estimated using the following equations:

$$\text{DE in kJ.kg}^{-1}\text{BW.day}^{-1} = 236x^{-0.145}\ (R^2 = 0.99), \quad (5a)$$

and for gain in weight:

$$\text{DE in MJ} = (8.33 + 5.06x - [0.088x^2])\ \text{kg daily gain} \quad (5b)$$

where x = age in months.

The NRC (2007) combines these two equations to give the daily DE needs of growing horses:

$$\text{DE in MJ/day} = \text{BW}(236x^{-0.145}) + \text{ADG}(8.33 + 5.06x - 0.088x^2) \quad (5c)$$

(see also Tables 2.2 and 6.6).

Heat production and efficiency of ME use

An inactive horse at the maintenance level of energy intake and expenditure does essentially no work on its surroundings, so that NE expended in maintenance is ultimately degraded to heat:

$$ME_m = NE + HI = \text{heat production at maintenance} \quad (6)$$

where HI is heat increment, or waste heat. The temperature of the horse's body is normally greater than that of the surroundings, to which heat is continuously being lost. This is an expression of that situation. Exposure to a cold or a wet and windy climate accelerates metabolic rate so that the rate of heat production keeps pace with the rate of heat loss in order to maintain a steady body

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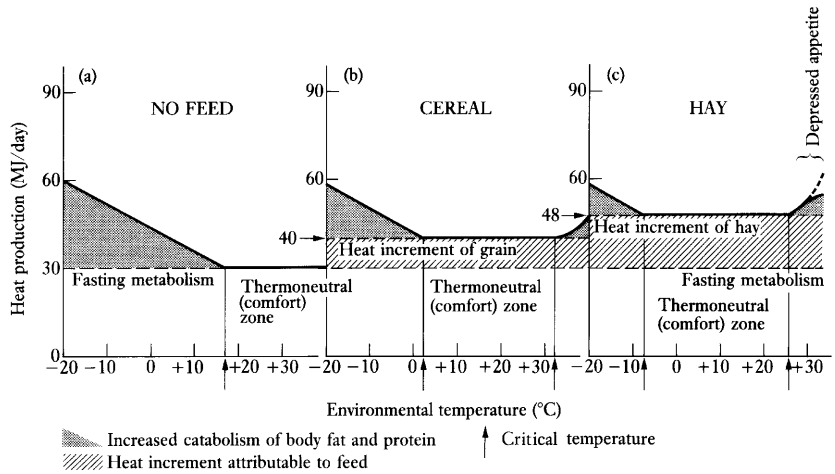


Figure 6.8 Interactions among the heat increment of feeds, environmental temperature, heat production and critical temperature. Data are approximate; they assume minimal air movement and are not based on direct experimentation. (a) Fasted horse (also with slightly less subcutaneous fat); (b) horse fed 3.5–4 kg grain providing 40 MJ ME and 30 MJ NE (maintenance level); (c) horse fed 5–6 kg hay providing 48 MJ of ME and 30 MJ of NE (maintenance level).

temperature, that is, the energy requirement for maintenance rises. Conversely, in hot climates, where the environmental temperature is higher than that of the horse, the heat produced must still be dissipated. This is done primarily by evaporation of sweat and of water from the lungs, but also by a rise in body temperature, inducing a physiological stress. Thus, in one environment heat production is a boon and in the other a hindrance. Cutting horses, working in a hot environment, expend 10–20% more energy (Hiney & Potter 1996) than would be predicted by the equations of Pagan & Hintz (1986b). Their rectal temperatures are often 41°C, and as metabolic rate is a function of body temperature, this is a cause of greater expenditure.

Can heat production be manipulated to the horse's advantage? HI is a measure of the efficiency of utilization of the ME of feed and it is known to vary between types of feed. If the NE available represents 80% of the ME (NE/ME = 0.8), then the remaining 20% is HI. When feeds are selected, their difference in HI should ideally be considered in the context of the climate and the purpose for which the horse is kept. Allowance for these differences is a basis of the justification for the French Institut National de la Recherche Agronomique (INRA) NE system (see The NE system, INRA 1984, updated 1990, this chapter). The data in Table 6.2 show that, in growing/fattening animals, 68% of the ME of meadow hay and 40% of the ME of mixed proteins are lost as waste heat, whereas only 23% of barley grain and 15% of fat ME are similarly lost in fattening. The so-called heating effect of cereals and other concentrates reflects a more rapid rise in blood glucose and metabolic rate after a large meal and the associated feeling of vigour in 'hot-blooded' breeds (see also Calming effects of dietary fats, Chapters 5 and 9). In Figure 6.8 the interactions among the HI of feeds, environmental temperature, and production of body heat and critical temperature are depicted.

Estimates of the likely efficiency of ME utilization by the horse are given in Table 6.1 and Figure 6.9. The efficiency values (k) in Table 6.2 subtracted from those in Table 6.1 show the proportion of energy lost as waste heat when the feed is used for maintenance or for fat deposition. Thus, 30% of the energy of meadow hay would be lost as waste heat at maintenance, whereas only 15% of the ME of barley would be similarly lost (N.B. the utilized energy is ultimately degraded to heat, but more hay would be required for maintenance). During winter, ample hay is a more appropriate feed than in the summer, or than barley, as the greater HI of hay contributes to the maintenance of body temperature. The partition of the GE of four feeds is described in the histograms in Figure 6.9. It should be clear that k represents, in the main, the efficiency of glycogen and of depot fat formation. Efficiency of utilization of these sources by muscles is approximately 0.35–0.45.

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Table 6.2 Estimated efficiency of utilization by the horse of ME (NE/ME), or *k*, for various energy sources.

	For maintenance* (k_m)	For fat deposition (k_f)
Mixed proteins	0.70	0.60
Meadow hay	0.70	0.32
Lucerne hay	0.82	0.58
Oats	0.83	0.68
Barley	0.85	0.77
Fat	0.97	0.85

*These values are higher than those for fattening mainly because the use of these nutrients for that purpose spares the breakdown of body fat.

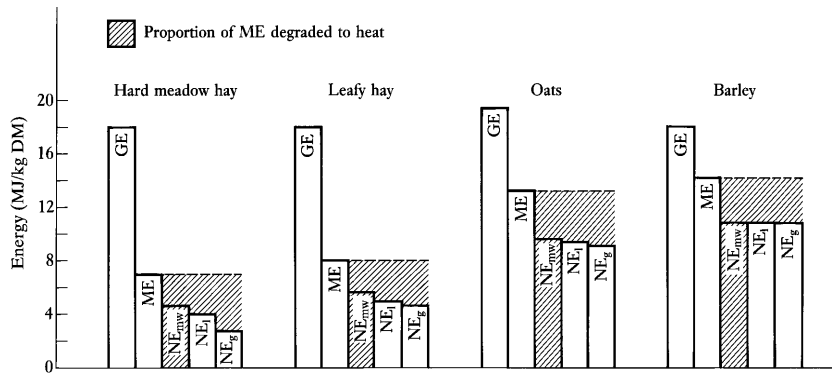


Figure 6.9 Estimated average utilization efficiency of gross energy (GE) of feeds for several production purposes: NE_{mw}, NE for maintenance, or for work, which can in practice be approximately the same; NE_l, NE for lactation; NE_g, NE for growth. Efficiency of utilization of GE as a source of ME = ME/GE = *q*; efficiency of utilization of ME as a source of NE = NE/ME = *k*; NE/GE = *qk*

The concept of work measured for flat racing, etc., is illusory, as the true efficiency can be measured only as a difference in energy expended between exercise on the level and that on a gradient, not on an inclined moving belt. When a horse moves up a gradient, work is done against the force of gravity, whereas on an inclined moving belt the horse does not rise. However, it is known that, for other reasons, a horse expends more energy running on an inclined, than on a horizontal, belt, moving at the same speed. Robert *et al.* (2000) showed that the electromyographic activity of hind-limb muscles during a trot increased with increasing slope of the treadmill at inclinations of 0%, 3% and 6%, implying an increasing workload.

The *k* value includes an allowance for the energy costs of both ingestion and digestion, that is the energy expended in eating, digesting and fermenting feed, in addition to differences amongst nutrients in the systemic efficiency of their metabolism in ATP, tissue, milk etc. synthesis (see Appendix C for maintenance k_m calculation). Note: nearly 2.5 times as much hard hay is required for maintenance as would be required of barley so that nearly 25% more heat is produced by hay at maintenance.

The ribs of both breeding and working horses in optimum condition cannot be seen, but can be felt with little fat between the skin and ribs. Acclimatization to cold weather does not necessitate excessive fat deposition but should allow sufficient time for the coat to grow. Horses should be provided with a shelter protecting them from rain, snow and the worst of the wind. Three sides and a roof provide sufficient protection in all seasons for properly fed adult animals. (The author has successfully held

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Table 6.3 DE demands for maintenance plus work on a slope of 90° at endurance rates (135 heart beats/min, 155 m/min).[1]

Body weight (kg)[2]	400	500	600
Distance travelled (km)	DE per day (MJ)		
1	68	79	90
2	76	88	100
4	89	103	117
6	99	114	127
8	107	121	133
Approximate appetite	108–113	125–130	140–145

[1]Based upon a quadratic equation relating energy requirements to body weight and work in Quarter Horses (Anderson *et al.* 1981, 1983).

[2]Average body weight 503 kg.

horses in this way at temperatures lower than −30°C.) A long hair coat, if dry, and a modicum of subcutaneous fat are an excellent insulation for horses given an ample roughage diet, so that the rate of waste heat production without shivering, a function of $(1 - k)$, equates with the rate of heat loss. In the spring when horses are brought in, daily grooming and 60–110 g oil added to the ration each day should accelerate the shedding of the winter coat.

Joyce & Blaxter (1965) determined the heat loss to the environment of sheep, measuring the insulation properties of the skin and fleece. The external insulation (I_E) of sheep is the resistance to heat flow from the skin surface to the environment and that resistance depends particularly on the temperature difference between the skin and the air and fleece length. External insulation of sheep was shown to be linearly related to fleece length (Joyce & Blaxter 1965). The situation in the horse is similar. Booth *et al.* (1998) took two mature Shetland pony stallions of 178 and 200 kg BW with full winter coat and, using indirect calorimetry, determined whether soaking them with water at 5.26°C, at an ambient temperature of 2.0–9.5°C, caused a change in the rate of heat production. They were given meadow hay at maintenance levels. Skin temperature decreased, but not rectal temperature, and heat production was not increased over three hours (see also Chapter 10).

Exercise needs (also Table 6.6)

Table 6.3 indicates the energy needs for maintenance plus endurance work (Anderson *et al.* 1983). Figure 6.7 gives the DE requirements of horses of various weights at maintenance and when engaged in work of a range of intensities, strenuous work causing a large increase in energy demand.

Weight-carrying ability

Height, cannon bone circumference, and loin width are indicators of weight-carrying ability in light horses (Powell *et al.* 2008). When the horses carried 15, 20, 25, or 30% of their body weight, plasma lactate concentrations immediately and 10 minutes after exercise differed when horses carried the 30% load compared with 15, 20, and 25%. A greater change in muscle soreness and muscle tightness occurred when carrying 25% or 30% of their weight. Loin width and cannon bone circumference were negatively correlated with changes in muscle soreness and tightness scores.

Partition of feed energy

Visual examination of feed reveals nothing about its ME content, but the feed can be weighed. The gross energy (GE) content of most horse feeds is just over 18 MJ/kg DM, although not for feeds containing much more than 80 g ash/kg or 35 g oil/kg. For example, oats on average contain 45 g oil and 19.4 MJ GE/kg DM. Figure 6.9 shows how the GE of samples of four different feeds might be utilized for growth or for hard extended work and the data give a revealing and objective comparison of roughages with cereals. The coefficient q represents the approximate efficiency by which the GE of each feed is utilized as a source of ME while the coefficient k represents the efficiency by which this ME is utilized for the functions of maintenance, growth etc. Therefore, $q \times k =$ NE/GE, or the overall efficiency of utilization of the 18 MJ. Note that $q \times k$ of hard meadow hay for growth (mainly fat deposition) is 0.12, whereas the equivalent for barley is 0.59 (4.9×0.12). The NE in hard meadow hay for growth, and probably for extended work, is only a quarter of that found in the two cereals, despite the similarity of their GEs. In energy terms, that is, MJ/kg feed, the losses in the utilization of ME from hay and cereals are not very different (Figure 6.9), but their k values are (Figure 6.10), because the ME values (MJ/kg) differ widely.

The efficiency by which ME is used depends on the type of metabolite. Vermorel *et al.* (1991) reported that the efficiency of conversion of DE to ME was 90% for a mixed diet and 87% for a hay diet while the efficiency of

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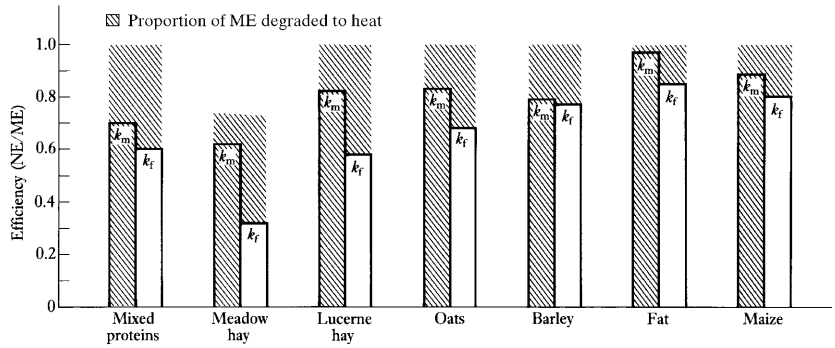


Figure 6.10 Estimated efficiency of utilization of ME (NE/ME), or *k*, for various energy sources: k_m = NE/ME for maintenance; k_f = NE/ME for fat deposition (later growth). (Maintenance values are higher than those for fattening, mainly because the breakdown of body fat is spared by the use of these feeds for maintenance.)

converting ME to NE is estimated by Vermorel & Martin-Rosset (1997) to be 85% for glucose, 80% for long chain fatty acids, 70% for amino acids and 63–68% for VFAs. The INRA system (see elsewhere this chapter) does not assign different NE values to feeds based on their efficiency of use for different physiological functions. Vermorel *et al.* (1997a) estimated that horses consuming a diet composed primarily of hay meet more than 80% of their energy needs from VFAs, while in forage-fed ponies it has been suggested (Simmons & Ford 1991) that 50–60% of circulating glucose originates from absorbed propionate.

It is recognized that roughage is required by horses and ponies, particularly in a long form, in order to maintain general metabolic health. However, are there lessons to be learnt from the above calculations? First, poor-quality roughage can be expensive if it is a major part of the ration of growing or hard-working animals. Second, hard-working animals can lose condition if poor-quality roughage forms a major portion of their diet. Finally, idle horses put on unwanted fat if given too much cereal.

The data available on the ME content of horse feeds and on their NE for various functions is limited. The large difference in the efficiency of DE utilization for productive activity between roughages and concentrates led Martin-Rosset and colleagues (1994) to develop the NE system (see elsewhere in this chapter). Estimated values for maintenance are provided by INRA and selected data are given in Appendix C, expressed as horse feed units (UFC). For proper use of the DE system it is necessary to follow certain rules (see 'Ration formulation using the DE and NE systems', this chapter) and to use the DE data provided in Appendix C.

DIGESTIBLE ENERGY, PROTEIN AND MINERAL REQUIREMENTS BASED ON NRC (2007) RECOMMENDATIONS

Reproduction and lactation

The dietary requirements of the breeding mare can be arbitrarily divided into those for: (1) the first eight months of gestation; (2) the last three months of gestation; and (3) lactation (which may coincide with 0–4 months of gestation). Gestation length for TBs is in the range 335–345 days and for other breeds 322–345 days. Lactations of 110–150 days are typical of many husbandry systems, although the nonpregnant mare would produce milk for much longer if given the opportunity.

Energy

The nutrient drain incurred during gestation to sustain normal foetal and placental growth is much less than that for lactation. The approximate energy contents of the foetus and the other products of conception at term, compared with the energy content of the mare's milk over a four-month lactation, are given in Table 6.4.

The first eight months of gestation have no practical impact on nutrient needs – that is, they do not raise requirements above those of the barren mare, nor do they increase the already high requirements of the lactating mare. After weaning, the mare's energy requirements approximate those of maintenance until eight months of gestation have

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Table 6.4 Approximate energy contents of the foetus and other products of conception at term compared with the energy content of mare's milk over a four-month lactation.

Mare's weight (kg)	200	400	500	600
Products of conception at term (MJ)	110	200	240	270
Lactation of 17 weeks (MJ)	1700	2840	3400	3900

been completed. Most of the foetal growth occurs during the last 90 days of gestation. Placental weight, without fluids, is about 4 kg for a 500 kg mare (Allen *et al.* 2002) and the uterine tissue increases in weight by about 4 kg during gestation, so that the rate of total non-foetal accretion (uterus plus placenta) in a 500 kg mare is estimated to be about 45 g/day (0.09 g/kg maternal BW/day) from day 150 to day 330 (NRC 2007). This tissue is metabolically very active and so has a high maintenance requirement per unit weight (279 kJ/kg tissue weight; Fowden *et al.* 2000a,b), assuming the efficiency of DE use for tissue deposition during pregnancy is 60% The NRC (2007) calculated the percentage of birth weight accumulated by the foetus during days 150–330, where birth weight was estimated to be 9.7% of dam weight:

$$\text{Foetal weight, as a \% of birth weight} = 10^{-7}\,x^{3.5512} \quad (7)$$
$$\left(R^2 = 0.929\right)$$

where x = days of gestation. The weight of a mare in moderate body condition would be expected to increase by 12–15% during a gestation of mean length 345 days.

The pregnant mare is estimated by the NRC (2007) to have a DE requirement 5–8% above that estimated by NRC (1989). For a 500-kg mare, in BCS 5 at conception and retaining its condition during gestation, the requirements would rise from 143 kJ/kg BW at the 5th month to 165 kJ/kg BW at the 11th month, implying increments above maintenance of 11%, 13% and 20% in the 9th, 10th and 11th months, respectively, of gestation. For the heavy mare the estimates are similar. (NRC 2007 concluded that for mature horses of 480–580 kg BW each unit of BCS increase requires 16–20 kg of weight gain. This requires 67–100 MJ DE/kg gain, or 1510 MJ DE for an increase of one unit of BCS.)

The DE required daily to meet the needs of the mare are given in Table 6.5 for comparison with the much greater demands of milk production. The NRC (2007) estimates are given in Table 6.6.

Table 6.5 DE required daily by the mare to meet the needs of foetal growth and lactation, excluding maintenance requirements for energy of the mare (based on data from NRC, 2007).

Mare's weight* (kg)	400	500
Products of conception at term (MJ)**	6.4	7.7
Average milk production (MJ)**	44.3	54.9

*The mare's weight should increase by 15% during gestation and her maintenance requirements rise proportionately.

**Arithmetic averages, only for comparisons between pregnancy and lactation. Reprinted with permission from *Nutrient Requirements of Horses*, (2007) by the National Academy of Sciences, Courtesy of the National Academies Press, Washington, D.C.

As the foetus occupies an increasing proportion of the mare's abdominal cavity, her capacity for bulky feed declines during the period in which nutrient requirements increase. This could correspond to an increase in the quality of grazing (see Chapter 10) but, where mares are given hay and concentrates, the quality of the forage should be improved during the last three months of gestation. Note that, if the mare is lactating during early pregnancy, her requirements then will exceed those of the last 3 months. However, the values are averages and the amounts of feed and therefore the DE given to individual pregnant mares should be adjusted to avoid either obesity or poor condition (see also Chapter 7).

During the 11th month, the DE requirement is equivalent to a 0.33 : 0.66 mixture of oats and hay of nearly 10 kg daily. Part of the demand in the 11th month is to sustain udder development. Feeding studies have not established whether birth weight of the foal is generally influenced by deviations from those rates. One investigation (Goater *et al.* 1981) demonstrated an increase of both 1.5 kg in birth weight and 0.24 kg in daily weight gain during the first 30 days of life as a result of providing the mare with 120% of the NRC (1978) gestation recommendations. Other experiments, in which Quarter Horse and TB mares were restricted to 55% and Arabian mares to 85% of the recommendations, led to weight losses by the pregnant mares without affecting the birth weight of the foal, in comparison with mares receiving the recommended rates. Clearly, healthy mares possess the capacity to adapt without the foal incurring any significant handicap.

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Table 6.6 DE requirements of growing, working and breeding horses, based upon NRC (2007) recommendations (in Mcal/100 kg BW per day and MJ/100 kg BW per day, except for ADG). N.B. (1) These requirements assume positive linearity with BW. (2) The increasing requirement per kg ADG with increasing age is largely accounted for by increasing fat content and a reduction in moisture content with increasing age. (3) The DE requirements will vary somewhat as between feed sources, owing to differences in metabolizability and net use of digestion products.

	Mcal	MJ
Maintenance, foals, 4–5 months*	4.6	19.2
Maintenance, foals, 6–11 months *	4.1	17.2
Maintenance, foals, 12–18 months *	3.9	16.3
Growth (g/kg ADG/day, 4–5 months)[A]	7.1	29.7
Growth (g/kg ADG/day, 6–11 months)[A]	10.5	43.9
Growth (g/kg ADG/day, 12–18 months)[A]	15.0	62.8
Maintenance, adults, minimum*	3.04	12.7
Maintenance, adults, average*	3.34	14.0
Maintenance, adults, maximum *	3.64	15.2
Maintenance and light exercise[A]	4.00	16.7
Maintenance and moderate exercise[A]	4.66	19.5
Maintenance and heavy exercise[A]	5.32	22.3
Maintenance and very heavy exercise[A]	6.90	28.9
Gestation, months 1–6[C]	3.40	14.2
Gestation, months 7–8[C]	3.53	14.8
Gestation, months 9–11[C]	3.71	15.5
Lactation, foaling to 3 months[BC]	6.30	26.4
Lactation, 4–5 months[BC]	5.78	24.2
Lactation, >5 months[BC]	5.44	22.8
Stallion, nonbreeding	3.64	15.2
Stallion, breeding	4.36	18.2

*Maintenance requirement is the dietary energy needed to prevent a change in total energy contained in the non-pregnant horse.

[A]Where appropriate growth requirement should be added to maintenance requirement, including yearlings in training.

[B]Requirements assume milk yield is directly and linearly proportional to BW.

[C]Including maintenance.

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Protein and minerals

The most critical nutrients for breeding mares given traditional feeds are protein, Ca and P. Mares kept during the last 90 days of gestation entirely on reasonably good-quality pasture or high-quality conserved forage containing some 30–40% leafy clover, lucerne or sainfoin require no other source of Ca, and, if the forage contains 10% protein per unit of dry matter, no supplementary protein.

An increase in the physiological demand for Ca leads to a decrease in serum total and ionized Ca concentrations and a stimulation of parathormone secretion. This occurs in periparturient mares during mammary Ca secretion when serum total Ca has been shown to decrease from 3.1 to 2.7 mmol/L (12.5–11 mg/dL) (Martin *et al.* 1996b). So the dietary requirement of mares averaging 510 kg BW in late pregnancy is closer to 5.5 g Ca/kg dietary DM (45 g Ca/day) than to 3.5 g Ca/kg DM when the Ca was derived from bluegrass–clover pasture and concentrates, a third of the daily Ca being derived from the pasture (cf. NRC 2007 values of 37 g Ca and 27 g P per day, see Table

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3.3). The P requirements would be met by good pasture (see Table 10.2), but as both grass and legume hays given to horses in the UK normally contain less than 2 g P/kg (0.2%), a supplement of dicalcium phosphate, or wheat bran will be required. The discrepancy would be provided daily by a supplement of 60 g dicalcium phosphate or 1.5 kg bran for horses and 40 g or 1 kg, respectively, for ponies.

If the hay contains 5% of protein (see Table 6.23) and constitutes 65% decreasing to 50% of the diet during gestation, then a concentrate source of protein will be required to supplement a cereal (Figure 6.11c). Appendix A shows the type of calculation needed.

Abundant good-quality pasture will meet the energy, protein, Ca and P needs of lactation, even though the minimum dietary protein requirement will have risen to127 g/kg DM (12 kg DM/day), during months 0–3 of lactation (see Table 2.3). Responses in milk yield have been obtained from Quarter Horse mares given mixed feeds containing up to 170 g protein/kg. However, grass and clover proteins are of high quality and it is unlikely that an economic response would be obtained by raising the protein level of the spring-grazing diet. If the stocking density is high, or good-quality pasture is otherwise moderately limited, supplementation can be provided by lower protein pony nuts (see Table 7.1), or a mixture of these and cereals. If pasture is more scarce, a mix containing 16% protein should be given to lactating mares. Any conserved forage provided should be leafy hay containing a mixture of clover and grass, or well-conserved haylage. Typically in the UK, grass hays of only moderate quality are fed when grazing is limited. These contain only 40–80 g crude protein/kg and so do not meet immediate needs (Figure 6.11d).

It is unlikely that one could overfeed lactating mares with roughage, except that large quantities of poor roughage might limit their capacity for concentrates, leading to a decrease in milk yield. When typical grass hays are given, a satisfactory milk yield is obtained only if at least 50% of the dry feed is provided as a 16% protein mix. This mix can be based on oats or barley and soya-bean meal, or an equivalent proprietary protein concentrate containing 440 g protein/kg. The reason why a 16% protein mix is sufficient for lactating horses, as well as for late pregnancy, despite a higher protein requirement in lactation, is that the mix forms a higher proportion of the lactation diet to meet the greater energy needs (Figures 6.11c,d). The amino acid composition of milk is given in Chapter 7, under 'Lactation', from which Wickens *et al.* (2002) calculated amino acid requirements, assuming a digestibility of 65%. For a typical lactation these requirements are approximately three times those of maintenance (see Table 2.3). Milk protein is richer in leucine, isoleucine, threonine and valine than skeletal muscle protein; and the authors calculate the maintenance requirement for each amino acid from muscle composition. However, these calculations assume a similar availability and muscle turn-over efficiencies for each amino acid, which are probably unjustified assumptions.

A grass hay, cereal, soya diet must be supplemented with a mineral mix composed of 35 g dicalcium phosphate, 65 g limestone and 70 g sodium chloride when the total daily intake of dry foods is 14 kg/500 kg BW. Proportionately less will be required for smaller rations. A proprietary mixture of trace elements and vitamins should also be given. The latter should include vitamin A for horses with no access to pasture (30 000 iu/500 kg BW daily, NRC 2007). Where large amounts of silage or haylage are used, supplementary vitamins D and E will be needed at levels, respectively, of 3300 and 1000 iu/500 kg BW daily (NRC 2007). Whereas the trace-element content of mare's milk (see 'Trace elements', Chapter 3), and therefore the adequacy of the foal's diet, is affected by the supplementation of the dam's diet, a deficiency of water, energy, protein, Ca or P will ultimately bring about a decrease in milk output, without altering its composition.

Growth of the foal

As horses grow they do not simply increase in weight and size, they also display development. Various tissues and organs of the body grow at different rates. In proportion to body size the potential rate of weight gain of the body as a whole is very much greater in the younger than in the older animal. From the suckling period onwards the rate of gain per 100 kg BW declines continuously, but the rate of growth of long bone and muscle declines at an even faster rate. An increasing proportion of the gain constitutes fat, which has much higher demands for feed energy. These trends are fundamental to a formulation of requirements for protein, Ca and P in particular, which decline as a proportion of total diet with increasing age of the foal and yearling (see Tables 2.3, 3.12, and 6.7). Further details of growth, and the way in which we should guide it, are given in Chapters 7 and 8.

RATION FORMULATION USING THE DE AND NE SYSTEMS

Both the DE and NE systems require the accumulation of two sets of information:

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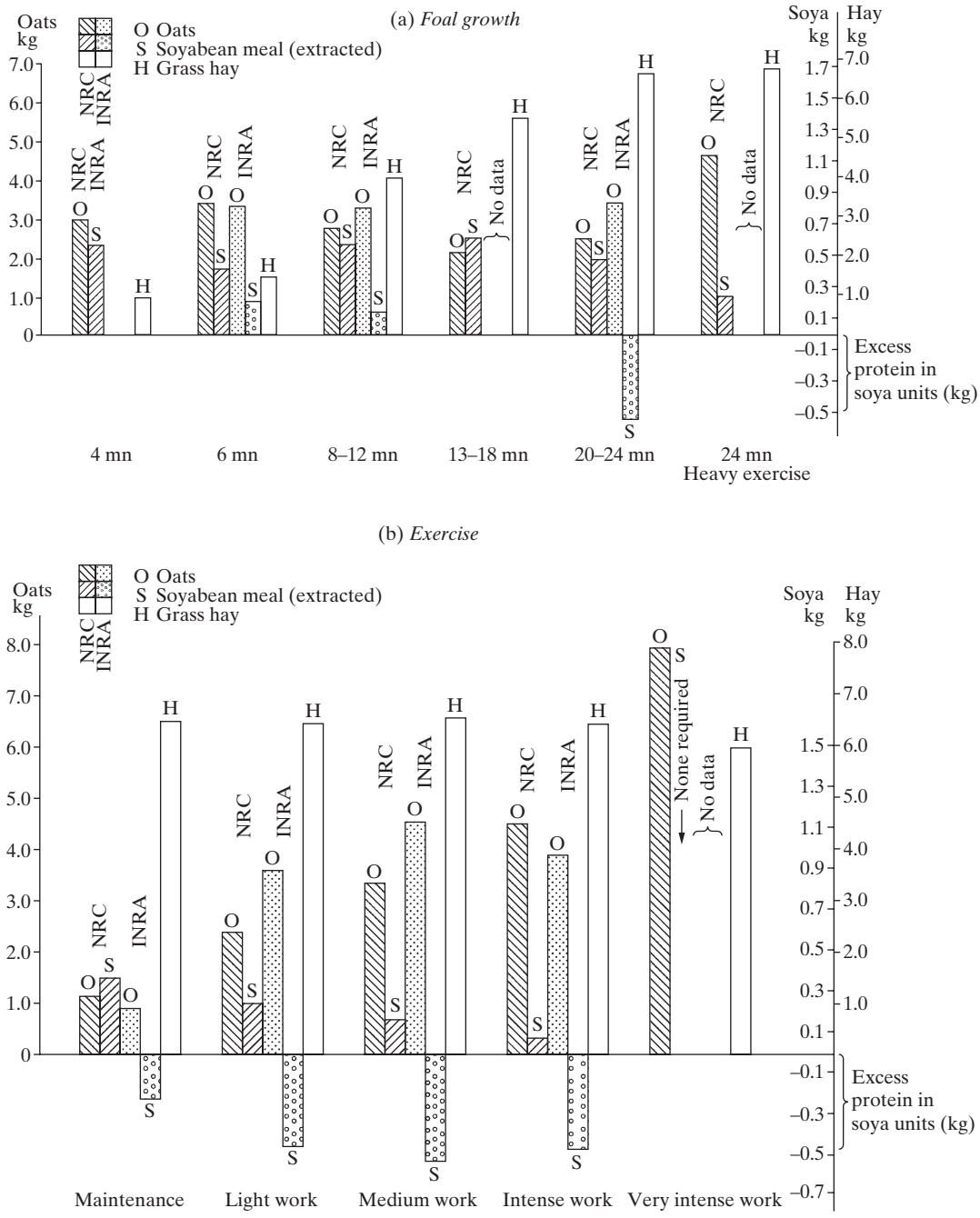
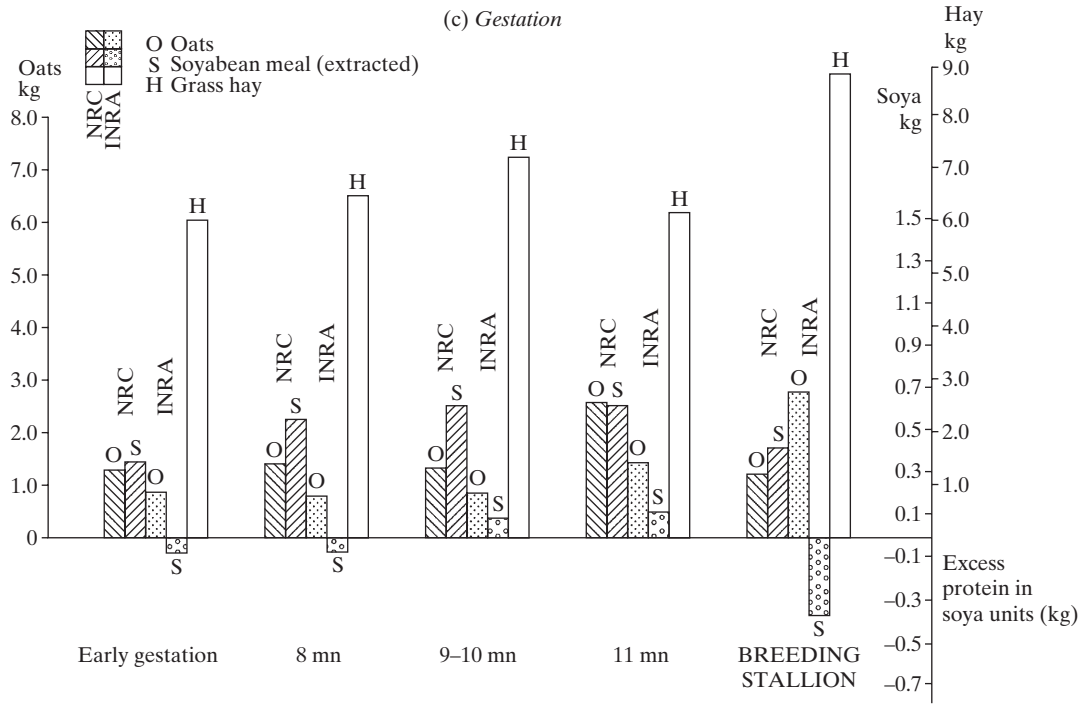


Figure 6.11 **(a–d)** Comparisons between the DE and CP systems (NRC 2007) and NE (UFC) and MADC systems (INRA 1984, 1990) for determining the protein and energy requirements for different activities (a, foal growth; b, exercise; c, gestation; d, lactation), as described by diets composed of oats, soyabean meal and grass hay, of compositions given in Table 6.23, excluding mineral and vitamin needs. The quantities of grass hay used were fixed and identical between the two systems in each comparison.

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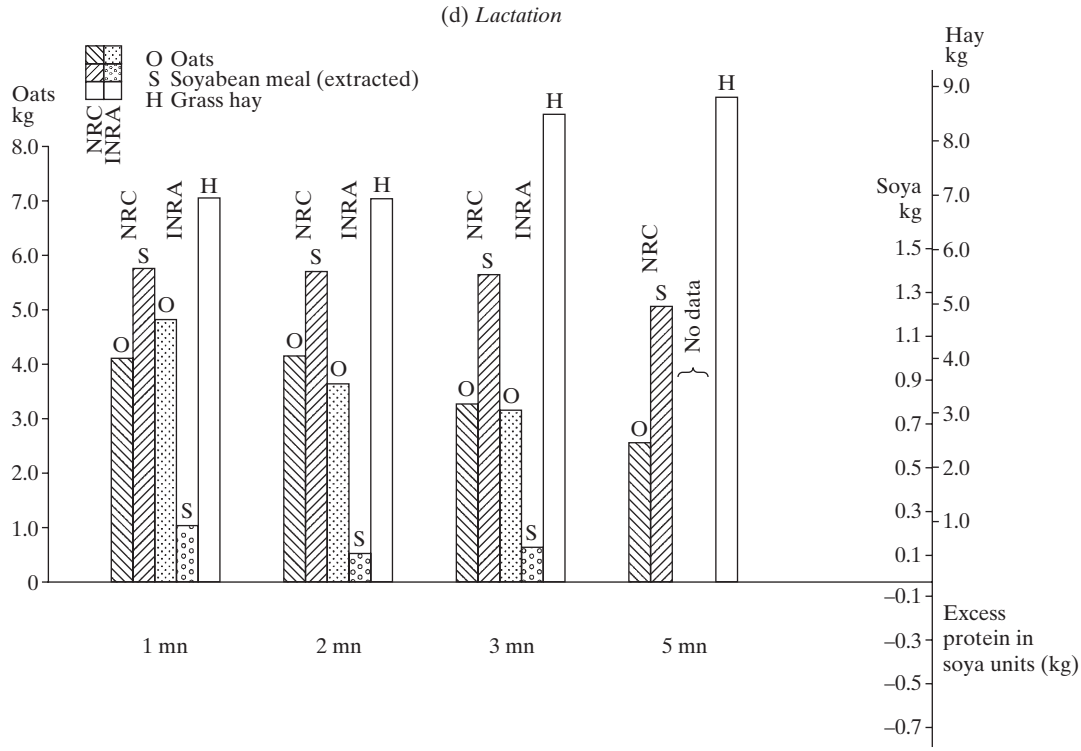


Figure 6.11 *Continued*

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Table 6.7 Nutrient concentration in diets for horses and ponies expressed on the basis of DM (based on NRC 2007).

	Intake (kg DM/d)	Crude protein (g/kg)	Ca (g/kg)	P (g/kg)
Mature horses and ponies at maintenance	7.45	85	2.8	1.9
Mare, last 90 days of gestation	7.35	118	4.9	3.6
Lactating mare, first 3 months	10.1	152	5.8	3.8
Lactating mare, 3 months to weaning	9.35	142	4.2	2.6
Creep feed		160	8.0	5.5
Foal (3 months old)	4.2	160	9.3	5.2
Weanling (6 months old)	5.0	135	7.7	4.3
Yearling (12 months old)	6.0	141	6.3	3.5
Long yearling (18 months old)	6.5	131	5.7	3.2
Two year old, light training	6.6	126	5.6	3.1
Mature working horse, light to intense work	10	97	3.7	2.1

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(1) nutrient content of feeds (Appendix C); and
(2) nutrient requirements of horses described in the same terms as for feed (see Table 6.6 for DE).

Energy needs

The principle that has been adopted in calculating the daily requirements for many nutrients is to assume that the needs of various functions of the horse are additive, i.e. a factorial system has been used, the factors or functions being maintenance, work, growth, reproduction, etc. This approach is not precisely supported by biological evidence, but it is the simplest approach and the values (coefficients) can be modified and the factors augmented as new information comes to hand, or as the activities of horses are extended. The daily energy needs for:

- maintenance (m), are a function of BW;
- work (w), are a function of BW, intensity (I) and time (T) spent;
- growth (g), are a function of weight gain (G) and BW relative to mature size;
- pregnancy (p), are a function of the mare's BW and stage of pregnancy (S_p); and
- lactation (l), are a function of stage of lactation (S_l) and yield per day (Y).

Rationing (kg feed/day) is based upon energy allowances and, in the case of a pregnant, lactating mare, energy requirements would be summated from:

$$\mathrm{m(BW)} + \mathrm{p(BW \times S_p)} + \mathrm{l(S_l \times Y)} = \text{energy needs (MJ/day)} \quad (8)$$

which, when divided by the energy content of the feed (for that energy system), gives the kg feed/day. Alternatively, appetite (defined as kg feed/day) would need to be estimated and then the required energy density of the diet calculated from:

$$\text{needs (MJ)/appetite (kg)} = \text{MJ/kg diet}$$

The proportions of roughage and concentrates are then calculated:

$$\text{MJ/kg diet} = x\,(\text{in MJ/kg roughage}) + (1-x)(\text{in MJ/kg concentrates}) \quad (9)$$

where x is the dietary fraction or proportion (typically 0.6–0.7) of roughage and the remainder of the diet $(1 - x)$ is concentrate (typically 0.4–0.3), ignoring water. This gives the proportions of roughage and concentrate making up the total appetite (kg/day).

The formulation of a ration requires estimates of: (1) total daily dry food intake, (2) the energy content of feeds, and (3) daily energy requirements (DE system, Tables 6.6 and 6.8). The two energy systems proposed for use, supplying information on (2) and (3), are the

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Table 6.8 DE demands of maintenance and work (based on NRC 2007).

Body weight (kg)	200	400	600
Approx. feed capacity per day (MJ DE)	60	100	150
Average maintenance requirement per day (MJ DE)	28	56	84
Elevated maintenance requirement per day (MJ DE)	31	61	91
	Energy requirements for work above maintenance (MJ DE)*		
Light exercise	5.4	11.3	16.7
Moderate exercise	10.9	22.2	33.5
Heavy exercise[E]	14.2	28.5	42.7
Very heavy exercise[E]	27.2	54.8	82.0

*1 kg concentrate provides about 12 MJ DE.
[E]In addition to elevated maintenance requirement.

NRC (DE) system (NRC 2007) and the INRA (NE) system (1984). The justification for each is that for:

- DE – digestibility is the most potent factor that segregates feeds that otherwise have similar GE values;
- NE – roughages and concentrates can be clearly segregated according to the efficiencies (k) by which the ME is utilized for maintenance and productive purposes. However, the INRA NE system uses barley as a reference unit, which is not a common horse feed in many parts of the world.

(Note: the $q \times k$ values will differ somewhat according to the function of the horse, e.g. maintenance, fattening, milk secretion, etc. However, for simplicity in application, the INRA system assumes that efficiencies (k) of ME for maintenance apply to all functions. Moreover, the energy requirements for growth account for only 20% of the total energy requirements of the growing animal (Vermorel & Martin-Rosset 1997). The remaining 80% is consumed in maintenance. In the final analysis, the practical value of each system depends on the reliability of its feed-evaluation system (Appendix C). Precise nutritional definitions are elusive for roughages and succulents and the relative value of the two systems will in practice depend on the development of this evaluation.

The DE system of feed evaluation

The DE required in MJ/day divided by dry feed intake in kg/day gives MJ of DE per kg of dry feed needed. The DE contents of roughages and concentrates are given in Appendix C and their required dietary proportions can then be roughly calculated using equation (9) above (Appendix A). Equation (9) has been used in deriving the proportions in Table 6.9. The greater the intensity of physical activity, the higher the proportion of cereals required. As the speed of the horse increases, the energy expended

Table 6.9 Effect of a range of required energy densities (MJ DE/kg air-dry feed) on the cereal content of the daily ration when hays of two energy contents are available.

Energy density of ration required	Oats (%)		Barley (%)	
	7.2[1]	7.8[1,2]	7.2[1]	7.8[1,2]
7.5	7	0	5	0
8.0	19	5	14	4
8.5	30	19	23	14
9.0	42	32	32	24
9.5	54	46	41	34
10.0	65	60	50	44
10.5	77	73	59	54
11.0	88	86	68	64

[1]Energy content of hay (MJ DE/kg): 7.2 MJ/kg, medium quality; 7.8 MJ/kg, good quality.
[2]Hay can be assumed to contain 86% DM and where haylage of 45% DM is to be used it can be substituted for the hay of 7.8 MJ DE in the proportions 1.8–1.9 kg haylage per 1 kg hay. Similarly, 1.6–1.7 kg haylage of 50% DM could be used.

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rises steeply on an hourly basis (Tables 6.6 and 6.8). The types of problem encountered can be quite different in horses undertaking strenuous effort compared with those asked to respond in a leisurely fashion. Furthermore, compared with ponies, large horses tend to require a higher proportion of concentrates in the ration when both are subjected to hard work.

The DE of a feed can be estimated from the following (Pagan 1998):

$$\begin{aligned}\text{DE in kcal/kg DM} = {} & 2118 + 12.18(\text{CP\%}) - 9.37(\text{ADF\%}) \\ & - 3.83(\text{hemicellulose\%}) + 47.18 \\ & (\text{fat\%}) + 20.35(\text{non-structural carbohydrate\%}) \\ & - 26.3(\text{ash\%});\ R^2 = 0.88,\end{aligned} \quad (10)$$

where

$$\text{hemicellulose} = \text{NDF} - \text{ADF, and nonstructural carbohydrates} = 100 - \text{NDF\%} - \text{fat\%} - \text{ash\%} - \text{CP\%}$$

For energy digestibility (ED; French & Dutch) take *in vivo* organic matter digestibility (OMD) data:

$$\text{ED}\ (\%) + 0.034 + \Delta + 0.9477\ \text{OMD}\ (\%) \quad (11)$$
$$\text{residual s.d.} = 1.1\ R^2 = 0.95$$

where $\Delta = -1.1$ for forages and +1.1 for concentrates.

$$\text{Thus, GE in MJ/kg DM (ED\%)} = \text{DE in MJ (kg/DM)} \quad (12)$$

(Smolders, Steg & Hindle 1990; Vermorel & Martin-Rosset 1997; Vermorel *et al.* 1997a,b)

Where OMD data for horses are unavailable, data from sheep have been applied, following comparative studies of horses and sheep:

For fresh grass and hays:

$$\text{OMD}_{\text{horses}}\ \% = -16.71 + 1.1436\ \text{OMD}_{\text{sheep}}\ (\%) \quad (13)$$
$$\text{residual s.d.} = 1.8\ R^2 = 0.98\ (\text{CVB 1996}).$$

Where neither sheep nor horse data are available the OM digestibility in horses is estimated from *in vitro* data:

$$\text{OMD}_{\text{horses}}\ \% = -8.66 + 0.9712\ \text{OMD},\ \textit{in vitro} + 9.07\ V\ (\%) \quad (14)$$

V = 0 for fresh grasses, hay, artificially dried forages (Tilley & Terry 1963).

The recommendations in Tables 6.6 and 6.9 are likely to be in error where low energy densities are required by working horses. In those situations the energy requirements are likely to be underestimated and so the French NE system is discussed below.

The NE system

The NE system was introduced in France by INRA in 1984 and updated in 1990.

Energy

The NE system provides the NE content of feedstuffs for maintenance. The feed values in this scheme are expressed in unitless horse feed units (*Unite Fourragere Cheval*, or UFCs), i.e. relative to a reference value of 1 for barley, where 1 kg standard barley has an NE value of 9.414 MJ (assuming barley contains 140 g moisture/kg). Thus:

$$1\ \text{UFC} = 9.414^*\ \text{MJ NE} \quad (15)$$

In the INRA tables, and Appendix C, UFCs are given per kg DM, so that UFC values are: barley 1.16, maize 1.33, etc. (i.e., each UFC value is divided by its fractional DM content). For barley, with 86% DM, the value is 1/0.86 = 1.16.

Energy value of feed

The NE contents of feeds for *maintenance* are calculated from their ME contents and the coefficients of their respective efficiencies of utilization for maintenance:

$$\text{NE} = \text{GE} \times \text{dE} \times \text{ME/DE} \times k_m \quad (16)$$

where dE is the digestibility calculated from the digestibility of OM and k_m is NE/ME for maintenance.

The UFC of a feed is its NE value relative to that of standard barley:

$$\text{UFC} = (\text{ME} \times k_m)/9.414$$

where NE of barley is 9.414* MJ/kg and ME is in units of MJ/kg (n.b. UFC is unitless).

Maintenance was chosen as it represents 50–90% of energy expenditure of horses and the NE feed values for maintenance are considered by INRA to be equivalent to those for physical activity, or work, a common equine function (i.e. $k_m \equiv k_w$). The NE for both maintenance and

* Increased to 9.42 MJ NE (Martin-Rosset *et al.* 2006).

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activity is expended mainly in ATP synthesis. In using the system one should apply both the INRA feed *and* requirement values.

INRA (1984) have calculated the NE requirement for three types of horse, annotated A (pony), B (warmblood), and C (draught). These requirements also form the basis of the Dutch (CVB) system (where VEP is the Dutch Horsefeed Unit), and they are set lower than ME_m, as the energy units of feed are adjusted for nutrient absorption and energy cost of eating, i.e. the NE of medium hay, as a per cent of ME is approximately 66%. The energy cost of eating has been reported to range from 1 to 28% of ME, i.e. the costs of voluntary activity and of digestion and absorption (Vermorel *et al.* 1997b; Martin-Rosset 2000).

Efficiency (k_m) of utilization of ME for a range of feeds:

$$\text{Concentrate feeds: } k_m = 0.85\,E_{Gl} + 0.80\,E_{LCFA} + 0.70\,E_{AA} + (0.63 \text{ to } 0.68)\,E_{VFA} \quad (17)$$

$$\text{Forages: } k_m = 0.85\,E_{Gl} + 0.80\,E_{LCFA} + 0.70\,E_{AA} + (0.63 \text{ to } 0.68)\,E_{VFA} - 0.14(76.4 - ED\%) \quad (18)$$

where E_{Gl}, E_{LCFA}, E_{AA}, and E_{VFA} = % of absorbed ME supplied by glucose, long chain fatty acids, amino acids and VFAs, respectively, and ED% = % digestible energy (Vermorel & Martin-Rosset 1997).

UFC values were chosen as a basis of feed formulation and discriminating between feedstuffs for two main reasons: first, they are approximately additive, that is, different combinations of feedstuffs yielding the same total UFC should have the same productive effect, or the UFC value of a feed is not influenced by other feeds with which it is combined. Martin-Rosset & Dulphy (1987) showed that, in the horse, digestibility of feed was also not influenced by feeding level, and that the digestibility of forage was not affected by the addition of concentrates to the ration, in contrast with the effects in sheep. The second, and most important, reason for selecting NE is that k_m draws a clear distinction between the productive values of forages and concentrates. The energy costs of mastication and propulsion of digesta through the GI tract and the heat of fermentation of forages in the hind-gut are greater than the heats of ingestion and digestion of starch. In addition, the efficiency of utilization of VFA derived from forage fermentation is less than the efficiency of glucose metabolism from starch. These costs and efficiencies both affect the value of k_m. In other words, the formulation of mixed feeds from the DE values of their constituent ingredients, with the objective of deriving a variety of mixtures with the same productive energy values, exaggerates the value of forages. The k_m value of standard barley is 0.79 and that of an average grass hay is 0.62. High quality fats are utilized much more efficiently with very high digestibility values and their metabolic utilization, once absorbed, is efficient e.g. for corn oil the ME/DE × k_m is ≃ 0.85, whereas the comparative value for a hay/grain diet is<0.60 (Kane *et al.* 1979). The comparisons (Table 6.10) between concentrates and forages on a DM basis exemplify the point. The ratios of the means indicate that, relative to concentrate values, the DE system overvalues these roughages by 15% for production. However, large effects occur only with materials at the extremes, i.e. maize and barley straw, otherwise the bias introduced by the DE system (ingestion only) is small (Table 6.11). INRA have data to support the adoption of the UFC system, and their conclusions are in line with proposals put forward by Frape and Tuck (1977).

Table 6.10 Comparison of the DE and NE values for concentrates (C) and forages (F) per unit DM.

	DE (MJ/kg)	UFC
Barley (C)	15.2	1.163
Maize (corn) (C)	16.1	1.35
Oats (C)	13.4	1.01
Maize silage (F)	11.2	0.88
Grass hay (F)	7.3	0.53
Barley straw (F)	6.8	0.28
Ratio F/C	0.566	0.480

Table 6.11 Energy cost of eating as a proportion of the ME value of the individual feed measured at 1.26 times maintenance (Vernet *et al.* 1995).

	Proportion of ME/kg feed DM expended in eating
Pelleted maize	0.010
Pelleted sugar-beet pulp	0.042
Long hay	0.102
Wheat straw	0.285

Energy cost of eating

Chewing, ingestion and digestion of feed involve muscular activity. In addition, there is an increase, above the fasting rate, in general metabolic rate. Vernet *et al.* (1995), using

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indirect calorimetry, measured the energy expended by sport horses during ingestion of feeds of different types, at the level of 1.26 times the maintenance energy requirement, when maintenance energy demands were increased by 38.8% during eating a mixed meal, and maintenance heat production is increased by 75–95% during a meal of straw or hay (Vermorel & Martin-Rosset 1997). For single feeds the energy costs of eating per kg DM intake are given in Table 6.11.

The energy cost of eating is mainly accounted for by the energy expended in chewing. An additional amount is contributed by cephalic activation of the sympathetic and parasympathetic nervous systems. The sight and smell of feed stimulates a physiological response in the whole GI tract and in some other organs. The measurements by Vernet *et al.* included only the increments in expenditure during eating and so excluded much of, for example, the HI of bacterial fermentation. The data do indicate that with poor-quality feeds, such as straw, over a quarter of the ME they contribute is lost during the process of taking the feed into the stomach. For mixed feeds, the energy costs of eating are 6–10% of ME (Vermorel *et al.* 1997b), and although the horse expends a similar amount of energy as that used by ruminants per minute, it expends two or three times as much as the ruminant per kg DM, owing to a lower rate of eating (Vermorel & Martin-Rosset 1997).

Protein

The scheme also accounts for the DCP content of feeds and, in the case of forages, this is reduced in proportion to the NPN contents. The useful protein is calculated from the estimated amounts of amino acids absorbed from the small and large intestine and is termed *matières azotées digestibles corrigées* (MADC) in French (corrected digestible N matter). The MADC, measured in g/kg DM, is derived from the DCP by multiplication with the K coefficient (not to be confused with the coefficient *k*). See further below.

ENERGY AND PROTEIN REQUIREMENTS BASED ON INRA FEED UNITS

The recommended allowances in UFCs for particular physiological functions were determined by a factorial method, or by feeding experiments. The scheme therefore partly allows for differing *k* values for maintenance, growth, lactation and work (N.B. the same *k* value applies for maintenance and work, see 'Energy value of feed', above) for each feedstuff. Thus, *k* values partly depend on the particular mix of nutrients, that is, the substrate for metabolism, i.e. VFAs, fats, glucose and the proportions of the amino acids (Appendix C). In these respects, the variation in *k* is greater for growth than for maintenance. Products of poor-quality roughage have relatively less value for growth than for maintenance. Although *k* values differ for different functions, UFC values will differ less as they are unitless ratios. The NE system could be more appropriate than the DE system for exercising horses, as DE overestimates the value of forages compared with grains or fats (NRC 2007). Martin-Rosset & Vermorel (2004) calculated that a diet of hay, barley and soyabean meal that met the DE requirements (NRC 1989) of growing horses exceeded the NE requirement estimated by the UFC system by about 19% (cf. Figure 6.11a).

Maintenance

The maintenance requirement of an idle horse is: (19)

$$0.038 \text{ UFC}/\text{kg BW}^{0.75}$$

This is approximately 4 UFC/day for a gelding weighing 500 kg.

The maintenance requirement of working horses, cf. horses at the maintenance level of activity, is assumed to be increased by 5–15% and that of working stallions is increased by 10–20%. The higher maintenance heat production assumed for working horses results from a higher metabolic rate.

Protein for maintenance of adult horses

Dietary protein required for maintenance of adult horses can be of poorer quality than that needed for growth and so, within the range of dietary proteins given to horses, no adjustment for amino acid balance is needed. The daily requirement is:

$$2.8 \text{ g MADC}/\text{kg BW}^{0.75} \quad (20)$$

so that a 500 kg horse requires 295 g MADC daily.

Protein for maintenance related to DM intake and endogenous faecal N losses

The horse needs protein, which is digested, yielding amino acids (and probably dipeptides) which are absorbed. The apparent digestibility of protein differs only to a small extent between sources. Apparent digestibility is expressed as:

$$\frac{(\text{N intake} - \text{faecal N})}{\text{N intake}}$$

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Meyer (1983b) concluded that precaecal apparent N digestibility was 0.5–0.6. As this is 'apparent' it makes no allowance for endogenous N secreted into the lumen of the GI tract, whereas true digestibility does. Mean true values for the small intestine should be slightly greater than 0.5–0.6, probably 0.5–0.8. Endogenous faecal N loss is proportional to body weight:

$$\text{Daily endogenous N loss} = 52 \text{ mg/kg BW}^{0.75}$$
(Slade *et al.* 1970)

although endogenous loss also varies with the level of feeding. This could account for the much higher estimate of Meyer (1983b) of 180 mg N/kg $BW^{0.75}$, giving:

$$\text{Endogenous N} = 3 \text{ g N/kg DM intake} \quad (21)$$

At constant DM intake, apparent N digestibility increases as the N content of the diet is increased, because the endogenous loss forms a smaller proportion of the total N. Likewise, true digestibility, for which the endogenous N loss is subtracted from the faecal N, is approximately similar for a range of dietary N concentrations. For most protein sources, true N digestibility falls in the range 0.7–0.9, and no allowance is made for any change in dietary N concentration.

Protein sources that contain anti-nutritive factors can increase endogenous protein secretions. In the pig, toasted field-bean protein, which contains some anti-nutritional factors, leads to a negative apparent N absorption in the small intestine (ileal N digestibility), although true N digestibility is reasonable. If this were to apply to the horse, it would have two consequences:

(1) the measurement of endogenous N production using protein-free diets underestimates endogenous secretions; and
(2) if only non-amino-acid N is absorbed from the large intestine, then amino acids contained in endogenous protein secretions passing the ileocaecal valve would be lost and the stimulation to secretion by anti-nutritional factors would compromise feeding value.

A consequence of this is that field-bean protein has a lower amino acid value than field-pea protein, despite similar apparent N digestibilities and not too dissimilar true N digestibilities. It should be noted that whereas apparent N digestibility reflects the net absorption of N to be metabolized by the horse, true digestibility does not, and requirements in that case must also be in terms of truly digested amino acids. Apparently digested N will overestimate usable N because a major proportion of N absorbed from the hind-gut will be NPN, providing no material benefit.

Endogenous urinary N

Horses given an N-free diet will continue to lose N in the urine, through the metabolism of tissue proteins. This is taken to be the minimal endogenous renal N loss, and was provisionally calculated (Meyer 1983b) as:

$$\text{Daily endogenous renal N} = 165 \text{ mg N} \times \text{kg BW}^{0.75} \quad (22)$$

There is renal conservation of N in that endogenous urinary loss is minimal in horses on low-N diets and the amount increases with an increase in dietary protein, whereas endogenous faecal N loss is relatively constant with a change in dietary protein content.

Endogenous integumental N

Losses of N from skin and hair should be allowed for. An average value of:

$$\text{Daily integumental N} = 35 \text{ mg N} \times \text{kg BW}^{0.75} \quad (23)$$

is taken (Meyer 1983b). This slightly underestimates the rate of loss during seasonal hair shedding.

Total endogenous N

A reasonable total endogenous N value from the three sources of loss is:

$$\text{Daily basal endogenous N} = 380 \text{ mg N} \times \text{kg BW}^{0.75} \quad (24)$$

(i.e. 180 + 165 + 35), equivalent to 2.4 g CP/kg $BW^{0.75}$.

N balance has been achieved during feeding trials with about 350 mg digestible N/kg $BW^{0.75}$; but horses thrive better with some accumulation of protein reserve. Therefore, in CP terms the maintenance requirement (m) for DCP is taken to be:

$$DCP_{(m)} \text{ in g/day} = 3.3 \text{ g} \times \text{kg BW}^{0.75} \quad (25)$$

(i.e. CP + N × 6.25).

The energy requirement for maintenance according to German workers (Meyer 1983b), is:

$$DE_{(m)} = 0.6 \text{ MJ} \times \text{kg BW}^{0.75} \quad (26)$$

(in accordance with the INRA value of 0.038 UFC or 0.36 MJ NE × kg $BW^{0.75}$).

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Table 6.12 Weight gain and composition of the foetus (Martin-Rosset *et al.* 1994).

During month	Weight gain (g/kg birth weight)	Foetus weight gain, fraction of birth weight	GE content (MJ/kg)*	Protein content (g/kg)
8	190	0.18	4.18	115
9	190	0.20	4.60	130
10	300	0.30	4.94	153
11	310	0.31	5.36	171

*Of conceptus (foetus + foetal membranes + uterus + udder).

So, in relation to energy, requirement for maintenance is:

$$5.5 \text{ g DCP/MJ DE, or } 9.2 \text{ g DCP/MJ NE} \quad (27)$$

Breeding mare

Energy for pregnancy

The amounts of energy deposited in the products of conception (foetus + placenta + foetal membranes + udder) are approximately:

- 8th month, 0.636 MJ/100 kg BW/day; and
- 11th month, 1.954 MJ/100 kg BW/day.

In the 8th–9th, 10th and 11th months of pregnancy, 14%, 41% and 45% of the total energy at birth, respectively, is deposited in these products (Table 6.6 and Table 6.12). The INRA system assumes an efficiency of ME utilization (k) for pregnancy of 0.25. The daily allowance in UFC is adjusted according to the mare's condition, as undernutrition of a mare in good condition has no adverse influence on the foal's birth weight, or on its growth rate. The effect of this adjustment on fertility at foal heat, etc. has not been established.

Energy for lactation

The mean characteristics of milk adopted in the INRA system are given in Table 6.13 (also Table 7.2). The efficiency of ME use (k) assumed in estimating feed requirements is 0.65.

Protein requirements

Prior to the eighth month of pregnancy, the mare's protein requirement does not materially exceed that for maintenance. Foetal growth, as a fraction of birth weight, increases each month from the eighth, when it is about 0.18 of that at birth (Table 6.12). In breeds weighing 500 kg, mature weight:

Table 6.13 Mare's milk yield and composition (Martin-Rosset *et al.* 1994).

Month	Daily milk yield (kg/100 kg BW)	Milk composition	
		Energy (MJ/kg)	Protein (g/kg)
1	3.0	2.41	24
2	2.5	2.09	24
3	2.5	2.09	24
4	2.0	1.99	21

$$\text{Foetal weight (kg)} = 0.00067x^2 - 20 \quad (28)$$

where x is time in days from fertilization (Meyer 1983b).

$$\text{Birth weight (kg)} = 0.45\ BW_m^{0.75} \quad (29)$$

where BW_m is mare's weight (kg) (Gotte 1972 in Meyer 1983b).

Equations 28 and 29 indicate that foetal growth rate accelerates towards the end of pregnancy and that birth weight is a function of mare's weight, i.e. small mares drop small foals. The mean birth weight of a foal out of a 500 kg mare is 47.6 kg.

The protein content of the foetus also increases from 115 g/kg in the eighth month to 171 g/kg at birth (Table 6.12) and the protein retention in foetal membranes, uterus and mammary gland is about 0.2 of that in the foal. The fraction of the total protein (a) retained in the foal at birth was estimated (Meyer 1983b), giving the additional daily protein requirement of a pregnant mare for foal's growth *in utero:*

$$\text{DCP (g/day)} = 6.15a(BW_m)^{0.75} \quad (30)$$

where BW_m is mare's weight in kg (Table 6.14).

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Table 6.14 Protein requirements of breeding mares (g DCP/day) (based on Meyer 1983b). These estimates differ from those in INRA tables (Anon 1984).

Month	Fraction (a) of total protein retained in foal at birth	Body weight of mare (kg)		
		200	500	800
Pregnancy				
8th	0.14	227	451	641
9th	0.22	250	500	700
10th	0.23	260	528*	740
11th	0.31	282	561	798
Last days	0.10			
Lactation				
1st		585	1163	1655
3rd		611	1214	1727
5th		468	931	1324

*For example, this value is the sum of 150 g for foetus (equation 19) + 30 g for membranes, etc. (0.2 of foetus) + 348 g for mare (equation 15).

The mare's dietary protein requirement increases from the eighth month of pregnancy as the fraction of the total protein (a) increases.

The milk production of mares increases up to about the third month, whereas its protein content decreases from about 24 g/kg milk in the first month to 21 g/kg in the fourth month (Table 6.13). Meyer (1983b) assumes that the efficiency of utilization of DCP for the products of conception and milk protein synthesis is 0.5, leading to the recommendations given in Table 6.14.

INRA protein requirements for pregnancy

For the growth of the uterus, including its contents, and of the udder, the amount of protein retained daily is 5 and 21 g/100 kg BW in the 8th and 11th months, respectively. INRA assumes a metabolic efficiency for DCP of 0.50–0.55. The extra concentrate protein required by a 500 kg mare is approximately 45–50 g at 8 months, and 190–210 g at 11 months (Table 6.15 values less mare's maintenance requirements; the values in this table have been updated in accord with 1990 INRA data.) These values are considerably lower than Meyer's (1983b) and than NRC (2007) values.

INRA protein requirements for lactation

The mean composition of milk is given in Table 6.13. Assuming a metabolic efficiency for DCP of 0.55, an INRA requirement is 44, 38, and 36 g DCP/kg milk in months 1, 2–3 and 4 of lactation, respectively, which is lower than Meyer's (1983b) and NRC (2007) estimates. This gives a requirement, including maintenance, of 950 g for a 500 kg mare in the first month, in agreement with the values quoted by Martin-Rosset *et al.* (1994), and approximately in accord with the revised values given in Table 6.15.

Growth

The efficiency of ME use for growth has not been established and will depend both on the energy substrates and on the proportions of fat and protein laid down (N.B. The index of the second term on the right side of equation 31 (**1.4**), probably derives from the increase in fat deposition resulting from faster growth rates). Maintenance accounts for 60%, or more, of the energy expended, depending principally on the rate of growth allowed. The total requirements are assumed to fit the following relationship:

$$\left(\text{UFC}/\text{kg BW}^{0.75}\right)\text{day}^{-1} = a + bG^{1.4} \qquad (31)$$

where a = coefficient of maintenance requirement, b = coefficient of gain, and G = average weight gain in kg/day (Table 6.16).

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Table 6.15 Daily energy (UFC) and protein (MADC, g) requirements of horses as proposed by INRA [Anon 1984; updated by INRA, Ed. Martin-Rosset (1990)]. All figures in brackets are assumed average daily gain (kg).

Adult weight (kg)	450		500		600	
	UFC	MADC	UFC	MADC	UFC	MADC
Maintenance	3.9	275	4.2	295	4.8	340
Light work	6.6	450	6.9	470	7.5	510
Medium work[1]	7.6	515	7.9	540	8.5	580
Intense work[2]	6.9	470	7.2	490	7.8	530
Mare, gestation						
month 8	3.8	315[5]	4.1	340[5]	4.7	395[5]
months 9–10	4.3	425	4.7	460	5.4	535
month 11	4.4	445	4.8	485	5.5	565
Mare, lactation						
month 1	8.2	865	8.9	950	10.5	1125
month 2	7.0	700	7.6	770	8.9	910
month 3	7.0	700	7.6	770	8.9	910
Growth[3]						
6 months	4.2 (0.70)	500	4.5 (0.75)	530	5.1 (0.85)	600
8–12 months	5.1 (0.70)	560	5.5 (0.75)	590	6.2 (0.85)	660
20–24 months	6.3 (0.40)	380	6.8 (0.45)	420	7.8 (0.55)	480
32–36 months	5.9 (0.15)	300	6.5 (0.20)	330	7.6 (0.30)	390
Stallion[4]						
resting	5.8	400	6.1	420	6.3	440
breeding	6.6–8.0	480–620	7.0–8.4	490–630	7.0–8.5	520–650

[1]2 hours daily.

[2]1 hour daily.

[3]Based on equations: daily UFC/kg $BW^{0.75}$ = a + $bG^{1.4}$, and daily MADC (g) = a $BW^{0.75}$ + bG, where a = coefficient of maintenance, b = coefficient of gain, and G = body weight gain (kg/day) (see Table 6.16).

[4]Including 0.5 hours medium exercise daily.

[5]These values agree with Martin-Rosset *et al.* (1994) resulting from a deposition of 5 g/100 g BW and 0.5–0.55 metabolic efficiency for protein in products of conception.

Table 6.16 INRA coefficients used in the estimates of requirements for growth of light breeds (daily UFC/kg $BW^{0.75}$ = a + $bG^{1.4}$, and daily MADC (g) = a$BW^{0.75}$ + bG).

	UFC		MADC	
Age (months)	a	b	a	b
6–12	0.0602	0.0183	3.5	450
18–24	0.0594	0.0252	2.8	270
30–36	0.0594	0.0252	2.8	270

where a = coefficient of maintenance requirement; b = coefficient of gain; G = average weight gain (kg/day).

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Table 6.17 Digestible CP recommendations (DCP, g/day)* for growing foals at moderate and fast growth rates (after Meyer 1983b).

	Weight of mature animal (kg) and growth rates									
	200		500		500		800		800	
			Moderate		Fast		Moderate		Fast	
Age (months)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)	Average BW (kg)	DCP (g)
3–6	85	290	170	503	175	544	232	635	292	870
7–12	130	260	258	474	288	546	360	695	444	736
13–18	160	225	333	461	365	493	500	731	548	621
19–24	175	230	388	454	425	488	612	688	612	622

*Assumes requirements for maintenance of 4.5 g DCP/kg $BW^{0.75}$ at 3–6 months, decreasing to 4.1 g DCP/kg $BW^{0.75}$ at 19–24 months of age.

Table 6.18 Digestible CP requirements per kg $BW^{0.75}$ in working horses (Meyer 1983b).

	Light work	Moderate work	Heavy work
DM intake (g)	70	80	100
Faecal N losses (mg)	210	240	300
N losses in sweat (mg)	12	30	50–145
DCP requirement (g)	3.7	4.1	5.3
DE requirement (MJ)	0.68	0.83	1.0
DCP/MJ DE (g/MJ)	5.4	4.9	5.3

Therefore, 1 kg gain at 250 kg BW,
requires = 5.5 UFC (maintenance 3.6 + gain 1.9)

1 kg gain at 350 kg BW, requires = 7.0 UFC
(maintenance 4.7 + gain 2.3)

0.5 kg gain at 300 kg BW, requirement = 5.0 UFC
(maintenance 4.2 + gain 0.8)

Economy is achieved by allowing a slower growth rate in winter on mixed feeds, followed by accelerated compensatory growth on pasture in the spring (see Chapter 8).

The protein requirements for growth are similarly calculated.

INRA requirements for growth: protein for growing foals

The rate of body-weight gain, as a fraction of mature weight, declines progressively with increasing age. Concurrently, the protein content of that gain decreases and the fat content increases as age increases. The protein content of each kg gained decreases from 197 g at 3–6 months to about 170 g at two years of age in horses of 500 kg mature weight. Breeds with lower mature weights grow faster, relative to their size, and reach near mature weights at a younger age than do larger breeds. With these considerations for growth in mind, the recommendations for DCP proposed by Meyer (1983b) are given in Tables 6.17 and 6.18 (INRA recommendations).

INRA protein requirements for maintenance of growing horses

The metabolic efficiency of DCP for growth is set at 0.45 and, as the amino acid turnover rate in growing horses is greater than in adults, so the maintenance requirement for MADC is higher during growth:

MADC maintenance requirement of growing horses
$= 3.5\ \text{g}/\text{kg BW}^{0.75}\cdot\text{day}$ (32)
cf, 2.8 g MADC/kg $BW^{0.75}$·day for adults

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INRA lysine requirements for growing horses

Lysine is the limiting amino acid for growth in most conventional diets. The lysine requirement for growing horses is set at 6 g/kg dietary DM at 6 months, and 4 g/kg dietary DM at the yearling stage.

Work

INRA protein requirements related to energy requirement

For work, the INRA recommendations are that total protein needs are proportional to energy needs, (in NRC 2007, total protein needs are less than proportional) and increase with increasing size of the horse. For simplicity, INRA recommends that the work of an adult horse above maintenance requires 60–65 g MADC/UFC, i.e. protein needs proportional to those of energy (Table 6.15), but growing horses in work would require more protein. (See also Chapter 9, where requirements are considered independently of N balance.)

Protein is not wasted when chemical energy in muscle is transformed into kinetic energy. Energy for work is derived mainly from glycogen and free fatty acids. However, in extended hard work, blood urea concentration rises as a consequence of protein catabolism. Therefore protein requirements (Table 6.18) are generally considered to rise above maintenance needs to achieve N balance in working horses, accommodating:

- muscle protein anabolism;
- N losses in sweat of 1.4 g N/L (although urinary losses decrease);
- increased feed intake that increases endogenous faecal N; and
- some muscle protein catabolism in hard work (adenine nucleotide cycling also increases in exercise, increasing uric acid excretion, see Chapter 9).

Thus, from Table 6.18 the protein requirements for work are taken to be 5.5 g DCP/MJ DE, in equation (27) for maintenance (cf. Tables 2.2 and 6.6 for effect of increasing work intensity).

ENERGY, PROTEIN, MINERAL AND MICRONUTRIENT FEED VALUES AS DETERMINED BY THE INRA SYSTEM

Feed evaluation

Critical to the adoption of a system for diet formulation is a valid assessment of the energy and protein values of raw materials. Not only are there large differences in these values amongst the species of material available, but the values vary considerably within species. The experimental determination of energy and protein values in horses is expensive and time consuming, more so where net values are required. INRA has therefore developed accurate and precise *in vitro* chemical methods following the conduct of digestibility trials.

Prediction of UFC values

One horse feed unit (1 UFC) is the net energy content of 1 kg of standard barley for maintenance, i.e. 1 UFC = 9.42 MJ NE.

The UFC value of concentrate feeds can be predicted from chemical components and digestible components (Martin-Rosset *et al.* 2006). The prediction error depends on the chemical characteristics used in the equation (see Table 6.25).

Improved predictions of UFC values for feeds, based upon more *in vivo* horse digestibility data, were introduced in 2004 (INRA-AFZ, Martin-Rosset, 2004). The concentrates included: cereal grain, cereals by-products, oil seeds and legume seeds, cakes, dehydrated beet or potatoes pulp, and molasses.

For raw materials with known digestible organic matter content:

$$\text{UFC} = -0.134 + 0.274\,\text{CF} - 0.362\,\text{CP} + 0.316\,\text{CC} + 0.0755\,\text{DE};\ R^2 = 0.995;\ \text{RSD} = 0.017 \quad (33a)$$

Other raw materials (except legume seeds and cakes with high CW content):

$$\text{UFC} = 0.815 - 0.947\,\text{CF} + 0.0345\,\text{CP} + 0.582\,\text{CC};\ R^2 = 0.931;\ \text{RSD} = 0.060 \quad (33b)$$

Dehydrated alfalfa and grass:

$$\text{UFC} = -0.056 + 0.562\,\text{CC} + 0.0619\,\text{DE};\ R^2 = 0.996;\ \text{RSD} = 0.007 \quad (33c)$$

Soya hulls:

$$\text{UFC} = -0.730 - 0.722\,\text{CP} + 0.572\,\text{OM} + 0.0941\,\text{DE};\ R^2 = 0.979;\ \text{RSD} = 0.033 \quad (33d)$$

Wheat straw:

$$\text{UFC} = -0.124 + 0.254\,\text{CC} + 1.330\,\text{DOM};\ R^2 = 0.988;\ \text{RSD} = 0.012. \quad (33e)$$

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(see Abbreviations or footnotes to Table 6.25 for interpretation of abbreviations).

All above values are either g/kg DM, or MJ/kg DM, as appropriate. Barley has been assigned an increased NE value of 9.42 MJ/kg (Vermorel and Martin-Rosset, 1997; Vermorel *et al.*, 1997a).

Prediction of MADC values

For the prediction of the feeding value of proteins, a practical approach has been adopted by INRA. This method does not yield the true amount of digestible amino acids derived directly from the feedstuff, but it is intended to include the absorbed amino acids derived from the microbial metabolism of the feed proteins, excluding absorbed inorganic N that is otherwise included in the estimation of DCP. To achieve this, the DCP value is adjusted by multiplying it by the appropriate K value. INRA and other organizations have determined the digestibility of raw materials, so allowing DCP to be predicted for materials in each class from their chemical composition (Martin-Rosset *et al.*, 1994, 2006; Martin-Rosset, 2004) all in g/kg DM:

(a) Forages

Hays (n = 72)

$$\text{DCP} = -27.57 + 0.8441\,\text{CP};\ \text{RSD}\ 8.6,\ R^2\ 0.929 \qquad (34a)$$

Dehydrated grass and grass hays (n = 47)

$$\text{DCP} = -25.96 + 0.8357\,\text{CP};\ \text{RSD}\ 7.1,\ R^2\ 0.941 \qquad (34b)$$

Dehydrated alfalfa and legume hay (n = 25)

$$\text{DCP} = -29.95 + 0.8673\text{CP} \quad \text{RSD}\ 9.2\ R^2\ 0.871 \qquad (34c)$$

(b) Concentrates feeds: raw materials

Cereals (n = 23)

$$\text{DCP} = -4.94 + 0.8533\,\text{CP};\ \text{RSD}\ 7.7,\ R^2\ 0.867 \qquad (34d)$$

Cereals by-products (n = 17)

$$\text{DCP} = -17.6 + 0.865\,\text{CP} + 0.051\,\text{CF};\ \text{RSD}\ 6.7,\ R^2\ 0.945 \qquad (34e)$$

Protein concentrates (cakes) (n = 14)

$$\text{DCP} = -43.6 + 0.989\,\text{CP} - 0.127\,\text{CF};\ \text{RSD}\ 6.3,\ R^2\ 0.996 \qquad (34f)$$

Legume seeds (n = 10)

$$\text{DCP} = -7.50 + 0.844\,\text{CP};\ \text{RSD}\ 6.4,\ R^2\ 0.980 \qquad (34g)$$

Further details can be obtained in the papers to which reference is given above.

The proportion of DCP N that is amino acid N is less in green forages, and especially in silages, cf. soya and other concentrate proteins. This observation is reflected in the K coefficient by which DCP of forages is multiplied to give MADC (corrected digestible N matter), that is:

K = 0.90 for green forages
K = 0.85 for hays and dehydrated forages
K = 0.80 for straws and chaffs
K = 0.70 for good grass silages in comparison with
K = 1.00 for soya, cereals, etc.

$$\text{DCP} \times \text{K} = \text{MADC in g/kg DM} \qquad (35)$$

The absorption of amino acids takes place almost entirely from the small intestine. Absorption, of amino acids released from forage and bacterial proteins, from the large intestine is of significance for horses receiving poor-quality forage diets, adequate in fermentable energy and supplemented with NPN. INRA proposals (Tisserand & Martin-Rosset 1996) assert that amino acids synthesized within the large intestine do not materially contribute to the amino acid supply of the horse. This conclusion is consonant with evidence from other sources referred to in Chapters 1 and 2.

The resolution of the issue concerning the absorption of amino acids from the large intestine is of practical significance. Bacterial amino acid synthesis in the hind-gut is considerable, and 50–60% of faecal N in the horse is accounted for by microbial protein (Tisserand & Martin-Rosset 1996). Cuddeford and colleagues (1992) reported that the apparent CP digestibility coefficient for high temperature-dried lucerne was 0.74, in comparison with a value of 0.36 for field-cured timothy hay cut at full bloom. Gibbs *et al.* (1988) showed that the precaecal N digestibility of high-protein lucerne hay was three-fold that of coastal Bermuda grass (in contrast to the data with sun-cured lucerne of Klendshoj *et al.* (1979) in Table 6.19), and the lucerne led to greater N retention. However, the net uptakes of N from the large intestine did not differ and the overall N digestibility of the lucerne was only 29% greater than that for the Bermuda grass hay. According to Schmidt *et al.* (1982) an amount of N equivalent to 25–30% of dietary intake flows from the ileum into the

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Table 6.19 True N digestibility (N g/kg N intake) in the digestive tract of the pony of mixtures of two ingredients providing equal amounts of CP and of hays (Klendshoj *et al.* 1979) in Martin-Rosset *et al.* (1994).

	CP intake (g/kg)	Small intestine	Large intestine
Bermuda grass hay + crimped oats	122	690	130
Bermuda grass hay + micronized oats	122	580	320
Bermuda grass hay + soya-bean meal	122	730	130
Bermuda grass hay	117	360	370
Lucerne hay	150	220	570
Lucerne hay	181	380	460

Table 6.20 CP (g) true digestibility coefficients of absorbable intestinal amino acids and MADC (g) (Jarridge & Tisserand 1984).

	Intake (g)		Small intestine	Large intestine		Digestion, total tract (g)	
	CP	Amino acids	True digestibility[1]	Entry[1] (g)	True digestibility[1]	DCP	MADC
Concentrates	180	171	0.85	26	0.90	148	147
Spring grass	180	162	0.70	49	0.80	128	117
Barley–maize	110	105	0.85	16	0.90	90	90
Grass hay	110	99	0.50	49	0.75	65	54[2]
Grass silage	110	82	0.50	41	0.75	65	44[2]

[1]Of amino acids.

[2]See K values in 'Protein and the evaluation of N absorbed from the large intestine', this chapter, for grass hay $65 \times 0.85 = 54$, for silage $65 \times 0.70 = 44$, thus allowing for the NPN content of the large intestinal true digestibility of 0.75.

caecum, irrespective of feeding level or type. The important point is that, if most of the N absorbed from the large intestine is ultimately lost in the urine, then the true differences in protein value between the lucerne and timothy found by Cuddeford and colleagues, was much more than two-fold. A maximum of 10–12% of the plasma pool of amino acids is of microbial origin and derived from the hind-gut. Thus, the range in coefficients for N retention is much wider than that of true digestibility. Stated differently, where the true digestibility coefficient is low, the potential retention coefficient is very much lower, whereas with high true digestibility coefficients there is a smaller decrement in the potential retention coefficient (N.B. the actual retention depends on the tissue needs). In Table 6.20, the true absorption coefficient of amino acids for concentrates and for barley–maize was the same, i.e. 0.85, (147/171 *vs.* 90/105); however, the amino acid balance of the cereals is likely to have been poorer, so the potential N retention for growth would be lower for the cereals.

Minerals

Calcium

The bioavailability, or true digestibility, of dietary Ca varies considerably (see also Chapter 3). The principal factors controlling this are:

- amount of dietary Ca (0.7 at requirement intake to 0.46 at several times requirement);
- amount of dietary P (10 g P added/kg diet containing 4 g Ca/kg reduced true Ca digestibility from 0.68 to 0.43);
- vitamin D status (of less significance in the horse than in some other domestic species);
- dietary oxalate and phytates (Ca : oxalate <0.5 causes NSHP; phytates bind Ca. Oxalate-rich grasses include:

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Table 6.21 Minerals and vitamins in feed, adequate per day for a horse of mature weight 500 kg (based on NRC 2007).

	Maintenance of a mature horse	Lactating mare	Growing horse, 12 mn
Sodium (g)	10.0	12.8	6.9
Potassium (g)	25.0	47.8	17.4
Magnesium (g)	7.5	11.2	5.4
Sulphur (g)	15.0	18.8	12.0
Iron (mg)	400.0	625.0	401.5
Zinc (mg)	400.0	500.0	321.2
Manganese (mg)	400.0	500.0	321.2
Copper (mg)	100.0	125.0	80.3
Iodine (mg)	3.5	4.4	2.8
Cobalt (mg)	0.5	0.6	0.4
Selenium (mg)	1.0	1.25	0.80
Cholecalciferol (vitamin D) (µg)[1]	82.5 (3.3 kIU)	82.5 (3.3 kIU)	139.7 (5.590 kIU)
Retinol (vitamin A) (mg)[2]	4.5 (15.0 kIU)	9.0 (30.0 kIU)	4.35 (14.5 k IU)
DL-α-tocopheryl acetate (vitamin E) (mg)[3]	500	1000	642
Thiamin (mg)	30.0	37.5	24.1
Riboflavin (mg)	20.0	25.0	16.1

[1] 1 iu is equal to the biopotency of 0.025 µg cholecalciferol (vitamin D_3) or ergocalciferol (vitamin D_2).

[2] 1 iu is equal to the biopotency of 0.3 µg retinol (vitamin A alcohol). Grass carotene has 0.025 of value of vitamin A on a weight basis.

[3] 1 iu vitamin E is the biopotency of 1 mg DL-α-tocopheryl acetate. Where 50 g supplementary fat of average composition is added per kg feed, the requirement rises to 45–50 mg α-tocopherol/kg, equivalent to 79–88 mg DL-α-tocopheryl acetate. Also see pp. 75–78 for working horses.

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napier, guinea grass, buffel, pangola, green panic, paragrass, kikuyu, setaria and probably some species of millet grass. Lucerne contains oxalic acid, but has a high Ca availability.);
- age of animal.

The major part of dietary Ca given to growing horses, is directed to bone calcification. Although maximum bone mineralization is reached by six years of age, 76% of maximum is achieved by one year (Lawrence *et al.* (1994). As bone growth is most rapid in the first year of life, the demand for dietary Ca is very pronounced during that time. There is speculation that horses predisposed to exercise-associated myopathies have a temporary failure in their ability to control intracellular Ca concentrations (Chapter 11); however there is no evidence that supplementary dietary Ca has any prophylactic influence.

Phosphorus

The bioavailability of plant P varies with the proportion present as phytates. Phytate P is digested by phytase (EC 3.1.3.8) present in the gut principally, or entirely, as microbial phytase. The digestibility varies between 0.25 and 0.35. The addition of yeast cultures to the diet increases phytate P use, presumably by stimulating microbial activity in the hind-gut. Excess dietary P interferes with Ca utilization. Savage (1991) and Savage *et al.* (1993b) found that diets containing four times the NRC (1989) estimated requirement for P in growing foals (but providing the requirement for Ca), caused lesions of dyschondroplasia without signs of NSHP (see Chapter 8). Cortical bone porosity was increased in the growing foals by the high P diet and the extent of osteoid-covered surfaces of cancellous bone decreased with time, despite a depression in growth rate. By contrast, a diet containing over three times

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Table 6.22 Characteristics of a good water source.

	mg/L
Ammonia (albuminoid)	<1.0
Permanganate value (15 min)	<2.0
Nitrite, N	<1.5
Nitrate, N	<1.0
Calcium	50–170
Lead	<0.05
Cadmium	<0.05
pH 6.8–7.8	
Total dissolved solids	<1000

the NRC (1989) estimated requirement for Ca had no adverse effect.

Sodium, potassium, magnesium, trace elements and vitamins

Table 6.21 gives the requirements for vitamins, trace elements, sodium, potassium and magnesium, supplements of which should be unnecessary if a commercially prepared mixed feed is used at recommended rates. Where this is not so, a proprietary mixture of trace elements and vitamins should be used, because in high concentrations such nutrients are toxic and the horse owner is unlikely to have facilities for their proper handling (There is concern in the EU over concentrated Co dust inhalation.) Sometimes a compounded feed intended to form the entire concentrate portion of the ration is used as a supplement to oats, diluting its effects. Thus, a pellet providing 4000 iu vitamin A/kg, mixed 50 : 50 with cereals and in turn fed 40 : 60 with hard hay will provide approximately 800 iu vitamin A/kg total diet. Dilution of this kind is frequently a cause of incorrect Ca : P ratios in rations.

Electrolytes

A discussion of electrolytes – Na, K and chloride – is given in Chapter 9, where problems of heavy exercise are tackled. The K and Mg needs of normal activity should be automatically met where good-quality roughage is available. The Na needs (Table 6.21) can be met by providing NaCl, for simplicity ignoring the contribution made by natural ingredients. Na comprises 40% of NaCl so 8.75 g salt/kg provides 3.5 g Na. Any excessive salt intake is satisfactorily counteracted by making clean drinking water always available (Table 6.22), excepting the restrictions to its use discussed in Chapter 4.

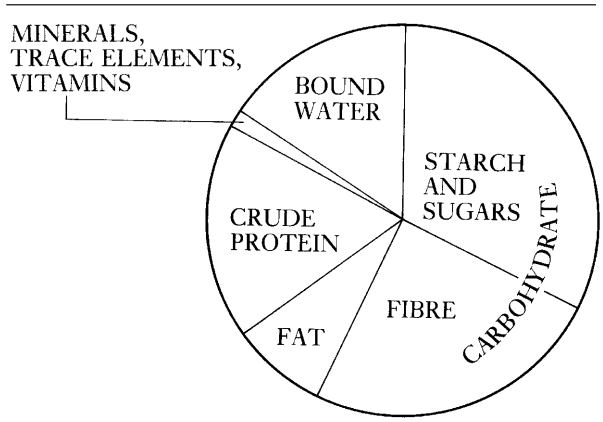


Figure 6.12 Principal components by weight of a stabled horse's diet (excluding drinking water).

Trace elements

The dietary allowances for trace elements, as distinct from those for the major minerals and electrolytes, do not change appreciably per unit of feed with a change in the function of the animal from growth to work or to various phases of reproduction. The secretion of iodide in mare's milk slightly increases her basal needs for I, but the margin given in Table 6.21 should satisfy all needs. The difference between this allowance and the toxic level (the margin of safety) for Se and I (the elements for which the difference in absolute quantities is least) is adequate in the hands of responsible individuals. However, it is unwise and potentially dangerous for the average horse owner to handle pure forms of trace elements.

Vitamins

The dietary allowances for the fat-soluble vitamins A, D and E per unit of concentrate feed (Table 6.21) again do not vary greatly per unit with a change in the function of the individual, bearing in mind also the capacity of the horse to store them. A reappraisal of the situation would be necessary if there were any radical change in the basic raw materials traditionally used for feeding horses, e.g., from dry cereal grains to root vegetables, or to high-moisture conserved cereals, the elimination of conserved pasture, etc. An example of this is given for vitamin E and breeding mares (Chapter 4). Appendix B gives examples of dietary errors encountered by the author in practice.

SIMPLE RATION FORMULATION

The principal chemical components of a stabled horse's diet are depicted in Figure 6.12. The amounts of protein,

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Table 6.23 Analytical DM values for raw materials used in Figures 6.11a–d for horses of mature weight 500 kg.

	DE (MJ/kg)	Protein (g/kg)	MADC (g/kg)	UFC (g/kg)
Grass hay	7.73	50	56 (41)	0.52
Soyabean meal	15.11	500.0	437	1.06
Oats	13.07	109.1	103	0.99

Ca and P needed per kg of ration are given in Tables 2.3, 3.12 and 6.7 and the levels in the concentrate–hay mixture can be calculated by multiplying their protein contents (g/kg) etc. (see Appendix C) by their proportions, and summing. Thus, for hard hay and oats:

$$(55\text{ g} \times 0.6) + (96\text{ g} \times 0.4) = 71.4\text{ g protein/kg mixture}$$

If the protein requirement is 100 g/kg, then the discrepancy of 28.6 g can be made good by substituting some soyabean meal (other suitable protein sources can be substituted in inverse proportion to their lysine contents) for some of the oats, but attributing the soya (or other protein) with a protein content that is the difference between its content and that of oats, that is:

$$440\text{ g} - 96\text{ g} = 345\text{ g protein (see Appendix C)}$$

Then the proportion of soya to include and of oats to remove per kg is:

$$28.6/345.0 = 0.083, \text{ i.e. or } 8.3\% \text{ or } 83\text{ g/kg mix}$$

For simplicity, the effects of this substitution on the energy, Ca and P contents of the mix can be ignored. A similar calculation using Appendix C, and detailed in Appendix A, will have been adopted for Ca and P, but the deficits can be made good by adding limestone flour and/or dicalcium phosphate without making any substitution as for soya/oats. A Ca source of this type will be essential, unless large amounts of lucerne or other legume forage are provided.

An approximate value for the DE content of a feed might be derived from its proximate composition, using INRA data above, or the equation derived by Pagan (1997):

$$\begin{aligned}\text{DE in MJ/kg DM} = {} & (0.0886 + 0.510\text{ CP} - 0.392\text{ ADF} \\ & - 0.160\text{ hemicellulose} + 1.974\text{ fat} \\ & + 0.851\text{ NSC} - 1.100\text{ ash})\text{ g/kg DM}, \\ & R^2 = 0.88\end{aligned} \quad (36)$$

NB: NDF = hemicellulose + ADF

Relationships derived by the author incorporate NSC and NDF values (see Chapter 2):

$$\begin{aligned}\text{DE kJ/kg DM} = {} & (17.5\text{ CP} + 36.5\text{ EE} + 17.6\text{ NSC} \\ & + 6.6\text{ NDF})\text{ g/kg}\end{aligned} \quad (37)$$

for diets in which diet CF is 110–130 g/kg DM

Comparison of the DE and NE systems for ration formulation

Exact comparisons between the DE and NE systems for protein and energy are not possible from tabulated data because a definitive description is not provided for the feeds selected, so the assumed identity of feeds between systems is not assured. This is particularly so for grass hay, the allowance for which is the same for the two systems. In deriving the comparisons, alternative values were tested for the hay and the same pattern of differences between the systems was shown to exist. Graphic comparisons are given in Figures 6.11(a-d), using the 'common currency' of extracted soyabean meal, oats and grass hay from Table 6.23 and requirements indicated by NRC (2007) and INRA (1984, 1990), ignoring mineral and vitamin needs.

The following conclusions seem to be justified in respect of the horse of 500 kg mature weight:

(1) The NRC (2007) system proposes higher protein requirements for maintenance, growth, exercise, gestation, lactation and for the breeding stallion, conclusions that concur with those of Meyer (1983b).
(2) The NRC (2007) proposes higher energy requirements for maintenance, and gestation and slightly higher energy requirements for lactation.
(3) Although some differences between systems, e.g. in level of growth rate, work or lactation yield, might have been assumed, the findings in respect of maintenance, gestation and the stallion are particularly noteworthy.
(4) These differences are in part in line with the conclusions of Martin-Rosset & Vermorel (2004) who

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compared the UFC system with those of NRC (1989). However, the present comparison emphasizes differences in protein requirements that are independent of differences between systems for CP and digestible protein requirements.

The NE system should ultimately lead to economies in the selection of roughages for horses, as analytical work enlarges the fund of data on raw materials. These data should predict the extent of precaecal digestibility and the energy yield during microbial digestion. The relative complexity of the NE system indicates that adjustments for a variety of husbandry practices are accommodated. However, direct comparison of the two systems under several practical environments is required to determine their comparative effects on work, lactation, pregnancy, growth, health, etc.

Somewhat less than 20% of the energy apparently digested is lost to the horse by excretion in equine urine and by expulsion as methane (CH_4) and H_2. Vermorel *et al.* (1997b) estimate that, with normal diets, horses lose about 4.2% of the GE of diets (8.0% of DE) in urine, but obviously greater amounts are lost with high protein diets metabolized as energy sources. Methane energy loss, owing to the activity of the cellulolytic and methanogenic bacterium *Archaea methanogenesis*, amounts to 1.95% (SE 0.45%) of GE in horses (Vermorel *et al.* 1997b) and it is assumed that a loss of energy amounting to about 30–65% of that lost as methane is lost as heat of fermentation (Webster *et al.* 1975). These losses differ amongst feeds, and losses during intermediary metabolism in tissues differ as between the nutrients, glucose, VFA, etc. The ratio ME : DE is approximately 0.85 for grass hays and 0.90 for mixed diets in horses (Vermorel *et al.* 1997a) and k_m% values vary from about 60–80 amongst feeds in the horse (Vermorel & Martin-Rosset 1997). Thus the efficiency of DE use for maintenance varies over a range of approximately 50–70% in the horse. The practical value of an NE system replacing a DE system depends substantially on this variation being reliably estimated amongst feeds that are available at economic prices.

Computers and diet formulation

(This section has been written with the assistance of John C. Dickins.) During the past 35 years, computers, with an array of programs of increasing power, have been used in the formulation of commercial feed mixtures. Programs of lesser complexity are also available for use on the home personal computer. For this development to be truly successful it is requisite that several needs are met. These are that:

(1) the nutrient requirements of the animal are reasonably well established;
(2) the nutrient contents and costs of a wide range of available feed materials are known and that the nutrient contents apply to the batch of each material available to the current user;
(3) the needs for, and nutrient and non-nutrient characteristics of, roughages to be used are accommodated in any formulation. (By subtracting the nutrient content of the forage portion of the ration from the total requirement per kg total feed, the remainder gives the required nutrient content of the concentrate portion.) This procedure makes assumptions concerning the appetite of the horse in kg DM/day, as discussed earlier (see 'Relationship of capacity for feed to body weight', 'Energy needs' and 'Appetite', this chapter);
(4) physical, non-nutrient chemical and microbiological characteristics of all feeds concerned are known; and
(5) the effects of the characteristics in (3) and (4) above on each class of horse are understood and allowed for during formulation.

Where this information is comprehensive it is possible to generate the formulae of mixtures that should be safe, nutritionally adequate, acceptable to the average horse and economical to use.

Principles of least-cost feed formulation

The number of chemical characteristics of each feed material that are accommodated in a formulation program depends on its power. The simplest takes into account protein (g/kg) and energy (MJ DE or MJ NE) and the more comprehensive include ranges of essential amino acids and fatty acid residues, minerals, trace elements, vitamins, oil, fibre (crude, neutral detergent, dietary etc.) and ash. Certain of these characteristics provide limited nutritional information, but their declaration is a statutory requirement on product labels within the EU (see Chapter 5). The computational technique is typically known as 'linear programming', as it is assumed that each additional amount of a nutrient contributed by one ingredient, substituting for another ingredient's nutrient, causes a similar quantitative response.

More complex, nonlinear models of feed formulation (e.g. integer and quadratic programming) are also accommodated in some computer packages, allowing, for

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example, for the calculation of a formula in whole units of the ingredient. The term 'least-cost' is used as the computation calculates a mix at minimum cost, consistent with formulation nutrient levels that are not less than the minimum requirements plus safety margins, that is, minimum nutrient constraints. The safety margins allow for variation in requirements of individual animals and deviations in ingredient composition from those assumed. Maximum and even minimum formulation levels for many ingredients are also set as ingredient constraints for reasons that are listed below.

The requirements of the formulation, expressed in terms of nutrient and ingredient constraints, take the form of a set of linear inequalities. For example, if x_1, x_2 … x_n represents the amounts (g/100 g) of different ingredients in the mix with protein contents a_1, a_2 … a_n (g/100 g), then the inequality:

$$a_1x_1 + a_2x_2 + \ldots a_nx_n \geq b \qquad (38)$$

expresses the requirement that the protein content of the feed should not be less than b. In addition to such constraints, a similar linear combination of x_i, with cost coefficients in place of the analytical values, represents the total cost to be minimized. The solution provides a set of x_i values (the formulation) which satisfies the constraints at minimum cost.

Feed manufacturers maintain, and regularly update, a database of analytical and cost information on a wide range of potential ingredients together with a set of nutrient and ingredient maxima and minima for each type of feed. Least-cost formulations are then produced automatically from this information periodically, as required. The computation also produces other values that give guidance on the sensitivity of the formula to small changes in the constraints, or costs.

Graphic example of linear programming

(An alternative simplified form of ration formulation is given in Appendix A.) The linear programming method of computing a least-cost formulation can be illustrated graphically for three ingredients, as shown in Figure 6.13, whereas the computer handles 30 or more potential ingredients. The two axes at right angles represent the percentages of the first two ingredients (x_1 and x_2) in the diet. The percentage of the third ingredient (x_3) in the diet is then determined, since all three inclusion rates must sum to 100.

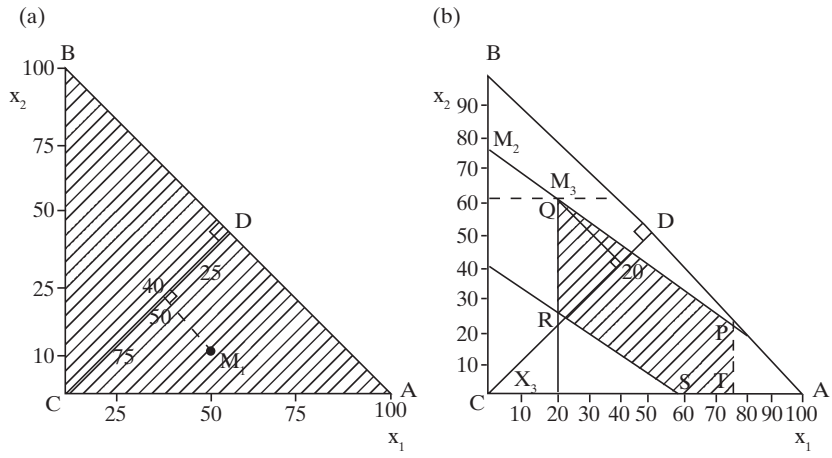
Each point within triangle (a) ABC represents a particular formulation, since the percentages of ingredients x_1 and x_2 determine the percentage of ingredient x_3. Negative inclusion rates are impossible, so admissible diets are restricted to the region defined by the triangle ABC (the hatched area), where the line AB represents mixtures of 100% ingredient x_1 and 0% ingredient x_2 to 100% of x_2 and 0% of x_1, excluding ingredient x_3. Point C represents 100% of ingredient x_3 and at point D the mix would contain 50% each of ingredients x_1 and x_2 and 0% of ingredient x_3. As indicated, a mix at point M_1 would contain 40% of ingredient x_3 and 50% and 10%, respectively, of each of the other two ingredients. Several of these mixes would be completely unacceptable; thus, ingredient and nutrient constraints are imposed. Reasons for such constraints include the following:

- A particular ingredient could contain an essential nutrient that is not accommodated in the program and so the ingredient's inclusion at a predetermined minimum level is necessary.
- Excessive amounts of some ingredients, e.g. molasses, reduce horse acceptance of the product, or may cause digestive upset. This upset can result from unacceptable concentrations of anti-nutritive substances, such as anti-proteases and glucosinolates (Chapter 5), or from excessive concentrations of some nutrients.
- Manufacturing rate and quality can be facilitated, for example, by the inclusion of minimum amounts of fat and pellet binders or exclusion of large amounts of abrasive ingredients.
- The provision of moderate amounts of each of several ingredients, rather than large quantities of one ingredient, is normally to be preferred. This policy should reduce the risk of problems arising when there are deviations in composition and quality of ingredients compared with the values attributed to the ingredient(s) in the program.

A linear analytical, or ingredient, constraint on the diet is represented by a straight line in Figure 6.13 graph (b) such that diets satisfying the constraint must lie on one side of the line. In this diagram, PQ and RS are constraints restricting the protein in the diet to the range 14–19%, and TP and QR are constraints limiting the inclusion of bran to the range 20–75%. Feasible diets must therefore lie within the region PQRST. The restriction of barley to a maximum of 60% does not affect the outcome in this example.

Diets of equal cost lie along parallel lines. In Figure 6.13 the costs assumed give a line with a negative slope of 0.5. By moving the line in the direction of the arrows, so reducing cost, we find the diet of least cost. This will lie on the boundary of the feasible region, normally at a

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Ingredient protein (%) cost

	Protein %	£/1000 kg
Bran, x_1	15	150
Barley, x_2	10	125
Beans, x_3	25	175

M_1: x_1 = 50%
x_2 = 10%
x_3 = 40%

Ingredient (%) constraint limits

	Minimum	Maximum
Bran, x_1	20	75
Barley, x_2	—	60
Beans, x_3	—	—

Protein (%) of mix

	Minimum	Maximum
Mix	14	19

M_2: x_1 = 0%
x_2 = 73%
x_3 = 27%

M_3: x_1 = 20%
x_2 = 60%
x_3 = 20%



Direction of decreasing cost of mix. In this example, with beans costing £175/1000 kg, slope = –0.5.

Figure 6.13 Derivation of a hypothetical least-cost mix of three ingredients with ingredient and protein constraints.

vertex. If no ingredient constraints had been applied, the mix M_2, containing 73% of ingredient x_2 and 27% of ingredient x_3, would have been obtained. As a result of applying the four constraints, the least-cost mix is that of M_3 at point Q, with the proportions 20%, 60% and 20%, respectively, of ingredients x_1, x_2 and x_3.

Figure 6.13 also shows that, in this example, the critical constraints determining the position of the optimum are the lines PQ (minimum protein requirement) and RQ (minimum bran requirement), whereas the outcome would be unaffected by changing the position of the bran and protein maxima. Increasing the cost of beans to

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Table 6.24 Procedure for calculating ingredient make-up of a simple ration using NRC and INRA systems.

Calculation	NRC DE system	INRA NE system
(1) Assess daily feed capacity of horse	kg/day, Figure 6.4	kg/day, Figure 6.4
(2) Assess daily energy requirement of horse	DE/day, Table 6.6	UFC/day, Table 6.15
(3) Multiply [(2).BW.100^{-1}]/(1)]	DE/kg	—
(4) Divide (2) by (1)		UFC/kg
(5) Calculate proportions of roughage : cereal	Appendices A and C	Appendices A and C
(6) Assess protein requirements per kg feed or per day	Table 6.7, CP	Table 6.15, MADC*
(7) Assess Ca and P needs per kg feed (90% DM)	Table 6.7	Table 6.7
(8) Calculate protein, Ca and P of mix	Appendices A and C	Appendices A and C
(9) Calculate soya, Ca and P additions necessary	Appendix A, Table 6.7	Appendix A, Tables 6.7 and 3.2
(10) Estimate other mineral and vitamin needs and add	Table 6.21	Table 6.21

*For relationship of DCP to MADC see equation (35), and to obtain MADC/kg feed divide daily MADC requirement by feed capacity per day (note: DM and 90% of DM feed estimates).

£250/1000 kg would reduce the slope of the cost line (make it more negative) to –0.8, and so change the optimum from M_3 to P. As the cost of beans increases past £200, the least-cost composition abruptly shifts from that at M_3 to that at point P. In practice, any such large change would be introduced in several small discrete steps, at intervals of time, to avoid possible implications regarding the health of the horse. The professional formulator will know the acceptable maximum size of each step for particular ingredient substitutions.

In conclusion, the practice of linear programming has improved the reliability and cost-effectiveness of commercial diets. It allows formulations to reflect economically the most recent established scientific evidence with minimum risk.

Procedure for calculating diets by the DE and NE systems

The sequence of calculations necessary in calculating a simple diet using the NRC DE and INRA NE systems is given in Table 6.24.

FEED TYPE, RATE OF INTAKE, APPETITE, FREQUENCY AND PROCESSING

Appetite

The regulation of appetite has been the subject of much research in the last decade. The discovery, in 1994, of the hormone leptin, and more recently of the hormones, ghrelin and adiponectin (see Chapter 2 and Glossary) have increased our understanding of appetite control. Appetite regulation is an immensely complex process involving many hormones, and both the central and autonomic nervous systems, cholecystokinin and other functions of the gastrointestinal tract; all modifying the hypothalamic response. Systemic mediators, such as TNFα, interleukins (IL) 1 and 6 and corticotropin-releasing hormone (CRH) influence appetite negatively; the mechanism probably explains why illness depresses appetite.

Reluctance or eagerness to eat can be assessed either as the amount of dry feed consumed consistently per day, or the amounts consumed in single meals. The ingesta derived from very coarse, poorly digested, long-fibrous feeds, if present in significant amounts, will be retained longer in the large intestine and depress the daily intake of DM. Although this is generally a disadvantage, it can occasionally help to contain the appetite of overfat animals. The proclivity of a horse to start eating energy-yielding feeds also depends upon the relative absence of gastric and intestinal ingesta and of blood metabolites, affecting release of the hormones listed above.

Under conditions of natural grazing, individuals will feed during 15 to 20 periods throughout the day. A series of small meals reflects not only the low capacity of the stomach, but also, probably the switching-off mechanism of digestion products. High caecal concentrations of VFAs, especially of propionate, have an immediate but small depressing effect on appetite by extending the interval between meals in ponies fed *ad libitum* and by reducing meal size, yet they have no sustained effect over 24 h.

An estimate of the horse's capacity for leafy hay and oats is given in Figure 6.4. Apparent loss of appetite,

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Table 6.25 Prediction equations for the UFC value of feeds for horses from chemical composition and digestible OM or DE content (from Martin-Rosset *et al.* 1994; Vermorel and Martin-Rosset, 1997). Reproduced from Martin-Rosset *et al.* (2006) with permission. © Elsevier.

Equations 33a–m	RSD	R^2
(a) Forages (n = 47)		
UFC = 0.825 – 1.090CF + 0.555CP	0.043	0.832
UFC = 0.568 – 0.650CF + 0.687CP + 1.804CC	0.031	0.922
UFC = –0.124 + 0.254CF + 1.330 DOM	0.012	0.988
UFC = –0.056 + 0.562CC + 0.0619DE	0.007	0.996
(b) Concentrates; raw materials (n = 51)		
UFC = 0.815 – 0.947CF + 0.0345CP + 0.582CC	0.060	0.931
UFC = 0.131 – 0.628CF – 0.282CP + 1.340DOM	0.041	0.967
UFC = –0.730 – 0.722CP + 0.572OM + 0.0941DE	0.033	0.979
UFC = –0.134 + 0.274CF – 0.362CP + 0.316CC + 0.0755DE	0.017	0.995
(c) Compound feeds		
UFCo = 1.326 – 1.937CFo – 0.135CPo	0.060	0.956
UFCo = 1.333 – 1.684ADFo – 0.096CPo	0.060	0.958
UFCo = 1.173 – 1.605CFo + 0.051CPo + 0.215STAo	0.043	0.976
UFCo = 1.181 – 1.397ADFo + 0.082CPo + 0.214STAo	0.040	0.978
UFCo = 1.219 – 0.852ADFo – 0.287NDFo – 0.857Llo + 0.034CPo + 0.207STAo	0.031	0.988

DM: dry matter; OM: organic matter; CF: crude fibre; CP: crude protein; CW: cell wall; CC: cytoplasmic carbohydrates (water-soluble carbohydrates + starch) in kg/kg DM; ADF: acid detergent fibre; NDF: neutral detergent fibre; STA: starch; CFo, CPo, etc. are expressed in kg/kg OM; DOM: digestible organic matter in kg/kg DM; DE: digestible energy in MJ/kg DM; UFC: horse feed unit (per kg DM); UFCo: horse feed unit (per kg OM).

frequently reflects lack of recognition of differences in the bulkiness and energy density of feeds when these are offered by the bowl (see Table 5.5).

Feed in an unacceptable physical condition, and more importantly with an unfamiliar or unattractive aroma, will also be a deterrent to the initiation of feeding. Stale feed, which has incurred the oxidation of many of its less-stable organic components, is unlikely to encourage a horse to eat. Occasionally, spices are used to camouflage inadequacies of acceptability, but they are to be discouraged as horses can become 'hooked' on these additives.

There is glib talk about the appetite of horses for particular feeds, but apparently they have a true appetite only for water, salt and sources of energy, and if given the choice, would not select a balanced diet. Hence, it is necessary to induce horses to consume mixtures most appropriate to their needs. Thirst will sometimes drive a horse to consume poisonous succulent plants, as a source of moisture, in an otherwise arid landscape. The appetites for water and for salt are interrelated. Thirst depends to a considerable extent on dehydration, or increased osmotic pressure of body fluids. When blood is hypertonic, horses will normally drink; if much salt has been lost through sweating, the body could be dehydrated, but the fluids can be hypotonic and thirst is then engendered by giving salt. The appetite for salt varies among horses – deprivation causes a greater craving in some than in others, despite a more uniform essentiality.

Rate of feeding

A too-rapid consumption of cereals and concentrates by stabled horses sometimes requires attention. Although the consumption of long hay is a relatively slow process, cereals and other concentrates are normally eaten first at a particular meal (there is a case to induce the consumption of some roughage first; see Chapter 2), but the form in which the concentrate is given can have an impact on the rate at which it is consumed. German workers (Meyer *et al.* 1975b) showed that 1 kg feed in the form of oat grain or a pelleted concentrate took horses weighing 450–550 kg about 10 min to consume, but meal took longer. Feeding time and the number of chewing movements

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were, however, increased by 3–100% if 10–20% of chopped roughage was added to the oats or concentrate. Finely ground meal mixes took longer to eat than crushed grains or pelleted feed; the addition of chopped roughage to fine mixes speeded up intake. Poor quality retarded consumption rate, suggesting a place for good quality barley straw in occupying the time of greedy horses.

Many horses develop vices, such as wood chewing, faeces eating (coprophagy) or, less frequently, cribbing (forced swallowing of gulps of air) (see Plate 11.1). Wood chewing and crib biting are responses to gastric inflammation and erosion. Such horses have a lower mean postprandial gastric pH with a wider pH range than is found normally (Lillie *et al.* 2003). The wider range could indicate variable biting, and so a variable salivary flow. The provision of an antacid supplement was shown to be associated with fewer gastric ulcers and a reduction in crib biting (Badnell-Waters *et al.* 2003). Boredom in isolated boxes is a contributory factor in vice initiation and the provision of long hay or good-quality straw does help to circumvent this, but does not eliminate its occurrence (see also 'Ailments related to housing, Weaving', Chapter 11).

Sound teeth enable horses to grind roughage to a small particle size, but decaying teeth, or molars with sharp points abrading the cheek and tongue (see Figure 1.1), encourage bolting and poor mastication with the consequences of rapid intake, choking and sometimes colic. Dental treatment is clearly indicated, together with the provision of coarsely and freshly ground cereals and roughage chopped in short lengths. These are given mixed together in a wet mash, which in turn can reduce wastage. The dampening of feed is, however, generally unnecessary except when large quantities of bran and beet pulp are given, in which case time for the thorough absorption of water should be allowed so that their true bulkiness is realized. Linseed also swells considerably on soaking and boiling; the latter, of course, is to be recommended, as described in Chapter 5. Where feeds are dusty, damping down lays the dust, so decreasing the likelihood of irritation to the respiratory tract in susceptible individuals. The damping of feed can also inhibit the segregation of its components. If feeds are too dry, minerals, for example, sift to the bottom and are left unconsumed in the manger.

Processing of feed

Preferred methods for processing raw cereal grains are coarse grinding, cracking, rolling, crimping (passing between corrugated rollers), steam flaking and micronizing. The overall objective of each is to improve digestibility and acceptability, but the last two may accelerate the onset of satiety during a meal. The processing of oats and barley is generally difficult to justify if the costs exceed 5% of the cost of the raw material, but it is necessary shortly before feeding for the small grains milo and wheat (see also discussion on laminitis, Chapter 2). Steam flaking or micronizing tends also to extend shelf life, destroys most heat-labile anti-nutritive substances and increases the proportion of starch digested in the small intestine when intake rates are high. Normal cooking is, however, inadequate for the destruction of some mould toxins found in poorly harvested crops (see also Chapter 5).

Feeding frequency and punctuality

Horses, like most other animals, are creatures of habit, and their reactions are in part affected by an inner clock. Thus, the wise groom sticks to regular feeding times, week in and week out. With regular exercise, metabolic upsets and accidents can be avoided, and damage to stable doors through chewing and kicking can be decreased. Designs for feed mangers and water bowls recommended by the French Ministry of Agriculture are shown in Figures 6.14 and 6.15.

Cereal costs and their energy contents

Cereals, and more particularly oats, of low bushel weight provide less DE per kg than cereals of higher bushel weights. Digestive upsets are minimized when changing from one feed, or cereal, type to another, by ensuring that the DE, or NE, intake does not simultaneously rise. A bowl of an average sample of oats provides only 56% of the DE of a similar bowl of average wheat (see Table 5.5). As a result of their bulkiness, oats tend to be a safer feed than the other cereals although, with careful and expert feed management, no differences in this respect should arise. Therefore, in assessing the feeding value of the various cereals, account should be taken of the purchase price per unit of DE or NE (see Appendix C).

Traditional feeds

In the past, many stables gave their horses grass hay, oats and a little cooked linseed. Before the advent of the tractor-drawn baler, hay was probably made for horses with greater care than it is today, when the horse is unlikely to reign supreme in its claims to conserved forage and grazing privileges. In terms of nutrients, this diet now leaves a little to be desired, but the adult horse has a capacity to adapt to its culinary shortcomings, because it takes a considerable time for a large non-productive animal to be depleted of essential nutrients. Many of these nutrients are made good where summer grazing is available; nutrients

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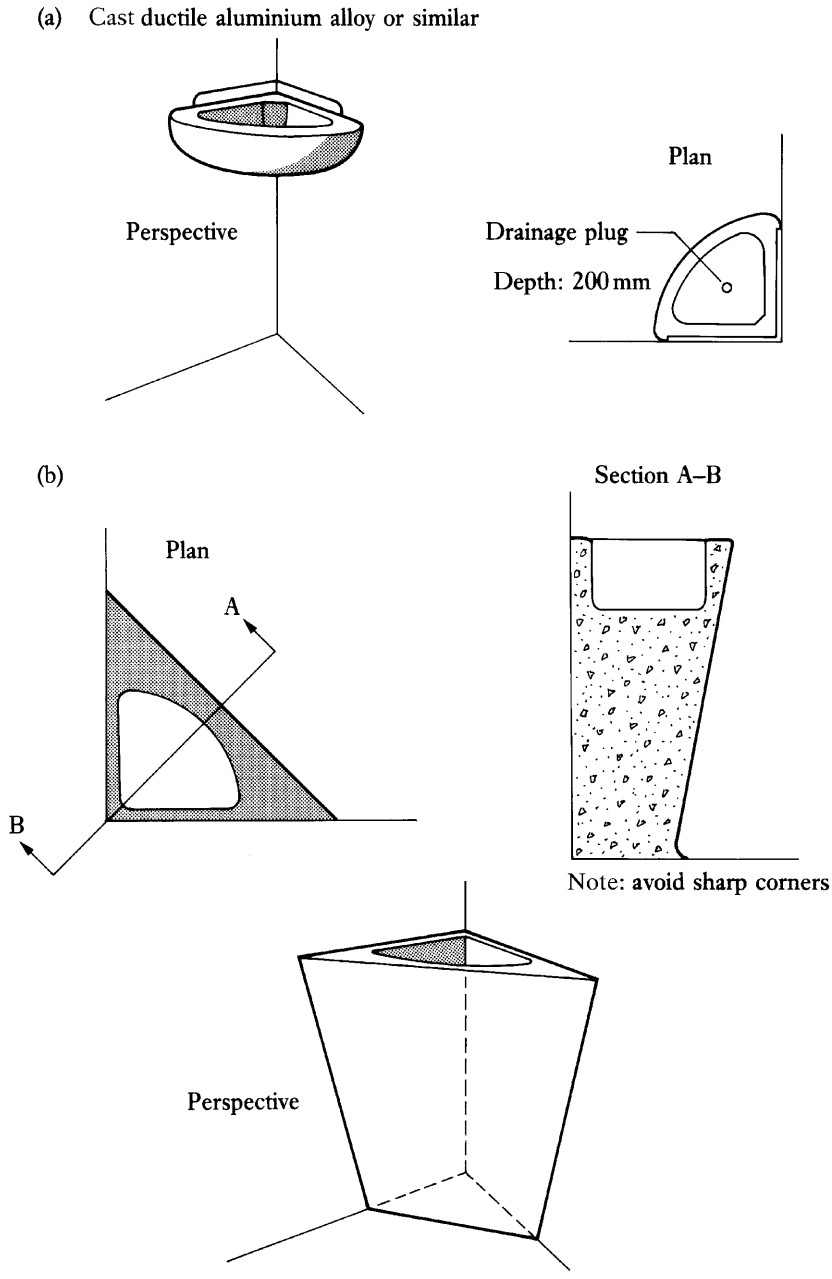


Figure 6.14 Designs for feed mangers: (a) unbreakable manger; (b) manger embedded in concrete (Ministère de l'Agriculture 1980).

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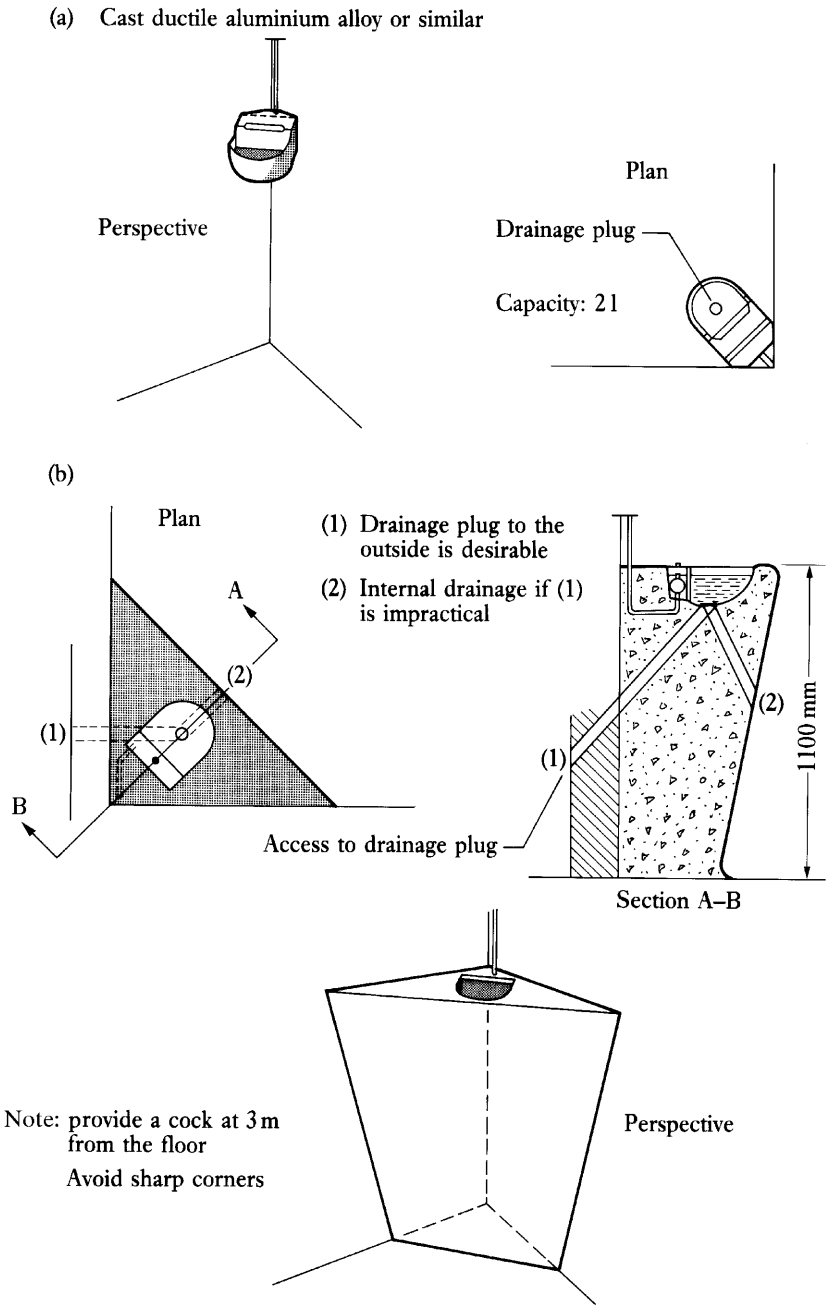


Figure 6.15 Designs for water bowls: (a) unbreakable water bowl; (b) water bowl embedded in concrete (Ministère de l'Agriculture 1980).

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provided by productive pasture in summer will serve as a reservoir for two months of the ensuing winter, but not generally until the spring grass again refurbishes losses. Lactation of the mare and early growth of young stock rely traditionally almost entirely on the availability of grazing, any deficiencies of which are soon revealed.

A diet of oats, hay and linseed is alien to the natural inclination of a browsing animal so that there is no good reason why other equally strange feeds should not be used if economies can be achieved and deficiencies rectified. There are now better protein sources than linseed and there are oils less prone to peroxidation than linseed oil, as these other oils contain added antioxidants (also see Polyunsaturated acids, Chapter 5). Stabilized forms of groundnut, soya, sunflower, rape and corn oils are widely available today. By understanding the necessity for protracted periods of adaptation, and by recognizing the differences in bulk and energy density among the various cereals, the careful feeder has a wide choice of feeds. The principal advantage of oats is to the novice feeder; but the traditional feeder knows by long experience how many bowls of oats a performance horse will consume without refusal. Without careful weighing, the quantities of alternative mixtures required can be misjudged and overfeeding or underfeeding can result.

Complete diets

Many horses and ponies receive diets based on compounded nuts and long hay or haylage. In normal circumstances this hay should be preferred to a source of ground fibre if the quality is good and the price acceptable. Compounded feeds described as ***complete*** pellets are for consumption by adult horses required to carry out moderate work where no other feeds are given. These pellets have the advantage of providing a reasonably balanced diet, relatively free from dust, for horses subject to respiratory irritation and allergy. The absence of long fibre is likely to cause a higher incidence of vices, such as wood chewing and coprophagy, but these annoyances are undoubtedly preferable to the exacerbation of a respiratory problem. Rationing is simplified by the constant bulk and energy densities of the feed and storage space is minimized, but some problems of boredom require ingenuity to overcome.

SHELF-LIFE OF FEEDS, FEED CONTAMINANTS AND GOVERNMENT REGULATIONS

The grinding of cereals disrupts their structure and increases the surface area exposed to the atmosphere, so deterioration proceeds continuously, retarded only by the presence of natural antioxidants. Staleness detracts from feed acceptability. Legislation permits the addition of limited amounts of synthetic antioxidants to products for consumption by animals. The most commonly used of these are butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT) and ethoxyquin (EU legislation permits 15 chemicals to be used as antioxidants and included in animal feeds (Schedule 3, UK Statutory Instruments 2000 No. 2481, The Feeding Stuffs Regulations 2000 and European Commission Regulation (EC) No. 2316/98; Anon 1995c). These substances are safe, have the effect of extending shelf-life and acceptability of feeds, and cause no infringement of the Rules of Racing.

Some nutrients are particularly susceptible to destruction by light. Hence, concentrated feedstuffs should be covered during storage, as the products of light destruction are also harmful to the stability of certain other nutrients. Atmospheric oxygen brings about the gradual destruction of fat-soluble vitamins and unsaturated fats. Among the more critical water-soluble vitamins, thiamin (vitamin B_1) and pantothenic acid are somewhat unstable. The loss of these nutrients and other unstable compounds detracts from the value of feedstuffs. However, whole grains in an uncrushed or unground form remain relatively stable for several years with only a slight decline in feeding value, although this, of course, assumes good storage, freedom from pests, moisture levels of less than 135 g/kg (the lower the better), and absence of cracked and moulded grains. Discolouration of cereal grains may indicate only superficial microbial damage during ripening, as observed when one compares good-quality Scots oats with their counterparts from Canadian or Australian sources. However, discolouration can be an indicator of more profound internal damage through fungal infection, which will seriously impair feeding value and stability in storage. Thus, bright and bold grains are to be preferred in the absence of other information. Further aspects of storage have already been discussed in Chapter 5.

Dressed seed corn or cereal grains that have been exposed to toxic pesticides should not be used for feeding to horses, although several pesticides leave quite harmless residues.

US government regulations relating to the horse's environment

The US Environmental Protection Agency (EPA) promulgated regulations under the Clean Water Act in 2003 that update the definition of Animal Feeding Operation and certain of these sites designated as Concentrated Animal

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Feeding Operations (CAFO). These regulations have serious implications for the American (and other) horse industries. Any Animal Feeding Operation that discharges pollutants directly into the waters of the US, or has animals in direct contact with such waters, may be designated as a CAFO. Each horse is counted by the EPA as two cattle units. Any large unit (described in the regulations) has a duty to seek coverage under a National Pollution Discharge Elimination System permit. In this provision a CAFO is required to contain all the runoff from a 25-year, 24-h storm event.

The environmental problems that are to be contained are principally:

- waste nitrogen (N) and phosphorus (P) (mainly derived from urine and faeces),
- faecal bacteria, and
- land erosion resulting from over-grazing.

Urea is converted to ammonia, a volatile toxic gas (associated with upper respiratory disease) and this can be oxidized to nitrous oxide (N_2O), a potent greenhouse gas. Also nitrates can run off into water courses (EPA has set an upper limit of 10 mg nitrates/L in ground water). P is stable and in the form of phosphate, in a soluble form, is responsible for toxic algae blooms in streams and rivers. These blooms deplete waters of dissolved oxygen, resulting in death of marine and aquatic life. Problems of this nature are known to be widespread in western countries and China, whereas many African soils are grossly deficient in P for plant growth (author). In parts of the US it is illegal to apply manure, or P fertilizers, to the land. There are also concerns over Na, Cu and Zn concentrations in water. In parts of the US the Na content of surface waters are too high for them to be used as drinking water (NRC 2007). The faecal bacteria of concern are certain serotypes of *Escherichia coli* e.g. 0157:H7, *Listeria, Salmonella* and some species of *Clostridia*. A study estimated that 80% of horses harbour *Salmonella* and 20% are active shedders (NAHMS 2001).

The NRC (2007) points out, that feeding diets that are excessively high in protein and minerals, particularly P, Cu and Zn, puts the US industry at significant risk for regulation by federal agencies in the future.

STUDY QUESTIONS

(1) What is the sequence of actions and decisions to be taken on introducing a rationing system and meeting the nutrient requirements of a stud and a riding stable?
(2) What are the merits and demerits of the two energy systems discussed: the NRC DE system and the INRA NE system?
(3) What should be the characteristics of a satisfactory feed store?

FURTHER READING

Martin-Rosset, W., Andrieu, J., Vermorel, M. & Jestin, M. (2006) *Livestock Science*, **100**, 53–69. Routine methods for predicting the net energy and protein values of concentrates for horses in the UFC and MADC systems.

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7
Feeding the Breeding Mare, Foal and Stallion

You should wean your foals at the beginning of winter, when it beginneth to grow cold, that is about Martinmas, or the middle of November, and wean them three days before full moon, and hang about their necks upon a piece of rope seven or eight inches of the end of a cow's horn, to catch hold of them upon occasion, after which bring them all into your stable, with racks and mangers pretty low set.

S. de Solleysel 1711

THE OESTROUS CYCLE AND FERTILITY

The natural season for maximum breeding activity in both the mare and the stallion in the UK is from April to November, but the breeding season can be shifted by artificially changing daylight length and by manipulating the diet. A move to the southern hemisphere can, of course, have comparable effects. During the season, the normal mare expresses consecutive oestrous cycles approximately 22 days long; within each cycle there is a period of oestrus of varying intensities that lasts on average six days. The fertility of the oestrus is low at the start of the season, but the creation of large follicles that ovulate and generate corpora lutea can be stimulated by the extension of day length and dietary adjustment, as the reproductive activity of both mares and stallions is under the influence of daylight length. A survey of 1393 TB mares visiting 22 stud farms in Newmarket, UK, revealed that mares were mated 1.88 times until diagnosed pregnant at 15 days after ovulation. Of the mated mares 89.7% remained pregnant by day 35 of pregnancy. Ultimately 82.7% of the mares surveyed gave birth to a live foal at term (Morris & Allen 2002).

Mares in poor body condition in the autumn and winter are more likely to become acyclic, i.e. to remain in anoestrus, than mares with higher body condition scores (BCS). Deep anoestrus is characterised by minimal ovarian follicular development- hypothalamic secretion of gonadotropin-releasing hormone (GnRH) is low and the pituitary content of luteinizing hormone (LH) is at a basal level. Periods of darkness are associated with a rise in plasma concentrations of melatonin, a protein hormone produced by the pineal gland that inhibits secretion of GnRH. During the winter, melatonin is secreted for a considerable length of time each day (Domingue *et al.* 1992). Increasing ambient photoperiod decreases the duration of melatonin exposure, leading to an increase in GnRH production. This triggers secretion of follicle-stimulating hormone (FSH) which induces follicular development. For early ovulation, starting photoperiod therapy at the beginning of December is more effective than starting at the beginning of either November or January in the northern hemisphere. Exposure of anoestrous mares to an artificial photoperiod regimen for 60–70 days remains the most effective with 15–16 h of light and 8–9 h of darkness (McCue *et al.* 2007). The seasonal pattern can be advanced by about 21 days. Thus, the extension of daylight, an increase in the plane of nutrition, and possibly a rise in ambient temperature during December will stimulate the onset of normal cycling three weeks earlier in the first months of the year.

The voluntary activity of athletic mares, with a box and paddock, obeys circadian rhythms. Their greatest daily activity occurs during the vernal equinox and the lowest during the winter solstice. Their acrophase (peak of the rhythm) occurs at the middle of the photoperiod, during both summer and winter days (Bertolucci *et al.* 2008). This situation is consistent with the observation that two-year-old Welsh Mountain pony colts ate greater quantities of a complete pelleted diet daily when the daylight length was extended to 16 h (220 lux) cf. 8 h (220 lux) (Fuller *et al.* 1998, 2001). The cyclic changes in growth rate of the body

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and pelage (coat) were synchronous with those of feed intake. However, interestingly, these three related physiological responses were delayed by 5–8 weeks following change in the day length. Cyclic hormonal responses to seasonal photoperiod occur in both sexes. Argo *et al.* (2001) determined that in prepubertal pony colts changes in pelage, plasma prolactin, FSH and testosterone concentrations were under the influence of photoperiodic mechanisms, but that LH release might be blocked by immaturity or active suppression. High fertility tends to coincide naturally with the flush of grass in late spring. Successive oestrous periods will be of increasing fertility in healthy, barren or maiden mares, and individuals that are increasing in body weight are more likely to conceive. Therefore, by starting with a lean individual in November and December, this objective is more likely to be achieved. It has been suggested that forcing* barren mares in December and January enhances the probability of twins, yet if early conception is desirable such a procedure is obligatory. Jordan (1982) noted that no reduction occurred in conception rate among pony mares losing 20% of BW during gestation, but allowed to gain weight during lactation. Henneke *et al.* (1981) reported that mares in this condition at foaling had reduced conception rates, longer postpartum intervals and more cycles per conception. Conception rates of mares in good condition at foaling, but who lost weight during lactation, were as good as those of mares in good or thin condition at foaling that maintained or gained weight in lactation.

Endocrine mechanisms are gradually being pieced together. Energy intake is important for ovulation and embryonic development but less so for foetal growth (Meyer 1998). Healthy mares given an energy restricted diet during gestation can produce foals of normal weight at birth and at 30 days of age (Sutton *et al.* 1977b). An excessive energy supply favours twins, whereas restriction prior to parturition can induce premature birth. Protein restriction seems to be important solely when the mare is extremely under-fed. Mares with a BCS of only 3.0–3.5 during the anovulatory period (autumn and winter), experienced longer and deeper anoestrus than those with a high BCS. This response was accompanied by lower plasma leptin, **insulin-like growth factor-1** (IGF-1) and prolactin concentrations (Gentry *et al.* 2002). Subsequent work by this group indicates that there is an interaction between leptin secretion and the secretion of the hormones, insulin, T_3 and growth hormone (Cartmill *et al.* 2003). Berg *et al.* (2007) recorded the *pre-* and *post-partum* concentrations of leptin, IGF-1 and TSH in blood and milk of mares and in blood of their offspring. Plasma IGF-1 concentrations in 1880 TB horses in three continents were stable, compared with GH concentrations. The mean IGF-1 concentration in gonad-intact males was 336 ± 5.6 ng/mL, 303 ± 3.2 ng/mL in females and 302 ± 3.2 ng/mL in geldings (Noble *et al.* 2007). Leptin is a protein hormone produced by adipose tissue that influences hypothalamic mechanisms regulating appetite and energy balance. Feeding acutely leads to an increase in plasma leptin concentration (hyperleptinaemia: >10 ng leptin/mL plasma) which is also positively correlated with BCS (Huff *et al.* 2008), whereas withholding feed leads to a rise in plasma growth hormone (Buff *et al.* 2002, 2005). The hyperleptinaemic condition persists for mares of BCS 7, under *ad libitum* pasture and native grass hay in an outdoor paddock (Storer *et al.* 2007). Huff *et al.* (2008) also found lactating mares on pasture had the highest leptin concentrations, including some with BCSs of 4–5.5. Rebreeding success was not affected by leptin classification.

Energy restriction appears to result in a rapid decrease in plasma IGF-1 concentration in non-pregnant mares (Sticker *et al.* 1995), pointing to a mechanism for the effects on fertility. When BCS was changed from 7 in September to either 8 or 3.0–3.5 in January, by feed control, Gentry *et al.* (2001) in Kentucky, found that a low score caused deep anoestrus, whereas the high-BCS mares continued to cycle. There was a higher prolactin response to **thyrotropin-releasing hormone** (TRH) and higher LH response to GnRH by those with high scores. Mares entering the autumn in a moderately fat, or fat, condition, and then given an energy-restricted diet, become anovulatory within 12 weeks. These mares responded to GnRH administration by a reduced LH response and to TRH administration by a reduced prolactin response (Gentry *et al.* 2002). Powell *et al.* (2003) observed a prolactin response in geldings to TRH only in horses receiving large amounts of concentrates. TRH interacts with growth hormone (Pruett *et al.* 2003; Hardy *et al.* 2003) and a TRH challenge causes a rise in circulating thyroid hormone in all horses.

Mares entering the breeding season in a thin condition cf. those in a moderate condition require more cycles for conception, and indeed, fat mares have no impairment to reproductive efficiency. Furthermore, mares foaling and lactating in a thin condition experience a longer interval from parturition to the second postpartum oestrus (Henneke *et al.* 1984). Breeding mares should be maintained at a BCS of 5–7 which should not be allowed to fall below 5 during gestation or lactation. A score of 8 does not impair

*The mares are given extra concentrate feed and extra daily light to bring foward the commencement of the oestrous cycle.

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or improve reproductive efficiency (Cavinder *et al.* 2005; NRC 2007).

The inclusion of cholesterol-rich animal fat in the diet of pregnant mares could stimulate circulating levels of LH and fertility by providing a precursor of steroid hormones (Hagstrom *et al.* 1999). Diets based on starch, compared with those rich in fat and fibre, should cause reduced insulin sensitivity in barren mares and increase the risk of overt insulin resistance (Hoffman *et al.* 2003a). Subsequently this group (Hoffman *et al.* 2003b) concluded that the maintenance of an ideal body condition in geldings, and the avoidance of meals rich in sugar and starch, would decrease the risk of insulin resistance. In contrast, for reproduction, where glucose demand and hormonal relationships are different, no difference in glycaemic effects have been noted between fibre and fat *vs.* starch and sugar diets (Williams *et al.* 2001a, b).

Pituitary and pancreatic hormone release is influenced by infusion of amino acids (Sticker *et al.* 2001) with the potential for control of reproductive cycles. The dicarboxylic amino acids, aspartic and glutamic acids, each given at a dose of 2.855 mmol/kg BW in water, caused the release of growth hormone; whereas the diamino-mono-carboxylic acids arginine and lysine, each at 2.855 mM/kg BW in water, caused the release of prolactin and insulin. The glutamate receptor agonist *N*-methyl-D,L-aspartic acid (1 mg/kg BW in water), stimulated growth hormone and gonadotropin release. Whether spring grass exerts a hormonal influence by this mechanism is as yet unclear. Nevertheless, it is evident that photoperiod and BCS influence the onset of cycling in barren mares, but the optimum composition and energy content of diet for mares of good condition have still to be established.

Freedom in the quantities of feed consumed seems possible where it is composed largely of hay. Doreau *et al.* (1990) offered pregnant mares of Anglo-Arab and Selle Français breeds *ad libitum* intakes of a 90 : 10 mixture of hay and concentrates from four weeks before foaling until five weeks after foaling. The hay was either of poor or high quality. The intakes for the poor and high quality mixtures before and after foaling averaged respectively 11.1 v. 12.4 kg DM and 18.6 v. 21.1 kg DM. The BCS of the mares receiving the lower quality diet was relatively poor at foaling and the consequential shortage of energy and inadequacy of protein (342 g, 426 g and 579 g MADC/day, see Chapter 6) during the last month of pregnancy, the first week of lactation and weeks 2 to 5of lactation, respectively, led to poorer foal growth rates to 5 weeks of age without having influenced birth weight. Although the mares were initially in similar condition, the outcome could have depended upon the extent of energy reserves of the pregnant mare. If lower foal growth rates are acceptable, adequate performance has been observed by Micol & Martin-Rosset (1995) under upland grazing conditions amongst French mares of heavy breeds (700–800 kg BW) such as Breton, Comtois and Ardennais, which lose 17–25% of their body weight between foal weaning and the next foaling. Of this loss, 12–14% is products of conception and 5–10% loss of body mass, indicating the mares' reserves had been liberally incorporated in foal tissues. Nevertheless, pregnant mares should be kept fit but not fat as this reduces foaling difficulties and provides greater freedom for controlling milk secretion during lactation.

The foal heat occurs within 14 days of foaling and subsequent heats occur at 22-day intervals in unbred mares. The recommended energy intakes of lactating mares are given in Tables 6.6 and 6.15, but it has not been clearly established whether these are the optimum rates for maximum fertility of the foal heat and subsequent oestrous periods. Milky mares, nevertheless, have a greater tendency to resorb fertilized eggs at first oestrus. This could be the reason for a putative association between overfeeding during the last three months of pregnancy and a reduced subsequent fertility.

Experience with dairy cows might suggest that if mares are fed too liberally through gestation and given inadequate feed during lactation they are more prone to a fatty liver condition, known to reduce fertility in the dairy cow. Observation of horses and ponies shows that various stresses during late pregnancy and early lactation, accompanied by an inadequate and impoverished diet, predispose the mares to an extreme metabolic upset associated with loss of appetite, abnormal reactions, diarrhoea, hyperlipidaemia and eventual death. **Hyperlipaemia** is a milky TAG discolouration of the plasma, following a period of negative energy balance, which is associated with excessive hepatic lipid accumulation and a high death rate. Mares in late gestation or early lactation having raised energy demands are especially susceptible. Lack of adequate food stimulates the release of hormone sensitive lipase causing lipolysis in adipose tissue and a rise in plasma NEFA that undergoes hepatic conversion into TAG. This is either stored or circulated causing hepatic and renal damage (Watson 1998). Any imposed weight reduction in obese pregnant mares should therefore take place before the last three months and, for preference, the fatness should be corrected before breeding is instigated. This is achieved by providing the mare with good-quality hay, but no concentrated feed.

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The BCS system was developed in Quarter Horse mares to assess subcutaneous fat at six body areas (neck, withers, shoulders, ribs, loin, and tail head). In TB geldings BCS can be determined with fewer body areas (Suagee *et al.* 2008). A BCS derived only from the neck, shoulders, ribs, and tailhead was found to accurately predict the mean BCS derived from the six body areas (see Chapter 11 for obesity and insulin resistance).

GESTATION

The gestation period of the mare commonly lasts for 335–345 days but can continue for up to a year. The period depends, in part, on the month of breeding. In the northern hemisphere, early-bred mares (those conceiving before the end of April) normally have a gestation period of between 350 and 365 days. Those bred in May normally foal after 340–360 days, and those bred in June and July foal after 320–350 days. The critical factor is day length during the last three months of pregnancy, as when the photoperiod was artificially extended to 16 h in late gestation of Quarter Horse mares (Hodge *et al.* 1981), the gestation period was shortened by 11 days and the interval from parturition to first ovulation decreased by 1.6 days, in comparison with mares subjected to natural day length.

Where diets are grossly imbalanced in terms of protein and minerals, especially Ca and P, the foal will be adversely affected at birth and reduced milk yield and infertility will ensue. A marginal Ca intake retards foetal growth and iodine and selenium status have considerable effects on fertility of the mare and the viability of the foal (Meyer 1998). A deficiency, or excess, of either I or Se depresses embryonic development and viability of the foal (author's observations and see 'Trace elements', Chapter 3). An extreme and chronic deficiency of vitamin A prejudices embryonic development and the ovarian cycle (Meyer 1998), whereas reports on specific effects of β-carotene have been contradictory.

Protein and energy requirements

Definitive statements on protein and energy requirements for breeding mares (see Chapter 6 for feed requirements of pregnant mares) are not as yet possible for two reasons:

(1) A large well-fed mare has considerable reserves of energy and protein on which she can draw during pregnancy if daily intakes fall below recommended levels.
(2) A reduction in intake generally seems to induce economies in metabolism so that deficiencies are partly offset.

Clearly the mare is capable of considerable adjustment to a variety of situations. However, in extremes, excesses or deficiencies of energy will lessen her reproductive efficiency. During winter or summer the ribs of a mare should not be seen, but should be detectable by touch with no appreciable layer of fat occurring between them and the skin. The condition of the over-fat mare can be improved by gradually reducing the cereal component of the ration while the protein and mineral mixture is maintained at previously determined levels of intake. Meyer (1983b), concluded that the mare should be 18% above normal weight before parturition to achieve a high fertility after parturition.

Pregnant mares are normally kept on pasture. Australian workers (Gallagher & McMeniman 1988) established that grass/legume pastures in southeast Queensland could support the nutritional requirements of TB brood mares by providing DE intakes of 68.0 and 91.7 MJ/day; this figure is similar to the NRC (2007) recommendation for months 9–11 of gestation (Tables 2.2 and 6.6) and digestible N intakes of 91.2 and 138 g/day during mid and late pregnancy, respectively. The first figure is less than, but the second figure is similar to, the requirements proposed by NRC (2007), see Table 2.3.

During periods of inadequate energy intake epinephrine secretion increases fat mobilization from body reserves leading to a rise in plasma NEFA concentrations in blood. In the normally fed human subject receiving three meals daily, plasma NEFA concentration decreases after each meal and rises before the next meal (Frape *et al.* 1997a, 2000). In the horse, plasma NEFA begins, and continues, to rise from 4 h following an afternoon meal in adult mares given 50% of their protein and energy needs for maintenance in two daily meals, and not in those given 100% of those needs (Sticker *et al.* 1995). This measurement provides a useful means of assessing energy status of the horse.

PARTURITION

In the 24 h before birth of the foal, the mare should be fed lightly with good-quality hay and a low-energy cereal mixture including bran, or proprietary horse and pony nuts (10–11% protein, 3% oil, 14–15% CF; see Table 7.1), with access to restricted quantities of warm water. The first feed after parturition can be a bran mash and the second can include some bran with small quantities of good-quality proprietary stud nuts (16–17% protein, 3% oil, 8% CF; Table 7.1) or a cereal protein mixture. Obese mares tend to be less active and so poorer muscle tone can lead to birth difficulties and delayed expulsion of the placenta,

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Table 7.1 Composition of foal milk replacer, stud concentrate mixture and horse and pony mix to be given with hay and water.

Foal milk replacer (see footnote for mixing[1])	(%)		Stud mixture[2] (%)	Horse and pony mix (%)
Glucose	20.0	Oatfeed	—	25.0
Fat-filled powder (20% fat)	5.0	Oats	46.0	33.0
		Wheat bran	15.0	20.0
Spray-dried skimmed-milk powder	40.0	High-protein grass-meal (16% crude protein)	15.0	10.0
Spray-dried whey powder	32.7	Extracted soya-bean meal	15.0	4.0
High-grade fat[3]	1.0	Molasses	5.0	5.0
Dicalcium phosphate	1.0	Fat[3]	1.0	1.0
Sodium chloride	0.2	Limestone	1.0	0.9
Vitamins/trace elements[4]	0.1	Dicalcium phosphate	1.1	0.5
		Salt	0.75	0.5
		Vitamins/trace elements[4]	0.1	0.1
Total	100.0		100.0	100.0

[1]Disperse in clean water at the rate of 175 g/L (for 2 days after colostrum at 250 g/L). Also can be pelleted and mixed with stud mixture as a weaning feed for orphan foals.

[2]This mixture is satisfactory as a creep feed and postweaning diet. However, a mix specifically for young weaned foals to be fed with grass hay could to advantage contain an extra 5% soya-bean meal replacing 5% oats.

[3]High-quality tallow and lard, including dispersing agent. Stabilized vegetable oil could alternatively be added at time of mixing.

[4]To provide vitamins A, D_3, E, K_3, riboflavin, thiamin, nicotinic acid, pantothenic acid, folic acid, cyanocobalamin, iron, copper, cobalt, manganese, zinc, iodine and selenium.

which should be passed during the first hour after birth. The rate of concentrate feeding up to day 10 should be restricted in order to avoid excessive milk secretion and digestive disturbance in the foal. However, inadequate amounts of energy contribute to the metabolic abnormalities outlined in 'The oestrous cycle and fertility', this chapter; see also recommendations in Tables 6.6 and 6.15.

Perhaps 5% (Rossdale & Ricketts 1980) or 10% (Jeffcott *et al.* 1982c) of foals are lost through perinatal mortality, including stillbirths and postnatal deaths. Of these, significantly more are male. Birth weight is a crucial factor in determining the prospects of foals and, despite the influence nutrition can have on this, size of the dam is a major controlling influence. An acceptable minimum weight must therefore depend on the breed and the purpose intended for the individual offspring. In TBs, an early rapid growth rate is normally expected and required for work at an early age, so foals of less than 35 kg at birth probably should not be kept. The total weight of twins approximates that of large single births with a mean in the region of 55 kg for TBs, implying that it is practical to retain only the heavier of the two.

Acquired immunity in the foal

The mare must pass adequate passive protection to her foal through the colostrum, and she should have been situated in the foaling area for, at the very least, 2 and preferably 4 weeks before foaling, so that she will confer some immunity to the strains of microorganism peculiar to her environment, for example, those causing scours, joint ill and septicaemia. Newborn foals will normally first suck within 30–180 min of birth. Colostrum is rich in protein (particularly immu-noglobulins), DM and vitamin A. If foals are deprived of colostrum, an injection of about 300 000 iu vitamin A is in order. Immunoglobulins do not pass through the dam's placenta and can be absorbed efficiently through the intestinal wall of the foal only during the first 12–24 h of life. The major causes of colostrum deprivation

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in the foal are premature birth and delayed suckling, small-intestinal malabsorption, premature leakage of milk through the teats or death of the mare. The immunoglobulins are concentrated by the mare in her udder within the last two weeks of gestation, when their level in the mare's serum falls. There is, therefore, a selective concentration of this protein fraction in the mammary gland.

If the foal is suckled normally, the concentration of the immunoglobulin fraction in the colostrum 12–15 h after birth is only 10–20% of the initial value. It is known that the protein content of mare's colostrum is around 19% during the first 30 min after parturition, but by 12 h the level falls to about 3.8% and after eight days it reaches a fairly constant level of 2.2% (Ullrey *et al.* 1966). The foal absorbs γ-globulin as intact undegraded molecules throughout the first 12 h of life. At birth the foal is agammaglobulinaemic but as it responds to the colostrum, its serum γ-globulin rises over 12 h to 8 g/L serum (Jeffcott 1974b,c,d). Amounts of specific antibodies so acquired by the foal's blood decline from 24 h of age; by three weeks the values are halved and by four months the titre of antibodies provided by the mother is barely detectable.

The foal's own system for building active immunity in the form of autogenous γ-globulins first provides detectable products at two weeks of age in the blood of colostrum-deprived foals and at four weeks in those reared normally. By four months of age the γ-globulins have attained adult plasma concentrations. Up to this age, therefore, the foal is more susceptible to infection than an adult in the same environment, particularly when it has received an inadequate quantity of colostrum, or colostrum at the wrong time.

If the mare has ejected much of her colostrum before foaling, it will be necessary to give the foal colostrum from another mare, preferably one accustomed to the same environment or, failing this, cow's colostrum, rather than milk. Commercial sources of cow's colostrum are now available (see 'Bovine colostrum', this chapter). Wohlfender *et al.* (2009) reported that colostrum administered by stomach tube cf. bottle was a risk factor for infectious and systemic diseases and diarrhoea amongst neonatal TB foals. This predisposition could be a reflection of weakness, or immaturity, disabling the ability to suck. After 18 h, the colostrum has little systemic immune value, although it does have some beneficial local effects within the intestinal tract.

A simple field test has been developed in which the turbidity of plasma is assessed following the addition of zinc sulphate; the results correlate well with concentrations of blood globulin in foals indicating whether sufficient antibodies have been absorbed in the neonatal period. This subject is discussed further under the section on orphan foals ('Blood plasma by parenteral or oral dosing', this chapter).

Neonatal problems

Hygiene is generally outside the scope of this book, yet the importance of cleanliness in the foaling area cannot be overemphasized. It is essential that the foal receives colostrum to provide it with protective passive immunity. The consumption of excessive quantities of milk can overload the digestive capacity of the foal and the milk then becomes a substrate for rapid bacterial growth in the intestines. This situation can precipitate diarrhoea, despite the consumption of normal quantities of colostrum.

The aetiology and treatment of foal diarrhoea involves several factors (Urquhart 1981). That caused by excessive milk intake and lactose intolerance is treated by decreasing or terminating milk consumption. The onset of so-called 'foal-heat diarrhoea' occurs 7–10 days post partum, frequently coinciding with the mare's first oestrus, but no correlation has been established between this form of diarrhoea and total solids, fat, protein or ash concentrations, bacterial count or oestrogenic activity of mares' milk. Of foals sampled by Sgorbini *et al.* (2008), 49% showed foal-heat diarrhoea and 28.3% were positive for yeasts. *Candida famata* was isolated nine times; *Geotrichum* spp., six; *Saccharomyces cerevisiae*, five; *Cryptococcus laurentii*, four; *Rhodotorula* spp., three; *Zygosaccharomyces* spp., two; and *Candida colliculosa*, *Candida globosa*, and *Candida pelliculosa* once each. Fifteen of 92 (16.3%) foals with heat diarrhoea and 11 of 92 (11.9%) asymptomatic animals yielded positive samples for yeasts. The authors concluded that equine gastrointestinal tract did not appear to be colonized by yeasts during the first 15 days of life, and yeasts are not involved in foal-heat diarrhoea.

Blood glucose concentrations of 29.1% of 515 foals admitted to hospital were within the reference range (4.2–7.3 mmol/L) at admission, 36.5% were hyperglycaemic, and 34.4% were hypoglycaemic (Hollis *et al.* 2008a). Foals with blood glucose concentrations <2.8 mmol/L (50 mg/dL) or >10 mmol/L (180 mg/dL) at admission were less likely to survive to discharge. These foals also had higher maximum and lower minimum blood glucose concentrations in the first 24 h of hospitalization, and higher blood glucose at 24 h and 36 h. Hypoglycaemia at admission was associated with sepsis, a positive blood culture, and systemic inflammatory response syndrome.

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There are two problems concerning the feeding of the foal unrelated to microbial disease, haemolytic icterus and passing the meconium.

Haemolytic icterus

The foal's blood differs immunologically from that of its dam and, on rare occasions, the foetus reacts with the dam's immune system, causing the production by the mare of isoantibodies to the foal's red cells. These antibodies are transmitted to the colostrum and the suckled foal absorbs sufficient to initiate a considerable destruction of red cells, precipitating an anaemia and jaundice, known as *haemolytic icterus*. In severe cases, the foal's urine will be discoloured with haemoglobin. If a mild attack is detected before icterus has occurred, the foal should not be allowed to nurse from its dam for 36 hours. Where the mare has previously produced foals with the condition, she can still carry similar antibodies. In this case the foal should automatically be given colostrum from another mare at the rate of 500 mL every 1–2 h for three to four feeds, followed by milk replacer until 36 h, when it can be returned to its dam. In the meantime, the mare should be milked out by hand.

If the problem is anticipated, blood samples can be taken from the foal and the red cells allowed to settle. The abnormality causes red cell haemolysis, producing pink plasma rather than the normal straw colour. In these circumstances the foal may be severely anaemic and red cells from the mother can be infused slowly into a vein after removing the plasma and suspending the red cells in physiological saline. For preference, however, the source of red cells should be three or four geldings that have not previously received transfusions so the risk of immunological reactions is minimized.

A simple procedure has been proposed as a means of precluding damage *before* colostrum is taken by foals that are considered to be at risk. One drop of umbilical blood is mixed with four drops of saline and five drops of mare's colostrum on a clean microscope slide, checking after several minutes for agglutination reactions.

Passing the meconium

At birth, much of the large intestine, including the caecum and rectum, contains a substance, the meconium, which is normally completely voided within the first two to three days of life. Suckling usually sets up a reflex promoting defaecation of this material. If this does not occur, the normal passage of colostrum and milk can become blocked so that the gases formed during their fermentation cause distension and pain to the foal. It may then go off suck, act abnormally, crouch, lift its tail and flex its hocks in an effort to pass the offending material, or roll over in pain. Eventually a yellowish milk dung reaches the rectum, the meconium is cleared and the signs subside.

The problem is treated conservatively by administration of a lubricant through a stomach tube, plus one or two enemas of soap and water, or liquid paraffin, and the injection of pain-relieving drugs. If the foal goes off suck for an extended period it should be given a fluid feed by stomach tube or appropriate intravenous solutions. Intravenous feeding of glucose and an isotonic electrolyte solution (see Table 9.2) is a life-saving procedure in cases of severe enteritis with consequent dehydration. In normal circumstances the foal will eat a quantity of its mare's faeces. In so doing it introduces beneficial microorganisms into the intestinal tract, which compete with pathogens present in the general environment.

LACTATION

At any given stage of lactation, the composition of mare's milk is remarkably similar among the various breeds of horse. The composition changes rapidly during the first days of lactation and then more slowly (Figures 7.1–7.4 and Tables 7.2 and 7.3). Milk contains about 2 MJ gross energy/kg. Eight TB and two Standardbred mares receiving a diet of concentrates and hay achieved yields of 16, 15 and 18 kg/day (3.1, 2.9 and 3.4% of BW/day, or 149, 139 and 163 g/kg $BW^{0.75}$) at 11, 25 and 39 days postpartum, respectively (Oftedal *et al.* 1983). Doreau *et al.* (1991) reported that, during weeks 0–5 of lactation, primiparous French Anglo-Arab mares, weighing 522 kg after foaling, produced less milk and of a lower fat content than was produced by multiparous mares (14.6 *vs.*16.6 kg/d and 16.5 *vs.*20.2 g/kg, respectively). This difference was associated with a lower DM intake during pregnancy and lower plasma NEFA concentration during pregnancy and lactation for the primiparous mares. French draft (Breton, Comtois) mares, weighing 726 kg, yielded 20 kg/day in the first week, rising to 27.5 kg/day by week 8 (Doreau *et al.* 1992) (Table 7.4).

The NRC (2007) concludes that average milk yields can be expressed by:

$$Y = a\left(d^{0.0953}\right)e^{(-0.0043d)},$$

where Y = yield in kg/day; a = 0.0274287 × BW in kg; and d = day of lactation.

Thus, a 500 kg mare will produce: 16.3 kg/day in month 1, 14.9 kg/day in month 3 and 12.2 kg/day in month 5. However, milk yields are markedly influenced by the mare's innate ability, by feed consumption during the

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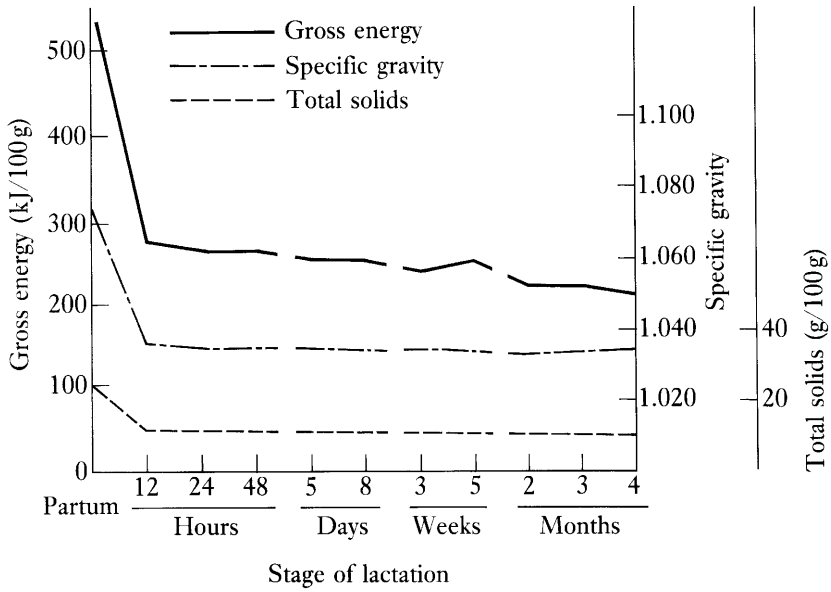


Figure 7.1 Changes in specific gravity and concentration of gross energy and total solids in mare's milk at various stages of lactation (after Ullrey *et al.* 1966).

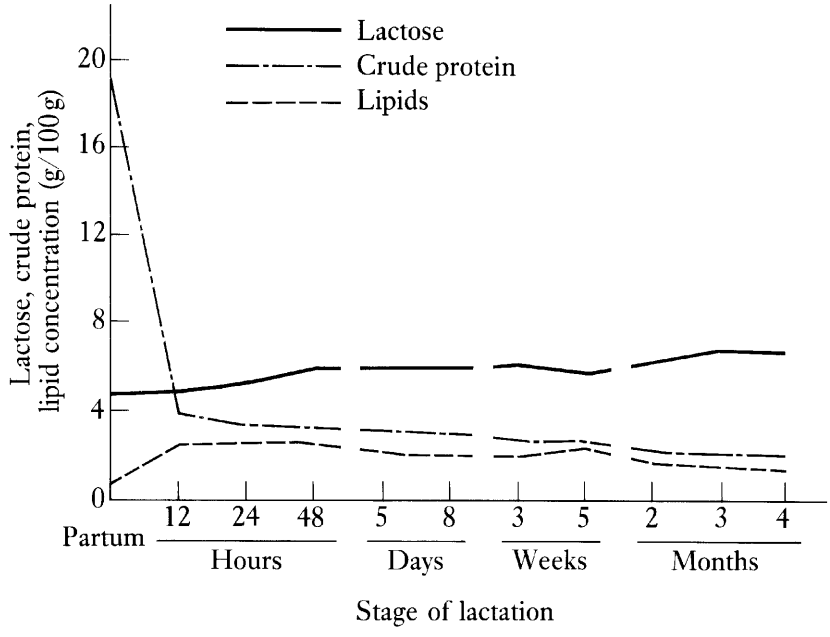


Figure 7.2 Changes in concentration of lactose, crude protein and lipids in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

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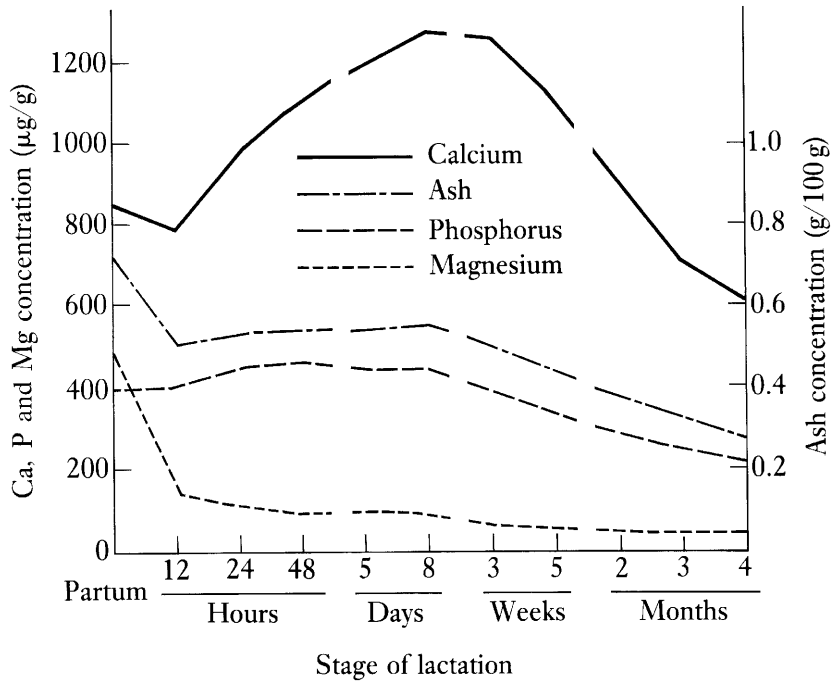


Figure 7.3 Changes in concentration of ash, calcium, phosphorus and magnesium in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

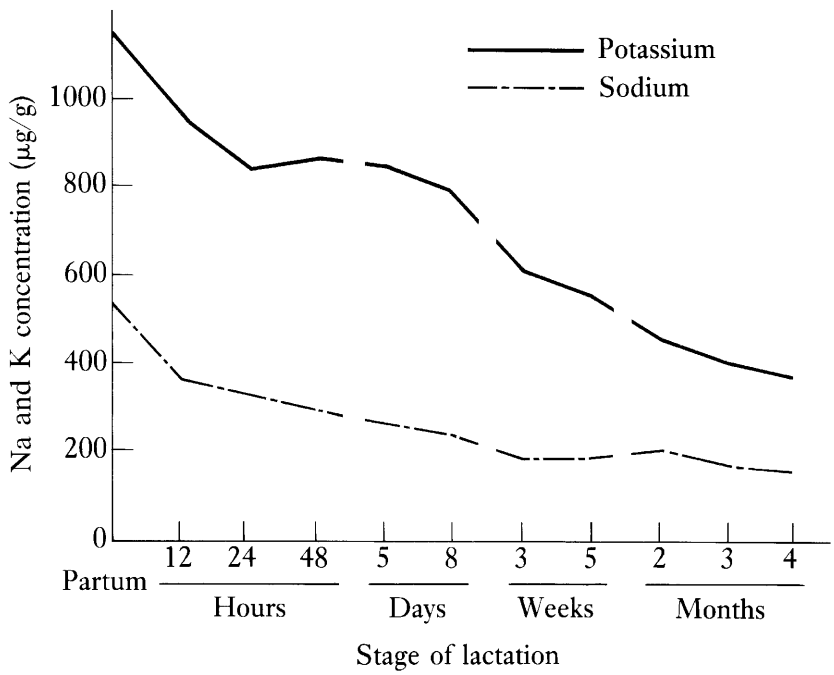


Figure 7.4 Changes in concentration of potassium and sodium in mare's milk at various stages of lactation (Ullrey *et al.* 1966).

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Table 7.2 Nutrient content of milk in Quarter Horse, TB, Dutch Warmblooded Saddlebred horse mares and some other breeds (Bouwman & van der Schee 1978; Gibbs *et al.* 1982; Oftedal *et al.* 1983; Schryver *et al.* 1986; Doreau *et al.* 1988; Saastamoinen *et al.* 1990; Martin *et al.* 1991).

	Dutch and some other breeds			Quarter Horse	TB
	Initial	28 days	196 days	2–150 days	24–54 days
Total solids (g/kg)	130	100–112	105	105	105
Gross energy (MJ/kg)	2.5	1.9–2.3	1.8–2	—	—
Fat (g/kg)	27	11–13	7	13–12	13
Protein (g/kg)	33	17–20	18	21–16	19
Ash (g/kg)	5.3	2	2.8	3	4
Calcium (g/kg)	1.2	0.7	0.8	0.8–0.6	0.9
Phosphorus (g/kg)	1	0.4	0.5	0.36–0.21	0.6
Lactose (g/kg)	58	66	66	—	69
Magnesium (mg/kg)	—	90	45	65–43	68
Potassium (mg/kg)	—	700	400	580–370	120
Sodium (mg/kg)	—	225	150	190–160	180
Copper (µg/kg)	—	450	200	—	200
Zinc (µg/kg)	—	2500	1800	—	2000

Table 7.3 The composition of mare's milk: amino acids* g/kg milk (Wickens *et al.* 2002).

	g/kg milk		g/kg milk
Arginine	1.17	Methionine	0.54
Histidine	0.56	Phenylalanine	0.90
Isoleucine	1.33	Threonine	1.22
Leucine	2.50	Valine	1.65
Lysine	1.70		

*These are considered to be the indispensable dietary amino acids, excepting the omission of tryptophan.

Table 7.4 Typical milk yields (kg/day) by mares of various body weights during the 1st to 25th weeks of lactation.

Weeks	1–2	4–5	5–12	20–25
Quarter Horse (500 kg)	10	14	10	—
TB, Standardbred (494 kg)	12–16	14–16	18	—
Dutch Saddlebred horse (600 kg)	14	16	19	11
French draft (726 kg)	20	25	27	—

latter stages of pregnancy, and, more importantly, by water availability (Table 7.5) and intake of energy and nutrients during lactation. Experimental work with Quarter Horse and TB mares has shown that a reduction in the energy intake to 75% of that recommended for lactation by the NRC (1978) does not lead to a parallel decrease in foal weight at 75 days old (Banach & Evans 1981a,b). Undoubtedly, the thriving mare has considerable capacity to adapt, within limits, to a restricted diet; but normal yields assume an ample water supply.

Saddlebred horses indicated that suckling frequency averaged 103 times/day in the first week, falling to 35 times/day by week 10, and on each occasion the foal suckled for 1.3–1.7 min (small amounts taken frequently are unlikely to cause digestive disturbances). At birth the foals weighed 57 kg and the weight doubled in the first 37 days of life. Suckling TB and Standardbred foals have been shown to take in daily milk DM equivalent to 3.1%, 2.1% and 2% of BW at 11, 25 and 39 days postpartum, respectively, when weight gain averaged 1.14 kg/day (Oftedal *et al.* 1983). The comparable daily intakes of gross energy were 39, 32 and 37 MJ, respectively.

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Table 7.5 Water requirements in the stud.

(a) Water provided by pasture herbage (kg) per kg herbage DM and total (including herbage) minimum water needs (kg) per kg feed DM consumed.

Water content of herbage per unit DM		Water requirement of horses per unit DM consumed	
Spring growth	4	Last 90 days of gestation	3
Dry summer	2.5*	First 3 months of lactation	4
Mild winter	3	Breeding stallion	3
		Weaned foal	3
		Barren mare	2

*When 'burnt' and bleached the proportion of water can be as low as 0.15–0.3.

(b) Minimum supplementary water requirements (L) per mare daily, assuming average milk yields.

Live weight of mare: (kg)	200	400	500
Last 90 days of gestation:[1]			
in stable	12.7	22.3	26.4
on pasture[2]	0	0	0
First 3 months of lactation:			
in stable	27.7	41.8	49.6
on pasture[2]	7.3	10.9	12.3

[1]The breeding stallion's needs are similar to those of the pregnant mare.

[2]These amounts will be insufficient for mares on parched pasture in environmental temperatures exceeding 30°C, where shade should be provided.

Effects of dietary energy, protein and urea on milk yield

Energy intake can be voluntarily modified to affect milk yield and composition. The voluntary food intake of draft broodmares was less when equal quantities of tall fescue hay and concentrates were given, compared with 95% hay and 5% concentrates (Doreau *et al.* 1992), causing differences in the protein and fat contents and in the fatty-acid profiles of the milk (Table 7.6). The milk from the high concentrate diet was richer in C18:2(*n*-6) (linoleic) and poorer in C18:3(*n*-3) (α-linolenic) acids. Thus, increased fibre and inadequate energy contents of feed increase the fat and protein contents of milk, but decrease yield. The effect on fat is similar to that in ruminants, but dietary fat content influences equine milk production. Breeding mares given 5% animal fat in their concentrate consumed less feed during gestation, their milk had a higher fat content and their foals' growth rates tended to be higher than that of mares given the unsupplemented diet. Reproductive performance was unaffected (Davison *et al.* 1987, 1991).

Dietary protein quality is known to affect the protein content of mare's milk. Glade & Luba (1987b) gave lactating mares 1.55 kg moderate quality protein/500 kg BW/day, or the same amount but with half provided as soya protein. This increased the protein content of the milk at seven days from 25.3 to 33.2 g/L. A difference persisted until the fourth week of lactation. Plasma methionine and lysine concentrations were higher in foals nursing the soya-supplemented mares and their withers height at seven weeks was greater. Poor pasture would therefore benefit from supplements of both good-quality protein and starch. Urea supplementation of mares given low-protein diets increases plasma and milk urea concentrations and reduces feed intake with adverse effects on milk intake and foal growth (Martin *et al.* 1991). Dietary energy source and amino acid intake within normal limits influence milk yield and composition as well as foal performance. The dietary essential amino acid requirements have now been estimated. The ratios of essential amino acids in mare's milk relative to those of lysine are in close agreement with the ratios in muscle, with the exceptions of histidine, leucine and valine (Wickens *et al.* 2002; Stamper *et al.* 2005). The NRC (2007) estimates the dietary requirements for milk production are 2.62 g digestible lysine/kg milk, so if a 500 kg mare produces 3.2% of her BW as milk/day she will require 41.9 g digestible lysine/day above maintenance. The dietary requirements above maintenance for other digestible amino acids (g)/kg milk, are: arginine, 1.81; histidine, 0.86; isoleucine, 2.04; leucine, 3.85; methionine, 0.84; phenylalanine, 1.39; threonine, 1.88; valine, 2.54 (Table 2.3). Stamper *et al.* (2005) reported that arginine, isoleucine and leucine were lower relative to lysine in milk replacers compared with mare's milk, indicating possibilities for improving the protein quality of milk replacers.

Mineral composition of milk

The mineral composition of TB mare's milk is given in Table 7.7 (other values are given in Table 7.2). The DM content was found to decline sharply from 12% in the first week to 10.8% in the third, and then slowly to 10.2% by the 15–17th weeks of lactation (Schryver *et al.* 1986).

Parturient paresis

Some high-yielding dairy cows suffer from a condition known as milk fever, or parturient paresis, which is

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Table 7.6 Effect of proportions of hay to concentrates in French draft mares (726 kg) on lactation (Doreau *et al.* 1992).

	Hay : concentrates 95 : 5	Hay : concentrates 50 : 50
DE (MJ/kg diet)	9.3	12.9
DCP (g/kg diet)	129	142
NE (UFC)*	0.65	0.89
MADC (g/kg DM)	74	100
Feed intake (kg DM/day)	22.9	21.4
Milk yield, week 4 (kg/day)	23.4	26.4
Fat, mean weeks 2 and 4 (g/kg milk)	14.7	11.6
CP, mean weeks 2 and 4 (g/kg milk)	26.3	24.4

*See Chapter 6, French INRA system.

Table 7.7 Mineral composition of mare's milk – electrolytes (mmol/L) and Cu and Zn (μmol/L) (Schryver *et al.* 1986).

Weeks postpartum	Ca	P	Mg	K	Na	Cu	Zn
1–2	33.3	29.2	4.64	17.0	9.4	12.1	44.4
3–4	27.9	22.6	3.66	13.0	7.5	7.5	35.2
5–6	23.1	19.5	2.96	10.0	8.0	6.8	30.6
7–8	21.6	19.0	2.47	11.5	6.5	5.0	29.1
9–14	20.7	18.2	2.18	9.8	6.4	2.7	27.5

probably caused by a sudden draining of blood calcium into the milk after parturition without an equivalent mobilization of bone calcium. A successful treatment entails giving cows a low-calcium diet two to four weeks before parturition and then providing a diet relatively rich in calcium during lactation. A regulated but high dose of vitamin D given 8–10 days before parturition has also been beneficial in some instances, but the dose has to be carefully calculated to avoid a gross and toxic excess. This uncommon clinical sign in the horse is associated with **hypocalcaemia**. Lactation **tetany** was formerly commonly observed in draught horse mares. Mares grazing lush pasture and with a heavy milk flow are particularly prone to tetany. For other causes see Chapters 3, 9, 10, 11 and Glossary. Pony and horse mares with a history of tetany (cf. paresis in ruminants) associated with depressed blood calcium in lactation might well benefit from being given a diet containing 1.5–2 g Ca/kg and approximately 3000 iu vitamin D/kg throughout the last two weeks of gestation. It is difficult to predict parturition with sufficient accuracy 10 days before foaling and therefore single doses of large amounts of vitamin D at that time cannot be recommended. The diet should, of course, be adequate in other respects and during lactation the total diet of such mares should contain 5–6 g Ca/kg. Horses that are suffering from this form of tetany are slowly given calcium gluconate intravenously while cardiac action is continuously monitored.

Grazing and drinking behaviour in the postnatal period

Grazing, lactating Welsh pony mares were shown (Crowell-Davis *et al.* 1985) to spend about 70% of their time feeding, while their foals at one week of age spent 10% of their time (excluding nursing) grazing, rising to about 50% at 21 weeks. Foals tended to eat when their mothers were feeding, which was of greater intensity early in the morning and the evening. Generally, the entire herd moved to water sources together, and the frequency of this increased with increasing environmental temperature to 35°C. However, foals under three weeks of age did not drink, and half the foals were not observed to drink before weaning (see also 'Water requirements', Chapter 4).

The growth rate of the suckling foal reflects the rate of milk secretion of its dam. Among environmental influences on milk yield, the quality of pasture and safe, free

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Table 7.8 Protein (g/L), neutral fat (g/L) and glucose (mmol/L) concentrations and alkaline phosphatase (ALP) activity (King Armstrong Unit) in the blood serum of TB foals (Sato *et al.* 1978).

Days from birth	Total protein	Albumin	Globulins	Neutral fat	Glucose	ALP
0	45.8	26.3	15.3	0.36	3.07	100
10	54.8	26.5	27.6	1.18	8.04	76
20	56.2	25.4	29.6	1.04	7.51	57
50	53.6	24.3	29.2	0.62	7.16	62
90	56.0	26.3	30.0	0.90	6.06	50
120	60.8	31.1	29.4	0.45	6.08	45

water availability are major influences, especially in hot weather. The supplementary feeding of lactating mares, which takes place in the stable at night or on the pasture, is another influence.

Blood biochemical values of the foal

Normal blood characteristics of the foal are given in many texts, and the subject is referred to in Chapter 12. The serum values given in Table 7.8 indicate that the neonatal foal is deficient in globulins, and this must be rectified by consumption of colostrum before any other feed. Serum alkaline phosphatase activity decreases with age, reflecting the relative decrease in the rate of bone growth with increasing age, and emphasizing that mishaps in proper bone development are established before, or shortly after, birth. Blood glucose and fat are in low concentrations at birth, reflecting the low energy reserves of the neonatal foal and its inability to maintain body warmth in a cold environment unless colostrum and milk are provided.

Creep feeding of the foal

Foals will start to nibble hay and concentrates at 10–21 days of age. If the milk supply of the dam, or the amount of grass, is inadequate, the provision of creep from this time may well enable a normal growth rate to be achieved. However, the principal objective of creep feeding is to accelerate the anatomical and physiological maturation of the GI tract of the foal so that, ultimately, weaning presents no particular stress or hazard, and digestive disturbances caused by abnormal fermentation of ingesta are prevented.

Where the mare secretes minimal quantities of milk, a creep feed based on dried skimmed milk provided from about two weeks of age is recommended. The composition of this feed should be changed gradually to that of a stud nut (16–17% CP, 3% oil, 8% CF) or a growing foal diet (17–18% CP, 3% oil, 7% CF) (see Tables 7.1 and 8.6) from 10–14 weeks of age. The use of milk pellets as a creep feed before weaning is contraindicated as it defeats the prime objective.

Control of growth abnormalities

Foals that are growing well with mares on pasture have no particular need for additional dry feed until two months before weaning, say at two to three months of age. Here the functions are to compensate for a waning milk production in the dam, to redress the effects of a decline in pasture quality and, probably more importantly, to accustom the foal to the dietary regime it must expect after weaning. The feed should be a concentrate of the type shown in Tables 7.1 and 8.6, on which the foal will be maintained throughout the forthcoming winter and spring.

Supplementary creep feeds should be restricted in quantity to 0.5–0.75 kg/100 kg BW. Such a restriction will give a measure of control over the incidence of growth-associated ailments including epiphysitis and contracted tendons (flexural deformity; Plate 7.1a–b). Where there is evidence of either of these, a restriction of growth rate, imposed by cutting the supplementary feed and by reducing the mare's feed for a period of 3–4 weeks, should not prejudice ultimate mature size if carefully regulated. The extent of restriction must depend on how serious the problem is. Foals that stand high on their toes at birth should be exercised regularly and allowed to grow at a submaximal rate if the condition is to be contained. Overtly contracted tendons at birth are relatively untreatable and possibly result from intrauterine malpositioning. If therapy is possible, splints or extension shoes are used and the foals are exercised regularly, with restricted access to feed until the abnormality is satisfactory (see also Chapter 8 for the aetiology of long bone growth abnormalities).

The vertical growth of normal foals is very rapid throughout the first 3–4 months of life. Access to creep feeders should be controlled by regulating the width of the

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(a)

Plate 7.1 Chronic contracted tendons in a yearling showing enlarged fetlock joints and upright stance. Later improvement was achieved by desmotomy of the superior check ligaments. Before surgery (a) and after surgery (b).

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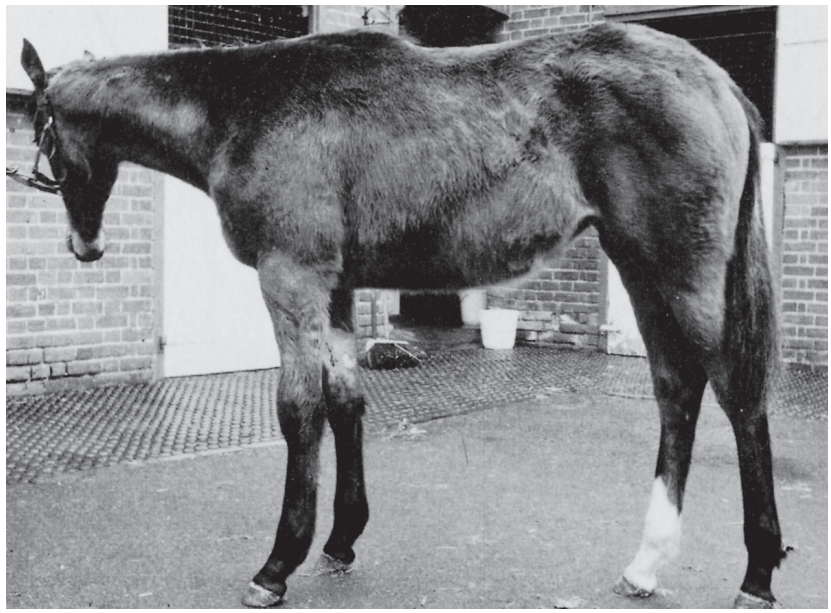
(b) 

Plate 7.1 *Continued*

entrance rather than by restricting its height. Foals of breeds with mature weights of 550 kg can be weaned easily when consuming nearly 1 kg of creep feed and 0.5 kg hay (or equivalent grass) daily, by which time the foal should be in excess of 140 kg. These restrictions normally ensure that growth will not falter during the first postweaning week and by the end of the second week the rate of gain will be at least 1 kg/day in healthy stock. Several days before weaning it is appropriate to remove the mare's daily concentrate allowance and access to hay and pasture can be restricted.

Epiphysitis (Plate 7.2a–c), probably more correctly called metaphysitis, is not uncommon in faster growing, larger, fine-boned foals of TB, Saddlebred or modern Quarter Horse breeding. It is encountered particularly in the fetlock joint at the end of the metacarpus and in the 'knee' joint at the distal end of the radius. Where it is slight, it will probably correct itself, but, where severe, supplementary feeds should be restricted to good-quality roughage, the milk intake should be limited and the animal should be boxed until the worst of the 'bumps' subside. A restriction in the rate of weight increase allows joint maturation to continue without the stress of excessive pressure on the joints. Light exercise must then be undertaken daily and the normal feeding regime gradually reinstated. Exercise is, however, damaging in severe epiphysitis, and in this case no analgesics should be used. Problems of this nature can arise in less than a week and are complicated by angular deformities in one limb together with epiphysitis in the opposite limb. Successful treatment is contingent on immediate action. Attention by the farrier to the hooves should allow small misalignments of the limbs to be corrected during growth.

Growth-associated ailments are characterized by enlarged growth centres, failure of bone formation from cartilage, occasional cartilage necrosis and cyst formation. The solution would seem to lie not only in the control of dietary energy and protein and the correction of errors in mineral and trace-element nutrition, but also in raising the number of daily feeds and decreasing their individual size. The logical extension of this is to change to a system of earlier weaning and feeding foals *ad libitum* a complete mix, as described in Chapter 8. This will encourage nibbling and avoid large postprandial surges in blood glucose and amino acids that stimulate insulin secretion.

Worming of the foal should not coincide with weaning, but the first dose of anthelmintic can be given at two to three months or, ideally, at four months of age and thereafter at six-weekly intervals (see Chapter 11).

WEANING PROCEDURE

Restricted feeding of the mare limits milk secretion, but after weaning the udder should not be milked out. Some breeders rub camphorated oil into the udder.

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(a)



Plate 7.2 Epiphysitis in the lower (distal) end of the metacarpus and the upper (proximal) end of the proximal phalanx (fore fetlock) (a) and (b); and spavin of the hock (c) in foals. (Photograph 7.2 (c) courtesy of Dr Peter Rossdale, FRCVS.)

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(b)



Plate 7.2 *Continued*

The psychological attachment of the foal to its dam is greatest between the second and 12th weeks of lactation with a peak around the third week, at which time separation leads to the greatest agitation of both. Risk of injury to the foal at weaning as a result of excitement induced by separation is a major factor to be contained. On large stud farms, three alternative procedures are practised:

(1) All the mares are removed from a year's crop of foals at the same time.
(2) One or two mares are removed at a time, starting with the first foaled, or most dominant mare, and allowing a few days to elapse before the next is removed.
(3) The foals are separated for increasing time periods, such that the foals are allowed to nurse three times per

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(c) 

Plate 7.2 *Continued*

day, twice and then only once on successive days, keeping mares and foals in sight of each other.

Methods (1) and (2) could require access to another farm to ensure that weaned stock are out of sight, sound and smell of their mothers. Method (2) can lead to some foals being kicked when searching for milk by more aggressive mares. The last method protracts the drying-up procedure, is more laborious, and not without risk.

It is, however, recommended that, unless there are other mitigating circumstances, mares should be abruptly removed from foals, starting with the mare having the largest and most independent foal, or with the most dominant mare, which is likely to cause problems to other foals. Several days should elapse before the next most dominant mare is removed, leaving the foals in familiar surroundings out of sight, sound and smell of their mothers. It is helpful to leave a gentle dry mare with the foals and any foal having a cold or other debilitating condition should not be weaned until it has regained health.

Foals at first become frantic and it is important to ensure that all have company, that there is ample space for play, that the pasture has a clean water source and a shelter, and that the shelter and fences are free from protruding nails, splinters and loose wire. The pasture should also be of good quality, without a worm burden, and free from flints and rabbit, or other, holes that might cause leg injury.

Early weaning

The procedures outlined for both liquid and dry feeding of orphaned foals can be followed for early weaning of foals, but the procedure is both labour intensive and an interruption to normal activities on the stud farm. From both a practical and an economic point of view therefore a general recommendation for early weaning is not given. Nevertheless, there will be circumstances where it has a practical value.

All early-weaned foals must receive colostrum during the first day of life, before they receive other feed. Weaning at three to five days of age, when the mare is not imprinted

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Table 7.9 Performance of TB foals weaned at 5 days of age (Pagan *et al.* 1993a).

Age (days)	Average BW (kg)	Milk DM intake (kg/day)	Pellet intake (kg/day)	Coarse feed intake (kg/day)	Total DM intake (kg/day)
5–14	72	1.28	0.26	0	1.54
15–28	85	1.34	0.44	0	1.78
29–42	100	1.29	0.63	0	1.92
43–56	115	1.32	0.89	0.10	2.31
57–70	130	1.21	1.00	0.16	2.37
71–84	144	1.25	1.40	0.19	2.84
85–98	158	0.70	1.87	0.51	3.08
99–112	171	0	2.09	0.64	2.73
113–126	182	0	2.03	0.46	2.49
127–140	194	0	2.15	0.47	2.62
141–154	206	0	2.21	0.74	2.95
155–168	220	0	2.27	0.83	3.10

on the foal, and under hygienic conditions, is safe. A separation of six hours without feed normally eliminates fretting, but initially leads to a lower rate of growth than occurs in foals suckling their dam. The reasons for this are the shock of weaning and a lower milk DM intake. In traditional systems, this lower intake is caused by a lower meal frequency than occurs with suckled foals. Compensation by larger meals precipitates diarrhoea. Early-weaned foals should receive a minimum of six meals of milk/day. The milk should contain about 120–130 g DM/L. Eight to ten meals/day would increase the early growth rate without harm; however, retardation of early growth is subject to partial compensation by six months of age. Dry pellets, containing dried skimmed milk and other high-quality protein sources are offered *ad libitum* during this time. The milk replacer should contain about 25% protein and 16% fat in the DM. Some evidence suggests that medium-chain triacylglycerols are a useful source of fat for foals with a compromised digestive function. Neutral fats, such as coconut oil, that contain medium chain fatty acids, are hydrolyzed more rapidly by pancreatic lipase. Any unhydrolyzed medium chain fat is absorbed directly across the small intestinal mucosa. The total daily feed should ideally average 20–25% protein and 12–15% fat. Pagan *et al.* (1993a) reported on the performance of TB foals weaned at five days of age (Table 7.9).

FEEDING THE ORPHAN FOAL

The artificial rearing of relatively small foals should not be attempted lightly. TBs of less than 40 kg are normally destroyed. Orphan foals are deprived of the warmth of the dam and in cold weather should be covered with lightweight quilted material. Normally, a nurse mare is desirable, but in the meantime artificial feeding is necessary. The initial concentration of milk provided should contain 22% DM for the first 1–2 days, dropping by 1% daily until a normal concentration of 14–15% DM is reached and maintained until weaning. If there is diarrhoea, the milk can be diluted, or preferably replaced, for a short period with a glucose–electrolyte solution, which provides sodium, potassium, chloride, organic base and glucose in particular (see Table 9.3). However, all neonatal foals must receive an adequate source of immunoglobulins following birth before they receive *any other* organic food.

Colostrum

The provision of the newborn with colostral immunoglobulins is crucial to its survival in normal environments. If the mare is lost after the first day, the prospects of foal survival are greatly enhanced as sufficient colostrum should have been sucked by then. Where this is not the case, the maintenance of a bank of frozen colostrum is an asset and its value is enhanced where it has been derived from mares in a similar microbiological environment to that experienced by the foal.

Hygiene in the collection of colostrum is vital, and the preservation process should be carried out by experienced persons to ensure that no bacterial contamination has occurred, otherwise organisms will proliferate when the frozen colostrum is thawed. Thus, minimum quantities, sufficient for each feed, should be stored in each container and then thawed individually. The colostrum should be consumed immediately on warming to preclude undesir-

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able bacterial growth. The foal should receive about 500 mL colostrum by nipple or stomach tube every hour for three or four feeds before 12 h of age. If plasma immunoglobulin concentration can be measured, and is found to be less than 4 g/L, and the foal is less than 12–15 h old, then 2 L colostrum, or an amount to raise plasma levels to 8 g/L, should be administered by stomach tube in stages over several hours. Colostrum deprivation in a normal environment inevitably leads to very low serum IgG concentrations and septicaemia with a variety of bacterial species (Robinson *et al.* 1993).

Equine colostrum is the ideal, but bovine colostrum is also of value. Chong *et al.* (1991) raised 21 of 22 full-term Welsh Mountain pony foals in isolation, under conditions free from equine herpesvirus (EVH-1/4), but without mare's colostrum. The foals were given antibiotic prophylaxis and bovine colostrum during the first day. This was followed by mare's milk replacer until weaning. The foals remained free from EHV-1/4 infection.

Evaluation of colostrum

The efficacy of colostrum depends on its IgG content. In the stud, the IgG content can be approximated using a hydrometer, designed for the purpose, that relates the specific gravity (SG) of the colostrum to its IgG content. Evidence indicates that the estimate is influenced by the fluid temperature, which must therefore be controlled to obtain reliable data. Colostrum with an SG of ≥1.085 (equivalent to >7000 mg/dL IgG) at 25°C is suitable for freezing as a source for foals deprived of adequate colostrum from their dams. Colostrum with an IgG concentration of SG >1.03 (>3000 mg/dL) at 25°C is considered to be adequate for direct ingestion from the mare's udder within the first 12 hours from birth, so long as no milk has been ingested previously. The IgG level of foal serum should exceed 800 mg/dL between 18 h and 24 h post-foaling.

Bovine colostrum

Sachets of certified bovine colostrum powder, rich in IgA, IgG and IgM are available commercially and they store well. This powder can be added, preferably to water or the very first feeds of glucose and water given to newborn foals. These other feeds should *not* be given *before* the colostrum. A reserve supply of this product is a good insurance policy for most studs.

Giving blood plasma by parenteral or oral dosing

Where the concentration of immunoglobulins in the foal's plasma is less than 400 mg/dL and where colostrum is unavailable, one can use blood plasma, preferably from a donor gelding horse or unrelated mare which has never received a blood transfusion and which has been on the farm for some time. The dose is about 20 mL/kg BW, given i.v. over a period of 1–2 h, to a total of approximately 1 L per foal. If the foal trembles, the rate of dosing should be reduced and rapid recovery will soon follow. It should raise the antibody titre of its blood to about 30% of the donor level. Oral dosing with plasma helps in cases of enteritis, but it should normally be given aseptically i.v.

Plasma therapy, given i.v., is sometimes indicated for horses of all ages, but it is necessary in foals exceeding 12–24 h of age which have received insufficient colostrum and immunoglobulins. It is also frequently indicated for septic foals, or for horses suffering considerable blood loss, where the blood pressure will be low. Therefore a supply of donor plasma held at −20°C is a very useful. Frozen plasma is normally thawed in a water bath at 37°C. This is a lengthy process and evidence indicates that careful thawing at a defrost setting in a microwave oven, alternating with short periods of agitation, until no ice particles remain, is a satisfactory and quick method. The process, carefully handled, causes no apparent damage to plasma proteins.

Taurine

Colostrum contains a high concentration of the non-protein amino acid, taurine. The blood plasma of sick neonatal foals contains low concentrations of this amino acid, presumably because the undeveloped metabolic system is unable to synthesize taurine from cysteine and cystine, as occurs in the adult. There is a case for including taurine in foal milk replacer diets, and certainly it is essential to include taurine in colostrum replacers for foals that do not receive colostrum naturally. A dietary concentration of 300 mg/kg dietary DM is appropriate.

Enteral and parenteral feeding

The requirement for frequent small feeds has been satisfactorily met by giving low-residue solutions, containing sodium and calcium caseinate, glucose and fat, providing 4.2 kJ/mL through indwelling 12 French enteral feeding tubes. This fluid is given every four hours by gravity flow at the rate of 0.35 mL/min per kg, building up to the total energy needs over four days. However, if gas colic occurs, then an increase in the interval between feeds should help, and water should be offered *ad libitum*. Ousey (1998) stated that foals suffering neonatal maladjustment syndrome achieved, per kg BW, a daily enteral intake of mare's milk, or milk replacer, only 25% of that achieved

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by healthy foals of the same age. The unhealthy ones were in negative energy balance, despite their lower activity. Their ME/GE intake ratio was lower than for healthy foals, indicating impaired digestion. Ousey concluded that sick foals should have a minimum GE intake of 260–290 kJ/day per kg BW and that minimal enteral feeding should be combined with parenteral feeding, as deprivation of enteral feeding impairs GI function.

The enteral feeding tubes are placed with the tip in the distal oesophagus and the exterior portion secured by stented suture to the nostril (a stent is a mould for holding a graft in place; in this case, it is a device for securing a tubular structure). An extension tube is placed in the free end and secured with a plastic guard to the halter. The tube is flushed with water and capped when not in use. This procedure avoids the risk of trauma to the pharynx caused by frequent intubation. Mineral oil is sometimes added to each feeding, at the rate of 1–4 mL, if constipation occurs, although its use should be limited as it will interrupt the absorption of fat-soluble nutrients. When enteral feeding is contraindicated total parenteral nutrition (PN) provides a source of energy and protein. Solutions of 20% lipid-containing PN facilitate the delivery of energy to the critically ill foal. Krause & McKenzie (2007) observed that more foals receiving an emulsified lipid PN developed hyperlipidaemia, found by Myers *et al.* (2009) to be associated with higher mortality during the acute phase of neonatal illness. Nevertheless the advantage of isotonicity and of higher energy content cf. dextrose is considerable. High dextrose solutions were associated with glucose intolerance.

Feeding frequency and method

Bucket feeding of milk is the traditional method of feeding, although with compromised foals other approaches are required. The dictum of a little and often applies forcefully to orphan foals. Frequent small amounts reduce the risk of digestive upset and of hypoglycaemia. Where the foal is to be trained to a bucket, the head can be drawn into it with a finger in the mouth – initially this might require the assistance of another person, but soon the foal will adapt to the procedure. All feeding utensils should be clean before each feed. The intake of liquid milk replacer, or a 50:50 mixture of skimmed milk and whole cow's milk, should be at the rate of 280 mL every 1.5 h so that the daily energy intake amounts to 9–10 MJ DE. As a mare's milk replacer, Ousey (1998) considered that neonatal foals tolerated goat's milk better than cow's milk , with fewer digestive disturbances. The GE requirement of healthy neonatal foals was estimated to be 210 kJ/day per kg BW, an estimate consistent with that given above. The initial feeding can take place to advantage close by a horse acting as a decoy, but never at the stable door, to avoid the association of it with feed. In order to minimize affinity with man, orphan foals should not be fondled.

Liquid milks are normally given at body temperature, but can equally be given cold. Within a few days the daily intake will attain 9–18 L and if the foal is permitted to drink freely it can reach 36 L. However, intake should be restricted to a maximum of 18 L in a large foal and with any evidence of diarrhoea the quantity should be reduced until the problem subsides. Once the first few days are over, the liquid can be provided in four then three feeds per day and any excess discarded.

Automatic liquid-milk feeders of French design have proved very successful on large studs for groups of foals, and they avoid the problem of humanizing. They are electrically operated; the water is warmed and mixed with milk replacer powder at an adjustable rate (Table 7.1). Fresh liquid is prepared to replace that used up as the foals drink. The appropriate concentrations of DM suggested above in 'Feeding the orphan foal' should be adhered to, as solutions that are either too weak or too concentrated can precipitate looseness or constipation. New foals are rapidly trained by experienced foals in the same yard.

Creep pellets in the form of stud nuts or concentrate mix, together with milk pellets and a little best-quality leafy hay from 7 days of age will encourage dry feeding. Access to *fresh* faeces from a healthy adult horse that has been wormed regularly will provide bacteria of the appropriate kind for seeding the intestinal tract. Any strongyle or ascarid eggs in the faeces should be immature and therefore of low infectivity, and so will passively traverse the Gl tract of the foal, but the foal's faeces should be removed regularly. If progress is normal, liquid milk can be discontinued from 30 days of age and intake of dry feed will rise rapidly. At this time the foal could be consuming as much as 2–3 kg dry feed/day, although the consumption of hay will still be rather slight.

Fostering

The least troublesome nurse is frequently an old cold-blooded mare, especially of piebald breeding, or even a nanny goat. The worst type is a young flighty TB. Prospective individuals should be checked for disease, and their milk should be examined. The udder and the tail should be thoroughly washed and disinfected. The mare can be brought to the stable hooded and disorientated by walking around the area. A strong smelling substance such as camphorated oil can be placed on the muzzle and the

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same substance smeared on both the mare's own foal and the orphan. When these two are held together for a while the mare confuses their sounds.

A fostering crate is a boon for nurse mares, enabling foals to be suckled without being kicked. It will have facilities for feeding and watering, but the mare should be allowed out for exercise at regular intervals. The crate should have a gate at each end. The critical dimensions are a length of about 250 cm, a width of 65 cm and a gap at both sides of one end 90 × 40 cm, the lower edge of which is 70 cm from the floor for access by the foals to the udder.

The sick neonatal foal

In addition to the tenets of good husbandry there are several important issues upon which action should be taken to further the prospects of the unhealthy neonatal foal.

Immunoglobulin status should be measured. Plasma concentration of IgG should be raised to a minimum of 8 g/L, by administration of approximately 2 L colostrum, derived preferably from the dam. This should occur before 12 h of age. For foals approaching 24 h of age, or for those that are septicaemic, or hypothermic, 2–4 L of equine plasma, containing at least 1.6 g globulins/L, from a suitable donor, should be administered aseptically and i.v.

Energy reserves are likely to be even lower than those of healthy foals. The risks of hypoglycaemia and hypothermia are considerable. The foal should be held in a warm draught-free box, but with clean air and near its dam.

Following administration of the colostrum (the injection of serum takes place subsequent to a meal of milk/dextrose etc.), milk, preferably derived from the dam, should be given orally (bucket or bottle), or by indwelling nasogastric tube, every 1 to 2 h, so that the 24 h intake is approximately 20% of the foal's body weight. By remaining within smelling distance of the dam the foal is more likely to be accepted later on, and by milking the dam regularly her supply should not dry up. She is then more likely to suckle her foal at a future date. Fresh drinking water should be available. If the dam's milk is unavailable then a low fat (2%) cow's milk can be used, fortified with 20 g dextrose/L.

The foal's urine should be monitored for the presence of glucose and the blood should be sampled before several meals to ascertain the status of glucose, TAG, ammonia, urea, potassium and haematocrit. Elevation of TAG levals may indicate hepatic malfunction and fat intake should remain low (but present), as long as there is no hyperglycaemia and urinary glucose. With hyperglycaemia the dextrose intake per meal should be reduced to lower urinary glucose level to under 1%, reducing the risk of renal damage. With elevation in both plasma TAG and glucose a mixture of dextrose and fructose might replace a pure dextrose solution and the frequency of feeding should be increased, giving smaller amounts per meal. Elevated plasma ammonia should be countered with a reduction in milk protein intake. If milk replacer is used, the milk must have been spray-dried and not roller-dried. The detection of an elevated haematocrit and/or plasma albumin concentration may indicate dehydration, compensation for which should be made with Ringer's solution.

If there is evidence of obstruction of the intestines and lack of bowel motility (ileus), impactions or malabsorption, then total parenteral nutrition (TPN) will be necessary. PN may be used as an adjunct to oral feeding, where digestive ability is poor. For a full account of the problems associated with PN and jugular catheterization the reader is referred to *The Equine Manual*, 2nd Edn (Higgins & Snyder 2006). The composition of an i.v. solution for TPN, to be given over four days, is presented in Table 7.10. Alternatively the vitamin supply can be given by sterile intraperitoneal or subcutaneous injection. Monitoring should be as indicated above, including that of electrolyte balance.

Thrombophlebitis is not infrequent and has several causes. The risk of thrombophlebitis is decreased by complete sterility and absence of septicaemia, the use of the most suitable catheters, the inclusion of heparin in the solution and by replacing approximately half the energy requirement with lipid emulsion such as homogenized soya oil (e.g. Intralipid™, Kabi Pharmacia Ltd, Milton Keynes, UK). Lipid clearance should be monitored and fat supplementation avoided in cases of liver failure. Lipid use can reduce the risk of hyperglycaemia and will reduce the total osmolality of the solution. If severe proteinuria is presented, the amino acid (especially glycine) content of the PN solution should be reduced until the renal problem is resolved.

Table 7.10 Hypertonic i.v. solution for TPN of foals (2.7–3 L/day for a 45 kg foal).

5% amino acid solution	1000 mL
50% dextrose solution	500 mL
KCl	30 mEq/L
$NaHCO_3$	30 mEq/L
Injectable vitamin preparation	+*

*Commercial preparation of fat- and water-soluble vitamins.

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Sugar tolerance

Glucose can be utilized by foals of all ages. Neonatal foals are intolerant of the disaccharides maltose and sucrose, which should therefore be excluded from milk replacers intended for foals less than 5–7 days old. Maltose is successfully digested and absorbed by foals over this age. Intestinal intolerance to specific foods occurs in some foals and older horses. For example, the failure of some neonatal foals to secrete lactase results in failure to digest lactose in the mother's milk. This causes osmotic diarrhoea, weight loss, lethargy, colic and abdominal distension. Lactose intolerance may also occur secondary to intestinal bacterial infection; but deficient foals will respond to oral administration of exogenous lactase (Roberts *et al.* 2008). Normal neonatal foals are tolerant of both lactose and glucose, as determined by a rise in blood glucose following oral administration of these sugars (N.B. galactose, a component of lactose, is an epimer of glucose, rapidly converted to glucose by hepatic UDP (uridine diphosphate)-galactose-4-epimerase).

Infections and hygiene

Both respiratory and enteric pathogens are major causes of morbidity in young foals. Browning *et al.* (1991) surveyed 326 diarrhoeic foals in the UK and southern Ireland from 1987 to 1989. They found that Group A rotaviruses were major pathogens in all age groups. Other pathogens included *Aeromonas hydrophilia*, whereas coronavirus, parvovirus, *Campylobacter* spp., *Salmonella* spp. and *Bacteroides fragilis* were likely to be minor putative pathogens in Western countries. Rotavirus is a predominant cause of enteritis among foals in the US. Whether vaccination is an effective defence for this virus is unclear, but disinfection, hygiene and sound management practice continue to be the primary control measures against the ravages of foal diarrhoea.

Gastric lesions in foals

Among foals under 10 days old showing no signs of gastric disease, gastric lesions are of frequent occurrence, but rare over in those 70 days old (Murray *et al.* 1990). In common with horses in training, the lesions are situated predominately in the squamous mucosa immediately adjacent to the margo plicatus along the greater curvature. These squamous cells are less extensively protected by mucus than is the glandular mucosa. Lesions were found by Murray *et al.* to occur with lower frequency in the squamous fundus, the glandular fundus and in the lesser curvature. Lesions were more prevalent where there had been diarrhoea, and a role for GI pathogens, such as rotavirus, may be implicated. However, environmental stress with adrenal medullary hormone secretion, causing mucosal ischaemia and gastric fermentation in unhealthy foals, may also be involved (see Chapter 11).

THE STALLION

The stallion is subject to the same seasonal influences that affect the breeding cycles of the mare: his fertility is greatest in the summer and least in the winter. A large number of studies have been undertaken at Colorado State University to increase our understanding of the mechanisms that control the breeding behaviour of mares and stallions, publications on which provide detailed information.

Seasonal changes in the blood concentrations of LH, FSH and testosterone, and consequential changes in testicular size, sperm production and libido, are functions of changes in photoperiod. Melatonin feed supplementation of stallions in July to September in Louisiana caused a reduction in plasma concentrations of the pituitary hormones prolactin and FSH without influencing testosterone, indicating that melatonin plays an inhibitory role in hypothalamic regulation of the pituitary (Storer *et al.* 2003).

Recrudescence in reproductive activity, as in the mare, is a response to increasing daylight length. Thus, evidence indicates that improved fertility in the early months of the year could be obtained by following a regime in which artificial light and richer feed are provided in late December and January. At no time should stallions be allowed to get too fat, and higher fibre, but balanced, feeds are quite satisfactory out of the breeding season. Poorer quality hay supplemented with high-fibre horse and pony nuts should then be satisfactory, and will facilitate the imposition of a rising plane of nutrition as the breeding season approaches, when stud nuts, or an equivalent concentrate mixture with good-quality hay, should be introduced (Table 7.1). The energy requirements of the stallion rise during the breeding season as a consequence of increased physical activity, in particular that of pacing his run or stall, but space for physical exercise is important to the stallion in all seasons.

There is little evidence to support the use of special supplements to enhance the fertility of stallions, but a diet of 0.75–1.5 kg cereal-based concentrates plus hay per 100 kg BW/day and clean water (Table 7.5) should suffice.

Artificial insemination

This subject is outside the scope of this text, but some nutritional information is available. Bruemmer *et al.* (2002) concluded that if stallion spermatozoa are cooled

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and stored for longer than 24 h their fertilizing capacity declines. However, if 2 mM pyruvate and skim milk are added to cooled spermatozoa, oxidative stress is reduced and motility and fertility are maintained for 48 h.

STUDY QUESTIONS

(1) What issues should be considered in deciding the optimum weaning age for a stud and for an individual foal?
(2) On making arrangements and provisions for colostrum-deprived foals what are the important issues?
(3) What action would you take in the event of diarrhoea occurring:
    (a) in a suckled foal;
    (b) in a weaned foal; and
    (c) when there is an outbreak among several foals of similar age?

FURTHER READING

Allen, W.R., Wilsher, S., Turnbull, C., *et al.* (2002) Influence of maternal size on placental, fetal and postnatal growth in the horse. I. Development *in utero*. *Reproduction*, **123**, 445–53.

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8
Growth

For it is easy to demonstrate that a horse may irrecoverably suffer in his shape and outward beauty as well as in strength by being underfed while he is young.

W. Gibson 1726

Normal growth patterns, growth quality and conformation in the horse are to a considerable extent beyond the control of the stud. Nevertheless, breeding and diet have increasingly measurable effects; consequently, their contribution will undoubtedly accelerate. A key to obtaining a benefit from those roles is an agreement of objectives that can be clearly characterized and measured.

IDEAL CONFORMATION

Selection of breeding partners for shape and size has undoubtedly concerned breeders since domestic horse husbandry began. However, the shape of a horse that is required to maximize its performance in the type of activity for which it has been bred has received scant objective attention. Variability in shape within breeds is considerable. Mawdsley (1993) found a wide variation in linear traits within TBs, providing possible scope for progress by performance testing. (Variation in tissue type for work is discussed in Chapter 9.) In order that progress is achieved to improve the performance potential and working lifespan of foals it is necessary:

- for measurable objectives to be agreed within breeds at specified ages;
- for progeny performance testing to be conducted; and
- that note is taken of corrections to malformations by the farrier and others.

BIRTH WEIGHT AND EARLY GROWTH

Growth proceeds through a process of cell division and enlargement initiated by the fertilization of the egg. Cells differentiate, to form the various embryonic tissues. Soon after birth the number of cells in most tissues reaches a maximum and further growth is accomplished by hypertrophy (enlargement) of the individual cells; in other tissues however, for example, in bone, endochondral and periosteal growth continues to adulthood, while in epithelial tissue, cell replication continues throughout life to replace cells that are sloughed off, and in hepatic tissue, growth occurs to compensate for malfunction. Not all tissues, organs and structures increase in size at the same rate, so that during growth the shape of the animal changes.

The potential for a maximum rate of growth, measured in kg/day BW gain, persists until about nine months of age in the horse, at which time it gradually declines and ceases as the adult size and shape are attained. However, the overall rate of growth measured relative to existing weight, i.e. say at 50 days of age $(\text{kg/day gain})_{t50}/BW_{t50}$, is initially slow (Figure 8.1); it accelerates to a maximum before birth and then declines. By the seventh month of gestation, merely 17% of the birth weight and only 10–15% of the birth dry matter have been accumulated. Thus, the accretion of most of the energy and minerals present at birth occurs during the last months of gestation.

Partly because the mature number of cells in many tissues of the adult has been achieved by birth or shortly afterwards, the maximum adult weight of horses and ponies (as distinct from height) is, to a large extent, determined by birth weight. As a rough guide, birth weight constitutes 10% of the adult weight. Among TBs, individuals weighing less than about 35 kg at birth are unlikely to reach normal mature size. The proportion of foals with a birth weight of less than 40 kg that actually raced was found to be much smaller than the proportion of those weighing more than 40 kg (Platt 1978). Horses out of small mares by small stallions will be small as adults, but will

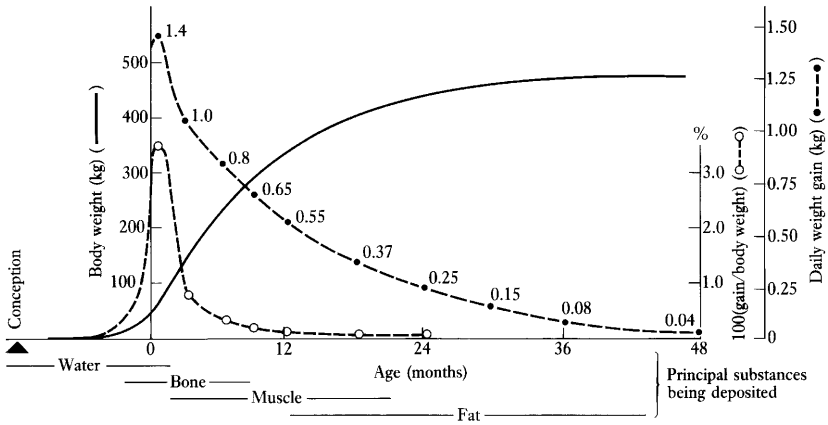


Figure 8.1 Normal growth curve for the horse (mature weight of example, 500 kg).

Table 8.1 Percentage of mature body weight and of withers height attained at different ages in various horse breeds (Hintz 1980a).

	6 months		12 months		18 months	
Age	Weight	Height	Weight	Height	Weight	Height
Shetland pony	52	86	73	94	83	97
Quarter Horse	44	84	66	91	80	95
Anglo-Arab	45	83	67	92	81	95
Arabian	46	84	66	91	80	95
TB	46	84	66	90	80	95
Percheron	40	79	59	89	74	92

achieve their mature size slightly sooner than the products of large parents. Differences between breeds in rate of attainment of mature weight are greater than the differences in rate of attainment of mature height.

Studies at the Thoroughbred Breeders' Association Equine Fertility Unit in Newmarket, at the Royal Stables in the United Arab Emirates (UAE) and at The Sheikh Mohammed Camel Reproduction Laboratory in Dubai, should throw more light on the effect of foetal size on adult size, extending the original research of John Hammond at Cambridge in the 1940s and 50s. The work in the UAE includes crosses in both directions between llamas and camels, embryo transfer and implantation in foster camels, and implantation of Arabian embryos in draught mares.

Table 8.1 gives data for several breeds of horses. As a general rule, foals attain 60% of their mature weight, 90% of their mature height and 95% of their eventual bone growth by 12 months of age. In the period from birth to maturity, the growth coefficients (the rate constant for the growth of a tissue, or structure, relative to that for the whole empty body) of the three major equine tissues in order of increasing magnitude are bone, muscle and fat, implying that bone is the earliest maturing tissue and fat the latest. This, and the earlier assertion that overall growth rate declines from birth (weight gain per unit empty live weight), signifies that ultimate height is determined in very early life, that early growth demands diets rich in bone-forming minerals, protein and vitamins, and that with increasing age an increasing proportion of dietary carbohydrate is required. The early rapid extension of the long bones of the legs, pronounced in tall breeds, render them subject to deformation through early malnutrition.

Staniar *et al*. (2004) fitted a sigmoid growth equation to the weight for age data of 2698 TB foals. A mature weight

of 542 ± 6.2 kg was reached at 7 years. Their equation can be used to identify significant deviations amongst growing horses:

$$W = A(1 + be(-kt))M$$

where W is BW at age t, A is the asymptotic value of W, b is a scaling parameter that defines the degree of maturity at $t = 0$, k is a rate constant, and M defines the point of inflection in the sigmoid curve in relation to age.

Valette *et al.* (2008) modelled the evolution of wither height from birth to adulthood in sport- and race-horses. Gompertz, logistic and cubic models correctly estimated height during the first 2 years. These gave a better fit than the linear, quadratic and cubic models. (Gompertz and logistic models are two sigmoidal functions that allow growth rate to vary with time, rather than assuming it remains constant. Gompertz models assume the relative growth rate declines at a constant rate. They are used widely, for example, to model population growth.) The logarithmic and power model was preferred during the period 2–6 years of age. Racehorses displayed an intense growth in their first months, whereas sport horses had more regular prolonged growth in their first year.

NRC (2007) calculated a weighted least squares estimate of parameters a and c in the following non-linear equation to derive Y, the per cent of mature body weight between 450–650 kg:

$$Y = 9.7 + (100 - 9.7)\left(1 - e^{(-0.0772x)}\right);\ R^2 = 0.99$$

where a = 9.7% of mature BW at birth; c = rate constant, 0.0772; e = 2.7183; and x = months of age.

It seems that this equation is derived from pooled within-breed data and not from observations on individual horses and so excludes the between-horse variance within a breed. Moreover, this is a smooth growth curve and, as is recognized, growth rate is somewhat retarded during the winter in northern latitudes, a condition that is followed by an accelerated rate in the spring on pasture (see effects of daylight length, Chapter 7). Ringler & Lawrence (2008) assessed the validity of the above NRC equation, which closely predicted BW of growing TB horses at between 6 and 9 months of age, but underestimated BW between 1 and 5 months of age (by as much as 10 kg), overestimated BW between 10 and 13 months of age (by as much as 9 kg) and underestimated BW by as much as 9 kg between 15 and 16 months of age. The explanation is that the foals were raised on pasture and increased in daily gain between 12 and 14 months of age.

Early nutrition and weaning age

The nutrition of the foal at birth is affected not only by the feeding of the mare but also by the physiological efficiency of the uterine environment. This could to some extent be affected by the age of the mare, as indicated by the data in Table 8.2. The BW, hypothalamo-pituitary-adrenal (HPA) activity and pancreatic β cell function of newborn foals are all affected by acute maternal nutritional deprivation in mid-gestation. Nutritional programming of the pancreatic β cells, but not the HPA axis, appears to depend on the level and nature of nutrition before and after a mare's weight loss period (Ousey *et al.* 2008). Feeding mares to obesity might, therefore, prevent adverse consequences of acute nutrient deprivation on the pancreatic β cells, without materially affecting birth weight or adrenocortical responses of the newborn foal. During weight loss the plasma insulin and glucose concentrations of pregnant mares are depressed. This is associated with their foals having higher insulin responses to glucose, cf. foals from mares given a near *ad libitum* regime throughout pregnancy. The exaggerated β-cell response after acute

Table 8.2 Effect of age of TB mare on body weight and height at withers of foals (Hintz 1980a; data based on 1992 foals).

Age of mare (years)	Age of foal			
	30 days		540 days	
	Foal weight (kg)	Height (cm)	Weight (kg)	Height (cm)
3–7	93.0	108.0	393.7	152.4
8–12	97.5	110.5	401.4	153.7
13–16	98.0	110.5	396.9	153.0
17–20	95.3	109.2	391.0	152.4

Table 8.3 Effect of sex on growth of TB foals (Hintz 1980a; data based on 1992 foals).

	Body weight (kg)		Withers height (cm)	
Age (days)	Colts	Fillies	Colts	Fillies
2	52.2	51.3	100.3	99.7
60	136.5	134.7	118.3	118.1
180	244.9	235.9	134.6	133.0
540	435.5	401.4	154.3	152.4

Table 8.4 Effect of month of birth on body weight and withers height of TBs (Hintz 1980a; data based on 1992 foals).

	Age			
	30 days		540 days	
Month of birth	Weight (kg)	Height (cm)	Weight (kg)	Height (cm)
February–March	95.3	109.2	396.9	153.7
April	97.5	110.5	402.8	153.7
May	100.7	111.1	403.7	153.7

maternal deprivation superimposed on a moderate nutritional plane has long-term sequelae, because high postprandial insulin levels have been linked to a range of debilitating diseases, including orthopaedic disorders, laminitis, and exertional rhabdomyolysis (Ousey *et al.* 2008). On the other hand, the effect on growth of small differences in birth weight brought about by nutritional deviations in the pregnant mare can be proportionately lessened by nutritional adjustments in early postnatal life. Although birth weight has a major impact on ultimate size, weaning age, under conditions of good management, has little influence. Foals weaned after receiving colostrum can achieve growth rates equal to that of those weaned at 2 to 4 months of age. These in turn can grow faster than foals weaned at 6 months of age. The appropriate weaning age, therefore, for any particular stud farm, is determined by the most convenient and reliable management practice.

LATER GROWTH AND CONFORMATIONAL CHANGES

Initial and ultimate weights and heights differ between colts and fillies (Table 8.3). The differences are small and Green (1969) failed to detect sex differences in linear measurements, or any between early- and late-born foals. Nonetheless, substantial evidence indicates that foals born late in the season are heavier and taller than early-born foals (Table 8.4) despite a somewhat shorter period of gestation. Gestation length seems to be a function of daylight length in late gestation (see Chapter 7). Birth weight and subsequent rates of growth also depend on the mature size of the breed. Micol & Martin-Rosset (1995) recorded that the rate of weight gain from birth to weaning on pasture of the heavy French breeds – Breton, Comtois and Ardennais (700–800 kg mature BW) is 1.3–1.7 kg/day. Even under harsh upland conditions the foals gain at the rate of 1.3–1.5 kg/day.

Differences in growth rate after the neonatal and post-weaning periods, have little influence on subsequent growth or skeletal measurements in TB and Quarter Horse foals (Peterson *et al.* 2003), or on mature size. Lawrence *et al.* (2003a) estimated that the variation in growth rate within the Morgan horse population had little impact on their ultimate height. Although maximum height is approached soon after 12 months of age, this can be delayed without a reduction in the ultimate measurement by reducing the rate of feeding slightly. Similarly, 90% of mature weight is achieved at 18 months, but delayed until 24 months by the same restriction. In Figure 8.2 the changes in height at the withers over the first 12 months

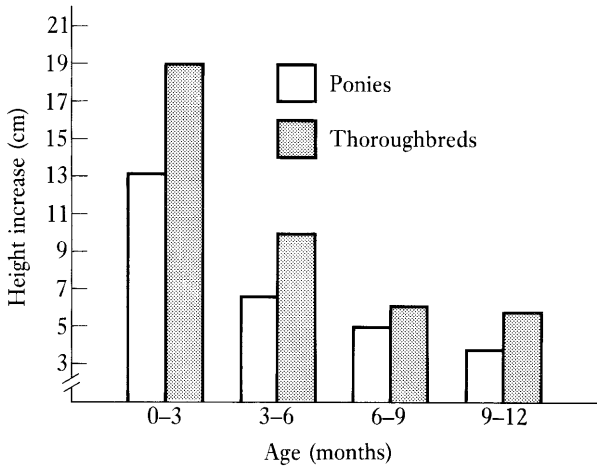


Figure 8.2 Height increase (cm) at withers of pony and TB foals (Campbell & Lee 1981).

of pony and TB foals are compared. Although the foals achieve very different heights, the pattern of growth is similar.

Height at the withers largely reflects linear growth of the long bones in the front legs. Long bones increase in diameter or thickness throughout their length, but no increase in length occurs within the shank, or diaphysis, after birth (Figures 8.3 and 8.4). They increase in length by growth in the metaphyseal plate at both the near and distant ends (proximal and distal) from the body. The rate of growth at each end is different (Table 8.5). Correction of distortions in bone growth, owing to bad feeding practices and other causes, is possible during the phase of rapid growth of the end in question. For the distal radius or tibia, such correction could be imposed up to 60 weeks of age, whereas fetlock distortion, a fairly common condition, requires treatment by three months. In either case, temporarily restricted growth will either not, or only slightly,

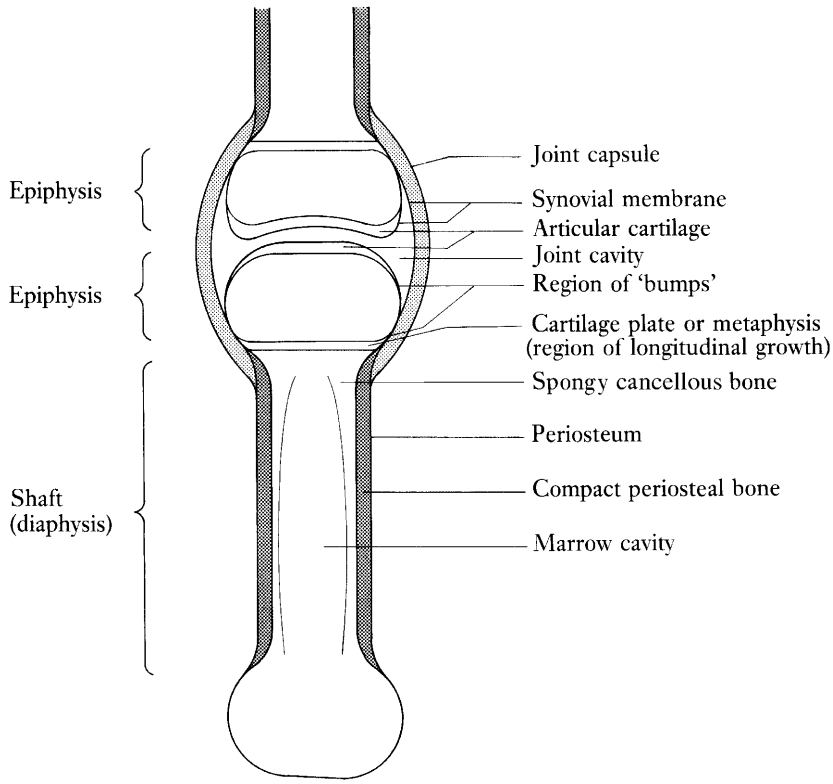


Figure 8.3 Long bones and their articulation in late growth (joint cavity has been expanded for purposes of visualization). Note the regions of growth: cartilage at either end of diaphysis; cartilage of epiphysis; and periosteum of shank.

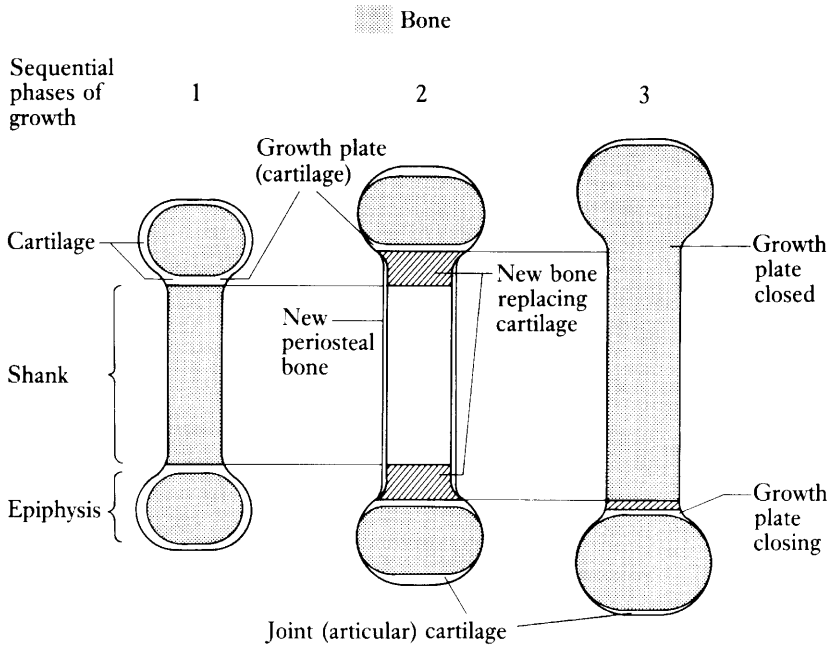


Figure 8.4 Typical growth of long bone, for example, radius or tibia (after Rossdale & Ricketts 1980).

Table 8.5 Growth of leg bones in three male and three female crossbred ponies (Campbell & Lee 1981).

	Length of bone (cm)		Increase at each end (%)		Age at closure of growth plates (weeks)	
Bone	0–7 days	2 years	Proximal	Distal	Proximal	Distal
Femur	21.6	30.9	24	20	55	55
Tibia	21.3	29.1	22	17	60	60
Radius	20.7	28.1	12	24	54	69
Humerus	16.0	22.6	31	11	80	70
Metatarsal	22.7	23.7	5	5	40–44	40–44
Metacarpal	18.4	19.4	5	5	40–44	40–44
Phalanges 1	5.9	6.6				
Phalanges 2	2.3	2.5				
Phalanges 3	5.6	6.2				

influence ultimate size (see 'Developmental orthopaedic disease', this Chapter; Brauer *et al*. 1999).

The bone-specific protein, osteocalcin, is an indicator of osteoid production and its serum concentration is increased during both growth and bone matrix formation and with increased bone turnover (mobilization and formation). Other biochemical markers of bone formation include type I collagen carboxy-terminal propeptide, and the bone-specific isoenzyme of ALP. Cross-linked telopeptide of type I collagen is a marker of bone resorption and N-terminal propeptide of type III collagen is a marker of soft tissue turnover (Price *et al*. 2001). Although a low dietary cation–anion balance (DCAD) is expected to increase bone mobilization (resorption) and urinary loss of

Ca, an adverse effect on skeletal growth might not occur (see 'Minerals, trace elements and DOD' this Chapter; Cooper *et al*. 1999a).

The balanced adult horse of good conformation has a height at the withers that equals its length from the tip of the shoulder to the point of the buttocks (see Figure 6.1). By contrast, a foal is taller than it is long (Plate 8.1) so that it inevitably trots wide behind. Fillies at birth tend to be fairly level across the top, but are as much as 5 cm higher over the croup than over the withers at one year and then balanced again by 5 years. The length of their bodies tends to be greater than their height at the withers. Colts are generally higher over the hip at birth but level by 3 years. Some horses that have grown poorly in the front legs are lower over the withers than the croup at maturity and, as nearly 60% of the body weight is normally carried by the front legs, this unusual conformation can force additional weight and stress on the forequarter, increasing the risk of damage.

Similar height increases at the withers and at the hip are found in TB foals between 14 and 588 days of age when given feed at recommended rates. The hip height is about 1 cm greater than the withers height throughout this period (Thompson 1995). Cymbaluk and colleagues (1990) found that Quarter Horses fed *ad libitum* cf. limit-fed at 6–24 months, have a slight tendency to gain relatively more in hind-body mass than in fore-body mass, but to gain slightly less in croup height than in withers height. Thus, adult conformation could be influenced by rate of feeding during growth, and very restricted feeding restricts development of the hindquarter powerhouse. The extent to which compensatory growth occurs after 24 months is unclear.

When fed liberally, the growth of ponies up to 9 months of age might increase to nearly 1.5 kg/day, although by 12 months the rate will have fallen to half this amount. Under hill conditions, where young stock can receive little in the way of concentrates, growth can be strikingly depressed. Morgan horses on range were shown to be between 23 and 46 kg lighter and 2.5 cm shorter at 5 years of age than their contemporaries in the same environment receiving supplementary cereal-based concentrates (Dawson *et al.* 1945). With only moderate restrictions, such horses attain normal mature height at 5 years, whereas with more liberal feeding they can reach it by 3 years (Kownacki 1983). New Forest filly ponies, fed from 6 months of age throughout the winter at a rate that only maintained body weight, grew faster during the following summer on rough pasture than their counterparts permitted to gain 0.4 kg/day in the winter (Ellis & Lawrence 1978a,b, 1979, 1980). Although body weight remains constant in such deprived circumstances, parts of the skeleton grow differentially so that the restricted fillies in this example were tall, thin and shallow-bodied. With a delay in the closure of growth plates these differences are partly corrected during later growth. Catch-up growth of this kind is not without risk among potentially tall breeds, and although the evidence against interrupted growth curves is slight, a smooth growth curve should be sought for TBs, Saddlebreds and the like (Figure 8.1).

It is concluded that compensatory growth occurs in horses with some small influence on ultimate conformation, but the influence of this on the extent of active working life is yet far from clear. Growth abnormalities are considered in 'Developmental orthopaedic disease', this chapter.

Supplementary feeding in the first winter

With full access to good summer pastures, yearlings require no supplementary feed (that is, assuming the land has no specific trace-element deficiencies). The amounts of supplementary feed necessary during the previous winter will depend, first, on the quality and, second, on the amount of grazing available. Winter pastures have little value as a stimulus to growth in young stock, but will provide reasonable roughage to complement cereal-based concentrates. Until the spring flush is available, stock at this age should receive from 1.25 to 1.5 kg concentrates containing 16% protein and appropriate vitamins and minerals per 100 kg BW/day. A high-fat, high-fibre supplement at weaning appears to relax foals cf. those given a high-sugar, high-starch supplement (Redgate *et al.* 2004).

When exercise is limited by in-wintering, nuts or other concentrate feeds should be given to young stock in two meals daily to avoid intestinal upsets that might cause oedema in the legs. Stabled, weaned horses are given free access to good-quality hay, and when they are receiving concentrates at the rate of 1.25–1.5% of BW, they will consume hay or other forage DM at a rate of about 0.5–1.5% of BW. Using good-quality hay, Ott & Kivipelto (2003) found similar growth rates (0.75 kg/day) and bone formation amongst TB and Quarter Horse weanlings when concentrate : hay ratios were 1 : 1 or 1.7 : 1.0 in the period from weaning at 112 to 224 days of age. New Forest and Welsh pony filly foals required for maintenance 14–14.6 MJ DE/100 kg BW, equivalent to 1.75 kg average hay or 1.1–1.2 kg average nuts per 100 kg BW/day (Ellis & Lawrence 1980). At the end of the first winter, when yearlings are 11 or 12 months old, colts and fillies should be separated. The first oestrus should occur before May, unless winter conditions and feed have been poor.



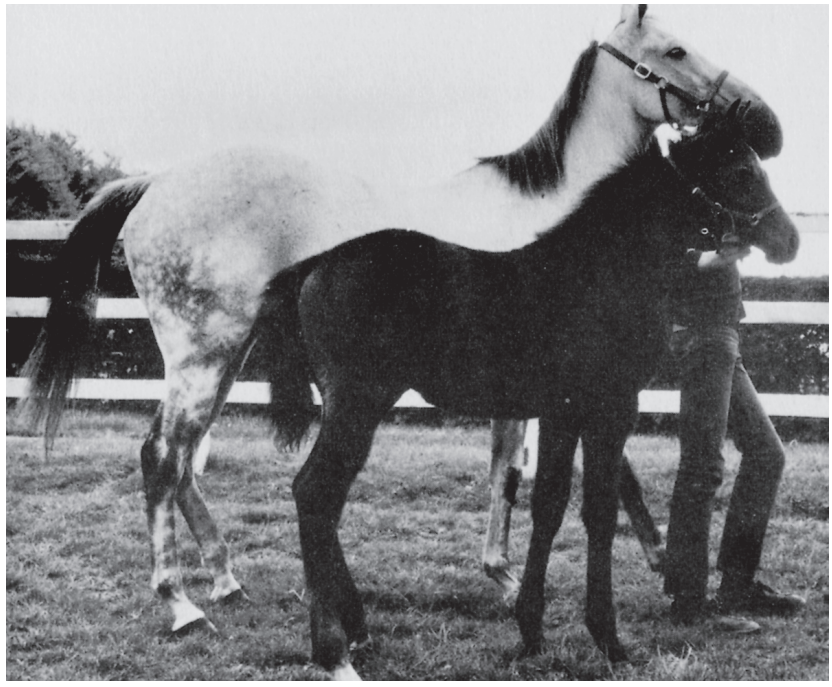


Plate 8.1 Grey colt (51 kg at birth) by Vitiges out of Castle Moon at Derisley Wood Stud, Newmarket: 5 days old (top) and 82 days old (below). Note that the height of the young foal exceeds its length, a characteristic that changes as adult proportions are attained. Over a period of as little as 77 days the length of the foal has increased considerably so the body proportions are already more like those of an adult. The height of the croup tends to increase faster than that of the withers during the first few months, but by three years of age in the TB the withers height has caught up, through differential growth of the leg bones.

Long yearlings

From 85–90% of mature weight, long yearlings of light breeds (during their second year of life) can be fed most economically on near maintenance rations as for mature horses. This should include good-quality hay and a concentrate offered at a rate of 0.75–1% of BW and providing cereals, a concentrate protein source, minerals and vitamins. However, when these more slowly growing stock are out on good-quality pasture continuously they should need no other source of feed, apart from a trace-element mixture should a specific deficiency exist. (See Chapter 11 for control of laminitis risks on spring pasture.)

Meat production

The heavy breeds (Breton, Comtois and Ardennais, 700–800 kg mature BW) are grown in France for meat in high forage systems. The systems include: (a) colts castrated at 18 months and surplus fillies slaughtered at weights exceeding 700 kg off grassland in mixed systems with cattle; (b) following grazing and finishing indoors at 10–15 months of age (450–500 kg BW) with continuous gains of 1.0–1.4 kg/day; (c) supplemented with cereals on grassland and slaughtered at the end of the grazing season at 18 months (550–580 kg BW); or (d) slaughtered at 22–24 months (620–670 kg BW) after finishing indoors during the second winter. The interesting observation that, as concentrate allowance to finishing horses is increased, the *ad libitum* intake of hay decreases by 1.26 kg DM for each 1.0 kg increase in concentrate, was made by Micol & Martin-Rosset (1995). Where maize silage is given (>30% DM), the silage DM intake decreases by 0.81 kg for each 1.0 kg increase in concentrate, so overall DM intake increases. The concentrate improves silage acceptability as the horses consume 24 g DM/kg BW of a 100% hay diet per day, compared with 16 g DM/kg BW of the 100% silage diet. The concentrate contained (g/kg): 900 ground barley and maize, 80 soya-bean meal and 20 minerals and vitamins. It was given in some studies 50 : 50 with maize silage or hay (Martin-Rosset & Dulphy 1987).

EFFECTS OF DIETARY COMPOSITION

Nutrient requirements are dealt with in Chapter 6. Certain aspects of those requirements that have engaged the interests of research workers are considered here, especially where they affect the quality of the skeleton and mobility of the horse.

Dietary protein

The NRC (2007) and others have published dietary CP requirements for growing horses (Tables 2.2, 6.17). Apparently digested CP is:

$$(\text{N intake} - \text{faecal N})/\text{N intake}$$

Three diets, meeting the recommendations of NRC (1978), except for protein, were compared in Arabian, TB and Standardbred foals from four months of age (Schryver *et al.* 1987a). The dietary CP and lysine concentrations were: 90, 140 and 200 g/kg diet and 2.8, 7 and 12.6 g/kg diet, respectively. The lowest protein intake depressed the rate of daily weight and height gain and gain in forecannon circumference, whereas there was no significant difference in the responses to the other two diets. At nine months of age, the foals that had received the 90 g protein/kg diet were changed to 200 g. After a further 140 days there was no significant difference in body weight, height or cannon circumference among the groups, indicating the capacity for compensatory growth.

Breeders have frequently limited the protein content of the weaned foal's diet to avoid the development of skeletal defects. There is little evidence that high intakes of protein are a cause of this frequent problem in several breeds (see 'Developmental orthopaedic disease', this chapter).

In the experiment of Schryver and colleagues, rate of feed intake, BW gain, height gain and the increase in forecannon bone girth were all greatest with the highest protein intake, although differing not significantly from the 140 g diet. Higher protein and lysine allowances increase cannon girth, whereas inadequate protein intake tends to produce more spindly bones that are only slightly shorter. Some evidence does indicate that bone density is slightly lower in foals that are allowed to grow at their maximum rate, and therefore bone strength for a given body size could be slightly compromised by very rapid growth rates.

Endogenous losses of N include faecal, urinary and cutaneous sources. NRC (2007) assumes a digestibility of 46% for CP and proposes an average protein maintenance requirement of 1.26 g/day per kg BW, with minimum and elevated extremes of 1.08 g CP and 1.44 g CP. For foals under 6 months of age the additional dietary requirement per kg BW gain is 512 g CP/day and after 6 months 844 g/day per kg gain, owing to a decreasing efficiency, E, of dietary CP use with increasing age (NRC 2007). NRC also assumes that the dietary lysine requirement is 4.3% of the CP requirement (Table 2.2, 6.17).

With growing horses of all ages it is important that the energy allowance is not excessive with respect to the protein allowance. Commencing at 120 days of age, poorer body condition has been observed in foals given 125% of the recommended NRC (1978) energy allowance but only 100% of the protein allowance, compared with foals given 100% or 125% of both NRC energy and protein allowances. Possibly as a result of this and other evidence the NRC (2007) recommendation for the protein allowance of yearlings is 20% greater than the 1978 recommendation, but similar to that of NRC (1989). The INRA (French) recommendation, given as MADC (see Chapter 6), is 66% of the NRC (1989) CP value at 6 months and only 54% at 12 months (Figure 6.11a). Very approximately the MADC value should be 70% of the CP value to be equivalent. The crucial factor is which proposal leads to a healthier ultimate development of the frame.

Grace *et al*. (1998a) observed that filly yearlings with an initial weight of 300 kg, gained at an average of 0.75 kg/day on a New Zealand pasture containing 201 g CP/kg DM. Their average DM intake was 6.5–7.5 kg/day. The pasture contained 11.4 MJ DE/kg DM, so the DE/day intake was 81 MJ DE, which was considered to be adequate. There was, indeed, abundant high-quality protein available in relation to digestible energy, yet amino acid digestibility was unknown.

In experiments by Thompson *et al.* (1988) energy, protein, Ca and P intakes, relative to NRC (1978) recommendations, were varied in the diet of growing foals. The results showed that inadequate dietary Ca caused lower increases in bone-mineral content and in long-bone length. Raised intakes of energy, or of protein, increased the rate of growth in length of several bones; but inadequate intakes of protein relative to that of energy compromised the development of cortical bone and led to smaller increases in withers height. Thus, fast rates of body-weight increase caused by excessive energy intakes introduce an increased risk of inadequate bone development when the balance of nutrients is incorrect, increasing the risk of conformational and musculoskeletal abnormalities (Thompson *et al.* 1988; Cymbaluk *et al.* 1990).

In the experiment of Schryver *et al.* (1987a) Ca metabolism was not adversely affected by high protein intakes, even though the 200 g CP diet contained nearly twice as much sulphur (S) as was contained in the 90 g CP diet. Other evidence indicates that a protein intake that is 130% of the NRC (1978) recommendation for foals causes an increase in the urinary excretion of Ca and P. This is primarily the result of the increased S-amino acid consumption causing a decrease in renal tubular filtrate pH and a consequential reduction in the renal tubular reabsorption of Ca and P. A low dietary cation–anion difference (DCAD), defined as mEq[(Na + K) − (Cl + S)]/kg DM, increases urinary Ca loss in exercising horses; but horses increase Ca absorption to compensate for increased urinary excretion of Ca (Cooper *et al.* 2000), and McKenzie *et al.* (2002) found that varying theDCAD did not alter daily balance of Ca and P. Whether this is true over a longer time period is unknown (see cation–anion balance, Chapters 9 and 11 and Appendix D). The difference between studies in the probable urinary Ca loss could be due to:

(1) differences between studies in the actual acid : base balance of the diets; and
(2) the age at measurement (There is a more rapid turnover of the amorphous calcium phosphate salts, found in greater abundance in the bones of younger animals, compared with the turnover of the more stable apatitic crystals that predominate in the bones of older animals.)

There is a potential risk to bone calcification, and dietary supplementation with sodium bicarbonate could be indicated with high protein diets. An optimum amino acid balance in the diet is of benefit, as excessive imbalanced protein may exacerbate urinary Ca loss.

Limiting amino acids

The first and second dietary limiting amino acids for the growth of foals are lysine and threonine, when they are given a diet of maize and oats, in equal amounts, plus soya, dried lucerne and coastal Bermuda grass hay (Graham *et al.* 1994). Staniar *et al*. (2001a) demonstrated that a supplement of lysine and threonine, included at rates of 6 g/kg and 4 g/kg, respectively, in a 9% protein concentrate and given to growing foals, surprisingly increased their weight gain on winter pasture, compared with the response to a concentrate containing 14% protein and 22% soyabean meal.

An excessive consumption of these amino acids does not materially compromise the tubular reabsorption of Ca. The lysine requirement, as a proportion of the diet, decreases rapidly with increasing age, but in the Finnhorse breed it exceeds 31 g/d up to ten months and it is important for normal blood haemoglobin and red blood cell formation that decreases during in-wintering (Saastamoinen & Koskinen 1993). An imbalance in dietary amino acids is likely to increase urinary losses of S, for a given rate of N retention, and therefore excessive poor-quality dietary protein compromises Ca balance.

Summer pasture generally contains more protein, and of a higher quality, than protein given to in-wintered horses. If inadequate dietary protein is given to foals during their first winter, Scandinavian evidence indicates that there is a fall in the concentrations of plasma albumin (Mäenpää *et al*. 1988a,b). Total plasma protein fell from 68 g/L in November to 56 g/L in April and reductions occurred in the plasma concentration of several of the free essential amino acids: isoleucine, leucine, lysine, phenylalanine, threonine and valine. Consistently under these conditions, decreases in plasma lysine, methionine and valine have been reported (Saastamoinen & Koskinen 1993). These decreases are associated with a marked interruption of the normal pattern of growth to 12 months of age. Manso Filho *et al.* (2009) measured the changes in the most abundant plasma and skeletal muscle free-amino acids between birth and 12 months to gauge their role. Other reports indicate that the effect of inadequate dietary protein on serum total protein has been slight, or absent, in adult ponies (Reitnour & Salsbury 1976) and among weanlings, when growth rate has been influenced (Godbee & Slade 1981).

Complete feed mixtures for growing horses

Complete mixtures of concentrates and chopped roughage are routinely given *ad libitum* to growing cattle, but to suggest that similar mixtures should be given to groups of growing TBs and horses of other breeds seems to be inappropriate. When a number of such stock are reared together through both the first and even the second winter months, there is much to recommend such a procedure. It does require sheltered *ad libitum* feed hoppers, as by ensuring that the feed is always present, risk of colic or of laminitis (founder) is low in foals. Complete feed mixtures should initially contain a cereal-based concentrate and good-quality chopped hay in a ratio of 2 : 1, gradually falling as the growth rate declines, to a ratio of 1 : 1. Stock will normally consume a total amount of dry feed daily equivalent to 3–3.5% of BW. The physical make-up of the mix and particle size should be so regulated that no segregation between the hay and the concentrate arises. Molasses is a good material to include in the mix as an aid in preventing segregation and some ingenuity is required to ensure that no bridging occurs in hoppers. The author's own evidence (Frape 1989) with such mixtures has been very encouraging, as assessed by conformation and control of DOD.

Preparation for sale

TB yearlings are normally prepared for the autumn sales by excluding them from pasture. A diet based on 1.5 kg good-quality stud nuts or other concentrate, plus 1–1.5 kg hay/100 kg BW is a typical practice. Traditional concentrate mixtures contain 75–90% of oats, plus bran, soya-bean meal, dried skimmed milk and sometimes a vitamin/mineral premix. There is no particular reason why these horses should not be provided with a complete mixture of concentrates and chopped hay as advocated for younger stock, but in a ratio of 1 : 1 concentrates to hay. Table 8.6 gives proposed feed mixtures for growing horses

Table 8.6 Feed mixture for growing horses for providing with chopped forage or grass hay or as a supplement to poor pasture.

	Concentrate mixtures (%)		
	Weaned foal	Yearling	Presale of long yearling
Oats	41.1	44.7	59.0
Wheat bran	15.0	15.0	10.0
High protein grass meal	15.0	15.0	10.0
Extracted soya-bean meal	18.0	15.0	10.0
Molasses	7.5	7.5	7.5
Feed-grade fat	1.0	1.0	2.0
Limestone	1.2	0.7	0.5
Dicalcium phosphate	0.5	0.5	0.4
Salt	0.5	0.5	0.5
Vitamins/trace elements*	0.2	0.1	0.1
Total	100.0	100.0	100.0

*See Table 7.1, p. 184.

Table 8.7 Daily feed allowances (kg) for growing horses (500 kg mature weight).

	Concentrates	Hay (clover/grass)
Foals (kg BW)		
100 (4–5 weeks)	0.5	—
130–180	2.2–3.2	1.3–1.9
180–230	2.9–3.9	1.8–2.3
230–270	3.6–4.8	2.2–2.8
270–320	4.0–4.6	2.7–3.2
Yearlings (kg BW)		
310–360	3.5–4.5	3.0–3.7
360–410	3.0–4.2	3.6–4.1
410–460	3.0–3.8	4.0–4.5

and Table 8.7 gives rates of feeding (on the basis of 90% DM).

DEVELOPMENTAL ORTHOPAEDIC DISEASE

Developmental orthopaedic disease (DOD) includes angular limb deformities, physitis, dyschondroplasia, subchondral bone cysts, osteochondrosis (OC), flexural limb deformities and cervical vertebral formation (wobbler syndrome). These conditions originate from abnormalities in endochondral ossification in one or both ends of long bones, the articular–epiphyseal cartilage complex, and the physis (growth plate) (Jeffcott 1991; McIlwraith 2004b).

The major disorders are summarized as:

- osteochondritis dissecans (OCD) occurs where there is inflammation and a separation of a piece of articular cartilage and bone. Thus, subchondral bone plays a role in osteochondral damage and osteoarthritis (Kawcak *et al*. 2001); the predilection sites of dyschondroplasia are the shoulder and fetlock in the foreleg, the stifle and hock in the hind leg and the cervical spine (Jeffcott 1991).
- physitis (physeal dysplasia or epiphysitis);
- angular limb deformities and flexural deformity (contracted tendons); and
- vertebral abnormalities (wobbler disease; see also 'Vitamin E', Chapter 4).

The probable causes of DOD include:

- congenital malpositioning and/or malnutrition;
- high rates of postnatal growth with unbalanced diets;
- biomechanical trauma;
- endocrinological dysfunction;
- toxicity, including iodine toxicosis; and
- heredity.

Many investigators have demonstrated heredity in several breeds (Sandgren 1993). N.B. Whereas most colts with signs are destroyed, most fillies are retained. Sire index is related to DOD, yet stallions having a propensity to sire affected offspring might not show signs themselves.

Radiographic signs of joint disease in the distal tarsal joints were found by Björnsdóttir *et al*. (2000) in 30.3% of Icelandic riding horses. Lepeule *et al.* (2008) estimated that DOD was present in 66% of 392 Warmbloods, Standardbreds and TB weanling foals following examination of the front- and hind-limb digit, carpus, hock and stifle joints. The prevalence for a particular site differed amongst these breeds. Draft horses and ponies have a lower risk of osteochondrotic bone disorders (Stromberg 1979). Stock & Distl (2006) found some additive genetic correlations between radiographic findings in the limbs of 3725 Hanoverian Warmblood riding horses and performance parameters.

Ekman *et al.* (2009) and the Stockholm Workshop (2008) concluded that OC-like lesions occurred in almost every young foal of susceptible breeds and that areas of extracellular matrix degeneration, not unlike those seen in OC, occurred in all samples from foetal tissues; but a clearly defined histological lesion has not been identified in the equine foetus (Lecocq *et al.* 2008). Lesions of OC have been detected 0 to 3 months of age (Sandgren 1993). OC results from a failure of endochondral ossification during skeletal growth. It is initiated by formation of foci of retained cartilage in subchondral bone, including subchondral fractures and cysts, OCD, cartilage flaps and synovitis. Articular cartilage is known to be an avascular tissue, whereas growth cartilage possesses a temporary blood supply. The incidence of OC is related to a disruption in the blood supply to this cartilage. Olstad *et al.* (2008a,b) showed that necrotic growth cartilage, resisting conversion to bone, occurred proximal to necrotic blood vessels where these vessels traversed the ossification front to enter the distal terminus of canals of the tarsus and femur in foals from 0 to 7 weeks of age from parents with OC. These pathological changes occur especially in regions of growth with a prolonged dependence on blood supply. When the abnormalities of OC have progressed to OCD then cracks and fissures occur in the cartilage. If this cartilage loosens, bone chips are often present in the joint or synovial space (McIlwraith 2004b).

Reactive oxygen species (ROS) are capable of degrading many components of the joint in the presence of

insufficient antioxidant defences. Dimock *et al*. (2000) found a higher amino-acid residue carbonyl (ketone) content and a marginally higher antioxidant content of the diseased synovial fluid protein in leg joints. Whether a higher carbonyl level is a precursor to damage has to be determined, as oxidative damage increases during the disease process and might accelerate it. Alternatively the higher antioxidant status of diseased joints could reflect an increase in the acute-phase protein content during an inflammatory response.

The proliferating chondrocytes produce and secrete the extracellular cartilage matrix of proteoglycans and collagen type II. In contrast to the proteoglycan component, the collagen component of cartilage is known to have very limited capacity for repair, owing to an extremely low turnover rate. Brama *et al*. (2000), using two-year-old TBs, showed that strenuous exercise caused alterations, specifically in the collagen network of the fetlock joint (metacarpo-phalangeal) articular cartilage, including a sharp decrease in hydroxylysylpyridinoline cross-linking. Exercise and time, 0–18 months of age, affected the site and depth associated variations in proteoglycan and collagen contents and cellular orientation of this joint (Brama *et al.* 2009b). Brama *et al.* (2009a) later found that habitual low-intensity loading elicited a greater positive quantitative response in bone mineral density (BMD) than high-intensity low-frequency loading of this joint between 7 days and 5 to 11 months of age.

Cu is a component of the enzyme lysyl oxidase required in the protein cross-linking process. Amongst other causes, a dietary Cu deficiency is associated with a failure of capillary endothelial cells to penetrate the distal region of the hypertrophic zone. Hypertrophic chondrocytes secrete: (a) metalloproteinase enzymes including lysyl oxidase, and (b) a basic fibroblast growth factor (bFGF), which stimulates proliferation and migration of endothelial cells and so the angiogenesis of this region in the foetus. The capillary invasion appears to be inhibited by large carbohydrate meals, and the hypertrophic zone then fails to mature.

The primary lesions of retained cartilage are susceptible to further damage within the joint and secondary lesions of OC, or OCD, result. Thus, the immature region extends, osteoid does not form normally, so calcification is faulty and the bone is weakened. Subchondral fracture lines are apparent, loose pieces of tissue appear in the joint and synovitis develops. Bone fragments occur particularly at the distal extremity of the tibia (Jeffcott 1991). Secondary osteoarthrosis, occurring in the articulations of the cervical spine, causes stenosis of the vertebral canal and signs of ataxia (wobbler syndrome).

During the suckling phase, the incidence of DOD is slightly greater in colts than in fillies. It has been observed in several breeds, including ponies (the author, unpublished observations). Dyschondroplasia can lead to joint stiffness, joint distension (e.g. 'bog spavin'), angular limb deformities and flexural deformity. The first signs may occur in very young foals, shortly after weaning, or in yearlings and older animals, particularly with the biomechanical stress of training. However, the peak incidence seems to occur between weaning and the end of December in northern latitudes.

Effects of diet and exercise

Diets providing excessive amounts of soluble carbohydrate (digestible starch) and total energy (129% of NRC recommendations in 1989) given to foals from 130 days of age, caused widespread lesions of DCP (Savage *et al.* 1993a,b). Part of the energy increase was achieved with extra fat; therefore a raised postprandial insulin response is not necessarily involved (see below).

IGF-1 also functions in normal maturation and growth of articular cartilage. Higher plasma IGF-1 levels were noted in TB foals given a soluble carbohydrate supplement to pasture, compared with those given a supplement of slowly fermentable carbohydrate and fat. Staniar *et al*. (2001b) suggested that the supplement had reduced the risk of DOD. In comparison, Ropp *et al*. (2003) failed to find an effect on growth in Quarter Horse foals, or on their serum IGF-1, when given brome hay with a concentrate containing 103 g fat and 240 g starch per kg cf. 22.1 g fat and 339 g starch/kg. A fat-supplemented diet, cf. a carbohydrate diet, decreased plasma triacylglycerol concentration in Shetland ponies, associated with an increase in plasma LPL activity, whereas a glucose tolerance test led to a massive increase in the insulin response in the fat-supplemented ponies (Schmidt *et al*. 2001) (see below, 'Dietary fats and fibre', for the effects of fat on Ca absorption). Thus, it is unclear how fat supplements might influence risks of DOD developing (N.B. Plasma IGF-1 concentrations in 1880 TB horses in three continents were stable, compared with GH concentrations. The mean IGF-1 concentration in gonad-intact males was 336 ± 5.6 ng/mL, 303 ± 3.2 ng/mL in females and 302 ± 3.2 ng/mL in geldings (Noble *et al*. 2007).

A dietary protein level of 126% of NRC (1989) recommendations had no adverse effect, compared with the effects of the recommended amounts (Savage *et al.* 1993a). High protein diets do not seem to predispose foals to DCP. The author increased the protein allowance and quality of a foal-weaning diet bringing about an increase in growth

rate and a reduction in the clinical evidence of physitis, and flexural and angular deformities among successive crops at three TB studs (Frape 1989). Foals weaned at three to four months of age were given this diet *ad libitum* as a coarse mix diluted with 100 g molassed chaff/kg.

Glade *et al.* (1984) and Glade (1986) have advanced an intriguing explanation of the relationship between epiphysitis and energy and protein consumption at each meal. Excessive intakes suppress normal postprandial hyperthyroxaemia, because an intense insulin secretion stimulates T_3 formation from T_4, in turn inhibiting TSH and thus T_4 secretion. As T_4 is required for bone maturation, hypothyroidism is known to cause skeletal manifestations (see Iodine, Chapter 3) similar to epiphysitis and OCD, whereas insulin stimulates the formation of immature cartilage and high concentrations can inhibit growth hormone action. Glade considered that the episodic transient postprandial hypothyroidaemia, produced by a high carbohydrate meal, could cause DCP. The postulate requires further investigation of the postprandial insulin response, as only 17% hydrolyzable carbohydrate in a diet, given at the rate of 1.6 kg twice daily was adequate to cause maximum postprandial insulin and glucose responses in TB yearlings (Staniar *et al*. 2002). Powell *et al*. (2000) found that TBs given only 70% of their daily energy needs failed to produce a normal T_3 or T_4 response to the consumption of 1.0 kg oats as the morning meal, the expected response that occurred when they received adequate amounts of energy daily. The influence of short-term feed restriction could occur through a reduction in the rate of glucose uptake, especially in horses adapted to high roughage. Donabédian *et al.* (2006) concluded that neither a high balanced feeding level, nor even an increased rate of BW gain and its increased bone loading, alone, were sufficient to increase the risk of DOD in foals, but fast bone development, as high endomembranous or endochondral ossification rate was more critical.

Foals affected with hock OC tended to have heavier birth weights, a higher average rate of weight gain and be heavier at 1.5 years of age; but foals with stifle OC were taller at the withers (Sandgren 1993; van Weeren *et al*. 1999; McIlwraith 2004b). Feeding level itself does not seem to influence the rate at which foals and yearlings are affected by OC; but there is a positive correlation between fast growth and occurrence (Donabédian *et al.* 2008). Contracted tendons and physitis occur following the compensatory weight gain on abundant diets after a period of restricted growth caused by poor diet and/or inclement weather (Hintz *et al*. 1976a; Rooney 1984; Cymbaluk & Christison 1989a). Faster growing horses tend to be more prone to OC and are likely to have been heavier at birth (Sandgren 1993). The risks of foals developing DOD is reduced by the adoption of modest growth rates, but the provision of creep feed to reduce the stress of weaning, is recommended, so that the rate of growth is relatively constant throughout (NRC 2007).

The frequency and severity of lesions are less where foals are exercised. Such exercise is generally to be recommended, except where severe lesions already exist. With adequate dietary energy, Raub *et al.* (1989) demonstrated that as little as 20 min of medium trot 5 days a week between 147 and 255 days of age in TB and Quarter Horses was sufficient to increase the radiographic density of the medial side of the third metacarpal and to increase its circumference (the increase in density could reflect an increase in circumference). An increase in the breaking strength of the bones should then have been achieved. Several studies confirm the value, and risks, that attend exercise in this context. Bird *et al*. (2000) demonstrated that the introduction of exercise (4 km at 12 m/s to 1 km at 15 m/s) in 24-month-old TB horses increased the rate of proteoglycan synthesis by the chondrocytes in leg joints. Firth *et al*. (1999) examined the effect of exercise on trabecular thickening (sometimes referred to as osteosclerosis) in the third and radial carpal bones. Exercise increased the density of the subchondral cancellous architecture in regions underlying sites of cartilage degradation. The authors considered that the initiation of localised osteogenesis of cancellous bone was not a consequence of microfractures, but could be purely physiological, occurring without loss of osseous tissue integrity. This hypertrophic stiffening response reduces the shock-absorbing capability, inducing destructive loading of overlying articular cartilage. The critical limits and timing of exercise need to be determined individually for horses. Adams & Silver (2009) reviewed the evidence for early enhanced exercise in relation to limb joint health.

Angular deformities, growth and exercise

The aetiology, management, options for correction of angular limb deformities have been reviewed (Witte & Hunt 2009). Brauer *et al*. (1999) analysed the intracarpal angular deviations of horse legs from radiographs. Horses possess a natural growth-correction mechanism that spontaneously corrects many deformities in foals born with abnormal conformation. The physeal growth curve states that, short of pathological limits, cells of the physis that are loaded more heavily grow faster and those loaded less heavily grow more slowly. As the concave side of the physis is the more heavily loaded, the acceleration in growth on

that side of the limb tends to lengthen the side until it is no longer concave, or until the load on the physis is balanced along the axis of the limb. Lesions that appear during the first 5 months *post partum* regress, although some will remain and become clinically important in the mature animal. No resolution of substantial lesions occurs after one year of age (Donabédian *et al.* 2008). Leibsle *et al*. (2005) found only 3% of newborn TB foals had 'straight' carpal conformation, cf. 55% with correct fetlocks. At 1.0–1.5 years of age only 7% of the same foals were considered straight at the carpus ('knee'), while 79% were straight at the fetlock. Sondergaard (2003) observed an age-related decrease in radiographic OCD from weaning through 2 years of age. Although many lesions were temporary, the 'age of no return' when regression was less likely to occur, differed between joints: hock was after 5 months and the stifle after 8–12 months of age (Dik *et al*. 1999).

From birth, joint cartilage adapts and develops according to the loading experienced. Brommer *et al.* (2005) using Warmbloods and TBs found that the thickness of joint cartilage did not change beyond 5 months of age. The loading, its heterogeneity and adaptive modulus, measured as **Young's modulus**, differed at two positions on the proximal articular surface of the first phalanx, in the foetus and up to maturity. At age 18 months no further adaptation occurs. This emphasizes the necessity to optimise joint loading during early life in order to create an optimal biomechanical quality of articular cartilage. The beneficial response to loading occurs only where there is dynamic loading, where the pressure is applied and removed intermittently (i.e. with exercise and rest). Static loading retards growth. At the pathological limit (extreme deformity), growth stops and the angulation worsens with continued growth on the convex side of the physis. Correction then requires surgery and or corrective foot care and exercise manipulation (Bramlage 1999).

Controlled exercise training from the age of 10 days until 18 months reduced glycosaminoglycan and collagen levels, but had no adverse effect on cartilage degeneration of the proximal phalanx. The controlled exercise increased hydroxylysylpyridinoline and pentosidine crosslinks (Van Weeren *et al.* 2008) and cross-links in collagen of subchondral bone (Brama *et al.* 2002) and had no adverse effect on the mid-carpal joint (Kim *et al.* 2009). Voluntary exercise on pasture tends to protect articular cartilage from development of OC lesions, cf. complete confinement (Barneveld & van Weeren 1999), and normal exercise also benefits bone mineral content of weanlings (Hiney *et al.* 2004) and of 2-year-old fillies (Jackson *et al.* 2003b). Intensive exercise can have adverse consequences. Enforced exercise of 2-month-old foals for 12 months reduced the number of thyroid C cells required for calcitonin secretion, involved in Ca homeostasis, and led to lowered BMD of the hind cannon bone (Ueki *et al.* 2003). Box-stalled foals required to undertake gallop sprints up to 5 months of age, and then turned out to pasture until 11 months of age, developed articular cartilage with a lower glycosaminoglycan content and a reduction in chondrocyte hypermetabolism, cf. unexercised, pastured foals. These observations indicate that enforced exercise reduced the vitality of the chondrocytes, even though the exercise increases BMD (Barneveld *et al.* 1999).

Training schedules are very important to allow leg bones to adjust to the stresses of racing. Parkin *et al.* (2004) concluded that racehorses should gallop between 201 m (1 furlong) and 1609 m (8 furlongs) per week to reduce the risk of fracture of the lateral condyle of the third metacarpus/metatarsus.

Dietary composition and insulin response of young horses

There has been a tendency for TB and many other breeders to provide weaned foals and yearlings with less dietary protein than is recommended by the NRC (2007). O'Donohue (1991) found that protein was the only dietary component for which the daily allowance of short yearlings on 46 Irish stud farms, was less than that recommended by the NRC. The effect of this will have been to lower the rate of growth.

O'Donohue indicated, in agreement with the author's experience, that the peak clinical expression of DOD occurred between weaning and the end of December, during the introduction of concentrate feeds. Bone mineral content was lower in weanlings and yearlings given a 15% protein supplement containing per kg 10.1 g Ca, 412 g NDF and 104 g fat, cf. that providing 7.7 g Ca, 153 g NDF and 24 g fat, as adjuncts to a blue grass/white clover pasture, or mixed grass/legume hay in winter (Hoffman *et al*. 1999a). There was a slightly greater incidence of angular and flexural deformities during June and angular deformities during January in foals given the supplement containing more fibre and fat. The formation of Ca soaps and fibre-capturing cations in the gut, might have adversely limited the supply of bone mineral. Moreover, a fat supplement has been shown previously to contribute to the incidence of OCD (Savage *et al.* 1993a).

It was considered that insulin resistance may be a contributory factor in OCD. OCD-affected foals have higher plasma insulin (Ralston 1996) and higher plasma glucose and insulin (Pagan 2003) concentrations than healthy

controls, and Treiber *et al*. (2005a) observed a reduction in insulin sensitivity in weanling foals given a high non-fibre carbohydrate diet cf. those given a fat–fibre concentrate. One consequence of rising labour costs has been to decrease the number of feeds per day. If energy-rich feeds are fed in large amounts once a day, rather than in smaller amounts in three meals, there may be an increasing risk of DOD from very large insulin responses. O'Donohue (1991) found that once daily feeding of TB short yearlings was gaining in popularity, with a tendency for DOD to be more prevalent where it was practiced, and that 67% of the 1711 foals he examined in Ireland showed some signs of DOD, although only 11.3% were deemed to need treatment. Of the treated cases, angular limb deformities and physeal dysplasia together constituted 72.9% of the total.

Ponies generally have a greater insulin-resistance than horses (Rijnen & van der Kolk 2003), yet have a lower incidence of DOD (Stromberg 1979). Weanling foals given a high starch diet cf. those given a low starch, high oil, diet did not develop poorer bone mineralization or more new OCD lesions (Ott *et al.* 2005) and the foals receiving the low starch, high corn oil, diet were less able to metabolize dietary glucose. The role of carbohydrate metabolism in the development of OCD is unclear. Concentrates containing an adequate amount of minerals given in a well regulated system do not seem to increase the risk of OCD. A balanced diet is necessary, especially up to 4 years of age, as the mineral density of both subchondral and trabecular bone should increase in horses up to that age (van der Harst *et al.* 2005).

Vitamin and mineral supplements

O'Donohue (1991) found that vitamin and mineral supplementation in studs was a 'hit and miss' affair. Supplementary allowances of vitamins A and D ranged from 0 to 18 times the NRC (2007) recommendations.

Minerals, trace elements and DOD

Excessive dietary P consistently produces lesions of DCP (Chapter 3), although clinical signs of NSHP do not normally occur when adequate dietary Ca is provided. Savage *et al.* (1993b) gave 130-day-old foals a diet providing 388% of NRC (1989) P recommendations, but 100% of the Ca recommendations, and found severe lesions of DCP without clinical signs of NSHP. The incidence of DCP was much greater than in those given 342% of the Ca with 100% of the P recommendations, or in those given 100% of both recommendations. The high P diet would have a lower cation : anion balance (see Chapter 9 and Appendix D) and could have induced bone Ca mobilization. A diet of low and negative dietary cation–anion difference (DCAD) causes chronic metabolic acidosis and an elevation of plasma PTH that stimulates the conversion of 25-$(OH)D_3$ to 1,25-$(OH)_2D_3$ in the kidney. This regulates the active transport of Ca across the intestine. If growing horses are receiving adequate dietary Ca, but a diet of low DCAD, then the stimulation to intestinal absorption of Ca compensates for the increased urinary Ca loss. Surprisingly, this has led in growing horses to a greater positive Ca balance than occurs with a diet providing the same adequate amounts of Ca, but a high DCAD (Cooper *et al*. 1999b). The importance of adequate dietary Ca is paramount.

Trace elements

Pregnant mare and foal diets containing <10 mg Cu/kg may cause DOD and flexural deformity. Lysyl oxidase is a Cu-containing enzyme required for the cross-linking of protein chains in elastin and collagen of cartilage through the lysine residues, by oxidizing the ε-amino group. Failure of this function disrupts normal bone cartilage development. Dietary Cu supplements for the pregnant mare and concentrations of up to 30–45 mg Cu/kg in the weanling's feed could decrease the incidence of DCP and physitis in growing horses. These levels of Cu are safe and well below a toxic threshold, as the horse has a considerable resistance to chronic Cu toxicity. Cu supplementation of the lactating mare has no measurable effect on the low Cu content of the milk (see also Chapters 3 and 7).

Glucosamine, methyl sulphonyl methane and chondroitin sulphate supplementation

Glucosamine and chondroitin sulphate

The rate-limiting step in the glycosylation of many proteins is probably the formation of glucosamine-6-phosphate catalyzed by the enzyme glucosamine synthetase. Glucosamine, modified as N-acetylglucosamine, constitutes half the molecular composition of hyaluronic acid. By isomerisation, glucosamine can be converted to galactosamine and so can contribute half the molecular composition of chondroitin sulphate. Glucosamine is an amino monosaccharide that the body is able to synthesise. Further metabolic modification of glucosamine generates intermediate substrates for chondrocytes and synoviocytes to synthesize various glycosaminoglycan (GAG) compounds, including hyaluronan, keratan sulphate and chondroitin sulphate (Neil *et al.* 2005).

Increased proteolytic enzyme activity is a major factor responsible for the degradation of the extracellular matrix of articular cartilage. Glucosamine and chondroitin

sulphate stimulate both proteoglycan and collagen synthesis, so *in vivo* they may help regeneration of cartilage matrix and proteoglycan synthesis and may reduce inflammation within damaged joints (Platt 2001). The addition of glucosamine to equine articular cartilage discs treated *in vitro* with lipopolysaccharide (LPS), induced decreased gelatinase and collagenase activity and inhibited nitric oxide production, so inhibiting prostaglandin release (Fenton *et al*. 1999), indicating that glucosamine may exert an anti-inflammatory effect within explants (Chan *et al.* 2005). Glucosamine and chondroitin sulphate under similar conditions limit GAG degradation and enhance GAG synthesis when explants are incubated with LPS, or preconditioned with IL-1 (Dechant *et al.* 2005). Byron *et al.* (2008) reported that, at clinically relevant concentrations, glucosamine had limited effects on chondrocyte and synoviocyte metabolism *in vitro*, although it did have some anti-inflammatory activity on IL-1–stimulated articular cells. These *in vitro* data do not assure *in vivo* efficacy.

A large proportion of the absorbed glucosamine molecule is modified by the liver and utilized for energy production, hence the most effective dose could be high. The bioavailability in horses of glucosamine-HCl (125 mg/kg BW) is much lower than that of low molecular weight chondroitin sulphate (3 g each of 8-kDa and 16.9-kDa forms), i.e. 2.5% cf. 32% and 22% (Du *et al.* 2004); although Laverty *et al.* (2005) achieved a bioavailability of glucosamine-HCl of 5.9% when 20 mg/kg BW were given by nasogastric administration. The concentrations detectable in the synovial fluid after oral administration were, however, much lower than those previously described for *in vitro* studies (Dechant *et al.* 2005). A salutary note is provided by Oke *et al.* (2006) who found that of 23 commercial products only two provided the estimated need of 10 g/day glucosamine for the averaged-sized mature horse. Moreover, the quality of *in vivo* efficacy studies on glucosamine and chondroitin is low, prohibiting meaningful interpretation of most results (Pearson & Lindinger 2009). Although both compounds seem to reduce pain from osteoarthritis, Gupta *et al.* (2009) showed that 480 mg undenatured type II collagen, providing 120 mg active collagen daily, was much more effective.

As well as glucosamine, mannosamine has been shown to inhibit equine 'knee' cartilage degradation, *in vitro*, in the presence of LPS, and mannosamine has been effective at less than half the concentration of glucosamine (Mello *et al*. 2001). Electron microscopic examination in humans shows that the consumption of a glucosamine supplement, cf. placebo, can improve physical performance and knee cartilage health, reversing cartilage degeneration.

Methyl sulphonyl methane and green-lipped mussel (Perna canaliculus) *extract*

Other compounds characterised to have 'chondroprotective' effects include green-lipped mussel (*P. canaliculus*) extract and methyl sulphonyl methane (MSM) (NRC 2007). The mussel extract consists of various GAG compounds and omega-3 fatty acids, although clinical responses have been equivocal (Cobb & Ernst 2005). MSM is a source of bioavailable sulphur. Anecdotal evidence indicates that it relieves inflammation and pain due to arthritis and muscular disorders, although there has been little equine evidence to show that it is absorbed and utilized. Pratt *et al*. (2001) demonstrated in adult horses that 55% of S of MSM origin was absorbed and large amounts accumulated in the blood. The technique employed indicated that the MSM was probably absorbed intact (see also Chapter 5 and 'Control of growth abnormalities', Chapter 7).

NRC (2007) concludes there is a need for appropriately designed long-term studies to determine the utility of oral joint supplements in the prevention of osteoarthritis. The treatment of osteoarthritis with dietary supplements has been reviewed by Dechant & Baxter (2007).

Measurement of changes in bone formation

Osteocalcin is a noncollagenous protein, synthesised by osteoblasts, which is incorporated into new bone matrix and is released into the circulation during bone resorption, so that the serum level reflects bone turnover. Collagen I is a major constitutive protein of bone matrix. Type I collagen carboxy-terminal telopeptide (CTX-1) is a byproduct of collagen I degradation by osteoclasts and is therefore a marker of bone resorption. In cartilage collagen II is synthesised by chondrocytes as procollagen, and is cleaved outside the cells resulting in the secretion of a C-terminal propeptide (CPII) and N-terminal propeptide. CPII is a serum marker of cartilage formation. Donabédian *et al.* (2008) found that plasma osteocalcin concentration at 2 weeks of age was strongly correlated with limb joint OC at 12 months of age.

Osteoarthritis and intra-articular treatment

A reduction of pain and lameness caused by osteoarthritis is frequently achieved by intra-articular treatment with glucocorticoids. There is weak evidence to support the co-administration of hyaluronan, a glycosaminoglycan, in achieving this goal (McCluskey 2007). Frisbie *et al.*

(2009) found that 250 mg polysulfated glycosaminoglycan, or 20 mg hyaluronan, each administered intra-articularly on days 14, 21 and 28 following the arthroscopic induction of osteoarthritis in a middle carpal joint, had beneficial disease-modifying effects in horses. Williams (2007) reviewed the evidence concerning the optimum average molecular weight of hyaluronic acid (HA) used for intra-articular administration. The beneficial effects of HA supplementation are attributable to its anti-inflammatory effects, improvement in the viscoelastic properties of the synovial fluid, and interaction with the synovial membrane affecting pain transmission and joint metabolism. However, the optimum molecular weight of HA for these purposes has not been demonstrated.

Summary of control of DOD

In the rearing of foals:

- Avoid large meals rich in glucose-yielding carbohydrate, but ensure that protein requirements are met with balanced amino acids.
- Provide adequate dietary Cu, but with a trace-element balance.
- Provide adequate dietary Ca with a correct Ca : P ratio.
- Provide adequate exercise daily.
- Avoid breeding from stallions genetically predisposed to produce DCP-affected foals.
- Where there are risks of DCP, the foals should be given no more than the recommended NRC rates of DE intake.
- Where clinical DOD occurs, decrease the energy intake of foals and minimize the joint trauma by restricting the foal to hand-walking until signs disappear, and/or provide farrier assistance.
- In studs where good labour is scarce, introduce automated feeding to avoid large meals, but ensure careful daily observation of each foal by experienced persons, for signs of DOD or obesity.

STUDY QUESTIONS

(1) What factors are important in selecting a suitable pasture for weaned foals?
(2) What action should be taken with a foal presenting mild signs of orthopaedic disease, or with moderate signs, at an early age, or postweaning?

FURTHER READING

Brama, P.A.J., Firth, E.C., van Weeren, P.R., *et al.* (2009) Influence of intensity and changes of physical activity on bone mineral density of immature equine subchondral bone. *Equine Veterinary Journal*, **41**, 564–71.

Witte, S. & Hunt, R. (2009) A review of angular limb deformities. *Equine Veterinary Education*, **21**, 378–87.

9
Feeding for Performance and the Metabolism of Nutrients During Exercise

But if you intend the next day to give him an heat (to which I now bend mine aim) you shall then only give him a quart of sweet oats and as soon as they are eaten, put on his bridle, and tie up his head, not forgetting all by ceremonies before declared.

T. De Gray 1639

Feed costs are major outgoings in the breeding, training and use of sport horses. Corbally (1995) found that, in the Irish sport-horse-breeding industry and at equestrian sports centres, feed was the second largest cost after labour, representing 16% and 19.4% of the total cost, respectively, when the costs attributable to amortization of buildings, and brood mares and the current expenditure on labour, stud, veterinary surgeons, schooling, bedding, farrier, showing, tack and equipment and registration were also included. It is important to the economy of the industry that feed has the optimum composition and is used judiciously. This chapter considers the principles underlying the feeding and metabolism of nutrients, so that composition of feed and methods of its provision can be manipulated to optimize performance and minimize the risks to the horse and rider.

WORK AND ENERGY EXPENDITURE

A major role of feed for working horses is the conversion of the chemical energy of feed into locomotion at speeds varying from 160 to over 900 m/min (6–35 mph) for distances varying from 1–150 km or more. This enormous range might superficially lead to equal fatigue in fit horses, but quite different processes of physiology are involved at the two extremes. At the one extreme a flat race of 6–8 furlongs (1.2–1.6 km) would *theoretically* increase the day's energy needs by a mere 4% (Plate 9.1), a barely perceptible effect, whereas at the other they would be increased by five- to six-fold. Training regimes recognize this, as well as the different responses of breeds to contrasting forms of work. Distinctive training regimes induce profound and dissimilar physiological changes in the attainment of fitness, and diets must be formulated to conform with their different needs. First, an adequate and optimum nutrition for a particular purpose implies an optimum supply of nutrients to each tissue and the efficient disposal of waste products (Snow *et al.* 1983a).

In sprint races, horses obtain much of their muscular energy from anaerobic pathways of respiration, whereas in extended work, such as endurance competitions, energy is derived almost exclusively through aerobic pathways. (Anaerobic respiration is the breakdown of organic nutrients in the absence of oxygen but with the release of energy captured by ATP.) A day's hunting, with episodes of hill climbing, cantering and jumping, and periods of waiting and walking, combines both processes and rates of energy expenditure. The expenditure by a hunter carrying a heavy huntsman during a long day exceeds the average daily consumption of feed energy by several-fold. The processes of energy metabolism are summarized here, as a greater understanding of them should allow more rational feeding, and foster comprehension of each development in the feeding of working horses.

What is work?

It is appropriate here to review the meaning of the terms 'force', 'power', 'work' and related terms and to see how they have been applied to the measurable work of horses. If a horse was a smooth ball on a frictionless horizontal surface, no energy would be required for it to continue moving at a constant speed in a straight line. Fortunately, for both the horse and feed manufacturer, all movement of the horse involves friction, both in the horse's motion (an



Plate 9.1 Rathgorman, ridden by Chris Bell, cantering 8 furlongs (1.6 km) on the all-weather track at Dunkeswick, West Yorkshire, watched by trainer Michael W. Dickinson. As part of the training for National Hunt racing, the horses canter five days per week. If a horse races every 7–21 days it has an easy canter on the day before and for several days after a race. A regular work programme is maintained during the racing season but with some variety in location, scenery and type of work.

assumed value of 2 J to move 1 kg BW 1 m horizontally) and in anything the horse might be pulling. It requires force to overcome the friction, and the force, acting over a given distance, equals the work output. If hill climbs intervene, the force of gravity has to be overcome as well. The extra energy required above maintenance is:

$$2\text{J}[\text{BW (kg)} \times \text{horizontal distance (m)}] + \frac{\text{work done}}{0.298}$$

where 0.298 represents the ratio of work done pulling/net energy used (cf. gross efficiency, Table 9.1). Force is measured in newtons (N), joules per metre (J m^{-1}), as the force necessary to accelerate 1 kg at a rate of 1 m/s^2 (kg m. s^{-2}). Work output is measured in newtons, N, for the distance in metres over which the force is applied, expressed as joules, N m = J.

The working horse

One of the most traditional forms of work for a horse must be that of pulling a plough. Several groups of workers have accurately measured the work of ploughing. The power output, measured in watts (J. s^{-1}) per unit live weight, in ploughing 4 h/day was similar between two Jersey crossbred oxen (323 kg BW per head) and four heavy donkeys (170 kg BW per head). Donkeys tended to plough at a slower pace than the oxen (Nengomasha *et al*. 1999b). Pérez *et al.* (1996) reported on five Chilean crossbred draught horses weighing 547 kg, pulling a mould-board plough, cutting a furrow of depth and width 12.6 and 22.3 cm, respectively. Of course, the measured performance is influenced by the soil characteristics, but the values they obtained (Table 9.1) agree well with those obtained by others and are informative to the nutritionist.

The riding horse

Energy expenditure and work type

Endurance work is at the rate of 154–224 m/min (5.75–8.35 mph) over variable slopes, whereas flat racing is at about 940 m/min (35 mph). For comparison, the stagecoach horse, pulling a heavy load, covered about 40 km (25 miles) at speeds similar to those required of endurance horses: 200–214 m/min (7.5–8 mph). Strenuous effort tends to depress appetite, so recovery from extreme effort requires several days for reserves to be replenished.

Table 9.1 Mean draught load, distance travelled, work done and net energy expended, in addition to maintenance, during a six-hour day, by five Chilean draught horses ploughing a field (Pérez *et al.* 1996). The horses were exercised under submaximal conditions, although there was a significant increase in blood cortisol concentration.

Live weight (kg)	547
Force applied, or draught load[1] (N)	905
Distance travelled (km)	13.6
Speed (m/s)[2]	0.93
Power developed (kJ/s or kW, i.e. kilowatts)	0.83
Work done in pulling above maintenance (kJ/horse)	12 276
Work done over six-hour day (kJ/kg $BW^{0.75}$)[3]	108
Estimated net energy for work, including horse moving itself (kJ/day)[4]	130 584
Estimated net energy used for work, including horse movement (kJ/kg $BW^{0.75}$)	1 153
Estimated gross efficiency of work, including horse movement, but excluding maintenance (%)	9.4
Net energy for work expressed as multiple of maintenance	2.24

[1]Mean force exerted by the five horses was equivalent to 17.7% of their body weight, or a draught power of 780 watts, Js^{-1}.

[2]18.5 hours would be required to plough 1 ha.

[3]This is greater than that achieved by oxen, as the horse requires about two-thirds of the time to achieve a similar task.

[4]Energy requirement for this work of six-hour is about 125% of that required for two-hour medium trot daily including rider and maintenance.

The data in Figure 6.4 and Table 6.8 show that the capacity of larger horses for feed is proportionally less than that of smaller horses, so that after undertaking strenuous work they require a longer period for recovery, especially if it is assumed that the weight of the rider is proportional to the weight of the horse. The information in Table 6.3 is based on experimental results, and, although there has been some extrapolation, it does indicate greater demands for feed energy than the theoretical NRC estimates for endurance work given in Table 6.8, which do, however, exceed the INRA estimates in Figures 6.11a–d.

Blaxter (1962) calculated that the energy expenditure for vertical effort in horses, in addition to any horizontal effort, amounted to 17 times that expended in horizontal movement, above the costs of energy metabolism at rest. A 400 kg horse is calculated to expend 0.67 kJ/m in horizontal work and 11.4 kJ/m vertically. Exercise over uneven and hilly ground can therefore be much more arduous than that on the flat. Sprint work on the flat must be considered a special case, as it is almost entirely confined to young horses which are still growing. Extended work, for which there is somewhat more published experimental evidence, is generally undertaken by older horses.

Work entails an increase in nutrient supply to the muscles, and in converting glucose or free fatty acids to high-energy phosphate compounds such as ATP and creatine phosphate or phosphocreatine (PCr), which the muscles use as an immediate source of energy. There is an increase in the rate of production of waste products, more particularly carbon dioxide and heat. The supply of nutrients and the effective disposal of wastes entail large physiological adjustments, which training seeks to encourage. The most critical feeds for work are those that provide energy, water and electrolytes (those elements that in solution carry an electrical charge), including sodium, potassium, magnesium, calcium, chloride and phosphate.

ENERGY SUBSTRATES AND THEIR EXPENDITURE

Most potential energy for muscular work is absorbed from the intestinal tract as glucose, VFAs (acetic, propionic, butyric and smaller quantities of related acids), longer-chain fatty acids, neutral fats and amino acids. Absorbed glucose, propionate and glucogenic amino acids are potential sources of blood glucose and of liver glycogen. Absorbed long-chain fatty acids, neutral fats, ketogenic amino acids and particularly acetate and butyrate, are potential sources of blood fats and fatty acids, storage fat and acetyl-CoA. Blood glucose and its precursors are also,

of course, potential sources of body fat through the key substance acetyl-CoA.

ATP and PCr formation and use

The liver, while storing energy in the form of glycogen and fat (and also as protein), serves the vital role of maintaining normal levels of blood glucose through the breakdown and re-storage of glycogen. Muscle cells also store glycogen and form high-energy phosphate compounds (ATP and PCr) necessary for muscular relaxation and contraction, by drawing on blood glucose and fatty acids as fuels. The complete release of chemical energy from these compounds requires a supply of oxygen (O_2) reaching the muscle cells through the arteries. However, an immediate and rapid release of energy, particularly important in short sprint races, can be achieved through the process of glycolysis, in which glucose is broken down to pyruvate in the muscle cell without the consumption of O_2, and also by the release of energy from previously stored ATP and PCr. The further and complete breakdown of pyruvate and of fatty acids demands the presence of O_2 and this process takes place exclusively in the mitochondria, through the agency of what are known as β-oxidation of fatty acids and the TCA cycle.

ATP resynthesis

Energy for muscle contraction is derived from PCr and ATP which are formed from the energy derived during the combustion of glucose and fatty acids. TBs possess a high proportion of PCr-rich fast-twitch fibres in skeletal muscles (Harris & Hultman 1992a), so that loss of adenine nucleotides during intense exercise is greater than in man (see 'Ammonia and the alanine vehicle', this chapter). After repeated gallops, a 50% loss of ATP was recorded (Harris *et al.* 1991c; Sewell & Harris 1991; Sewell *et al.* 1992a), associated with fatigue and a decrease in running speed (Harris *et al.* 1991b), and with lower glycolytic rates and muscle ATP:ADP ratios. The accumulation of ADP stimulates adenine nucleotides degradation to inosine monophosphate (IMP) with an increase in plasma NH_3 concentration. There is a critical pH below which ADP rephosphorylation declines (see 'Ammonia and the alanine vehicle', this chapter), PCr acting both as an intracellular buffer and a reservoir for this rephosphorylation. Supplementation of human subjects with creatine monohydrate has been shown to increase muscle total creatine and PCr contents (Harris *et al.* 1992).Aerobic horse training with, or without, daily supplementation of 75 g creatine monohydrate caused significant hypertrophy of the longissimus dorsi muscle and a reduction in the thickness of the fat layer (D'Angelis *et al.* 2007). No beneficial effect from creatine supplementation on the body composition was detected over 90 days. The energy reserves for these synthetic processes depend upon training, diet and the inherent characteristics of the horse. The slow rate of ATP resynthesis, when supported by FFA oxidation, means that inadequate glycogen storage in active muscle fibres causes early fatigue despite an abundance of fatty acids.

TCA cycle

The operation of the TCA cycle in the mitochondria requires the diffusion of O_2 to these organelles and the removal of CO_2 to the blood. During light work, this process proceeds smoothly, and with each turn of the cycle one unit of oxaloacetate, which is required for the subsequent metabolism of acetyl-CoA in the presence of O_2, is produced. However, when larger quantities of oxaloacetate are present, the metabolism of acetyl-CoA is likely to proceed more rapidly. When larger quantities of fatty acids are dissimilated to acetyl-CoA, the cycle must turn at an accelerating rate. This is also achieved by the provision of extra quantities of oxaloacetate from outside the mitochondrion. There must, therefore, be adequate quantities of pyruvate requiring an ample supply of glucose, lactate, glucogenic amino acids or even glycerol. During extended work, the trained healthy horse finds no difficulty in breaking down fatty acids to CO_2, as sufficient O_2 is taken in by normal respiration. In fact, such work leads to an accumulation of glycerol, signifying that it is not called on to form pyruvate units in any great quantities. If the utilization of fatty acids was interrupted, this would probably be expressed as a retarded metabolism of acetyl-CoA and there would be a build-up of blood ketones (acetoacetate and 3-hydroxybutyrate).

Work in Newmarket (Frape *et al.* 1979) has shown that these ketones accumulate in the plasma only after work stops (Figure 9.1), possibly implying that no bottleneck occurs to the complete combustion of fats. This post-exercise rise in plasma ketones probably reflects the redistribution of blood from muscle to adipose tissue from which NEFAs will then be flushed, rapidly increasing the plasma concentration of their metabolites, owing to a reduced need for their cellular consumption. Hyperlipidaemia, recognized in starving horses, reflects a blocking of fat metabolism in animals relying overwhelmingly on residual fat stores, in the relative absence of carbohydrate substrates. Enzymes required for this fat breakdown can also be depleted in an associated deficiency of dietary protein. Figure 9.2 gives a brief account of the pathways by which

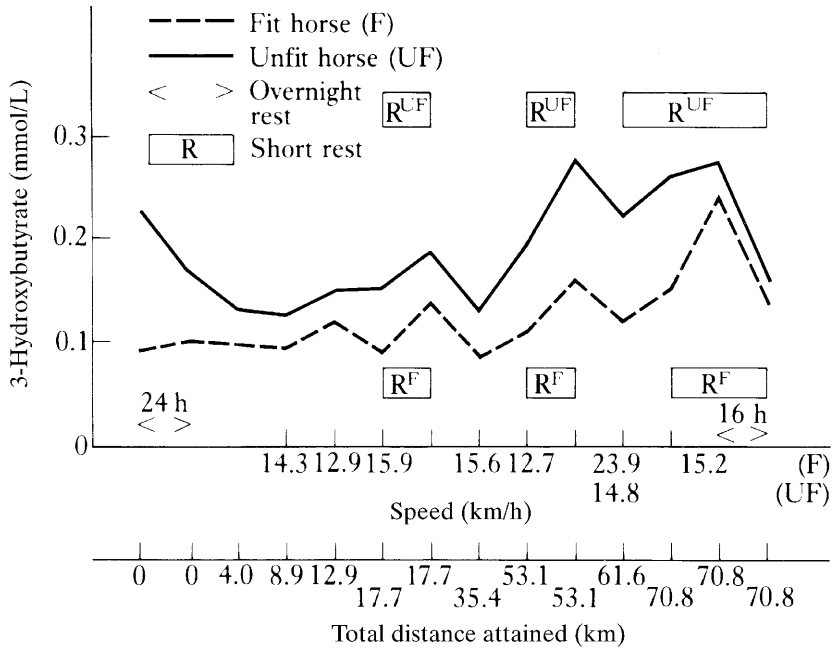


Figure 9.1 Effect of rest periods during an endurance ride on plasma 3-hydroxybutyrate in a fit and an unfit horse (Frape *et al.* 1979).

energy sources are metabolized in horse muscle cells and liver.

Glycogen, glucose, FFAs and VFAs

The amount of muscle and liver glycogen stored at any one time is variable, as reserves are considerably depleted during extended exercise and are replenished only over several meals. Nevertheless, the author calculates that, on average, liver reserves of glycogen are 5–10% of those in skeletal muscle. Glycogenolysis, or the breakdown of glycogen, continues following the completion of intensive exercise. Recovery of these glycogen stores is promoted by giving a meal of 1–2 kg cereal 1.5 hours following exercise, or by the i.v. infusion of 6 g glucose/kg BW, 30 min post-exercise. This can have implications for horses competing on successive days. On the other hand, in trained horses, glycogen stores do not limit sprint performance, as training can increase glycogen capacity of limb muscles by 39% (Guy & Snow 1977a).

With increasing rates of energy expenditure, the preference for glucose as a substrate increases, although the proportions of glucose and FFA used change rapidly with distance. Glycogen utilization rates of 2.68 and 1.06 mmol glucosyl units/kg dry muscle per s with total consumption of 27.3 and 32.5% of the initial store were caused, respectively, by 800- and 2000-m gallops (Harris *et al.* 1987). This suggests that over the greater distance, at a somewhat lower velocity, (aerobic) combustion of FFA increases considerably as a proportion of total energy expenditure.

In addition to FFA derived from the hydrolysis of fat stored in adipocytes, VFAs absorbed from the hind-gut are an important energy source. The contribution of acetate to oxidation in the hind limb was reduced from 32% in horses given roughage to 21% in those given a diet with an oats : roughage ratio of 0.52 : 0.48 (Pethick *et al.* 1993). This is one reason why higher roughage intakes are recommended for endurance horses (see also 'Sweat and dehydration', this chapter).

Pyruvate–lactate: the oxygen debt

The anaerobic production of energy by glycolysis would soon be halted by an excessive accumulation of pyruvate. A cunning mechanism has therefore evolved in which pyruvate is reversibly converted to lactate as an intermediate 'waste' product. This reduction even more importantly oxidizes NADH, essential for triggering an important glycolytic step. Therefore, in sprint racing, lactate diffuses into the blood from the muscle cell and there accumulates

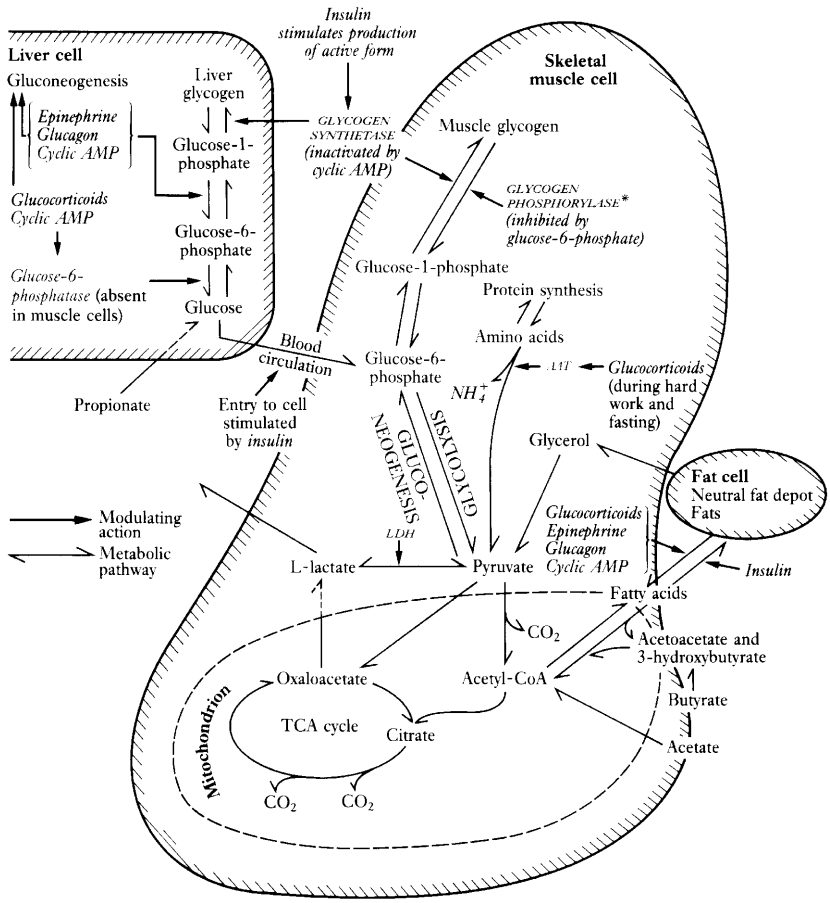


Figure 9.2 Major pathways of energy metabolism in muscle and liver cells and their modulation by the endocrine system (intermediate steps have been excluded). Glycolysis: the oxidation of glycogen or glucose to pyruvate and lactate. Gluconeogenesis: the formation of glucose or glycogen from non-carbohydrate sources. The principal substrates for this are glucogenic amino acids, lactate, pyruvate, glycerol and, to a lesser extent, propionate.
*Bursts of muscular action with the secretion of glucagon and epinephrine and nervous stimulation of the release of Ca^{2+} initiate the formation of cyclic adenosine monophosphate (AMP) and then the phosphorylation and activation of glycogen phosphorylase.

until sufficient O_2 is available for its hepatic conversion to pyruvate. This mechanism allows more energy to be obtained by glycolysis than would otherwise be possible. As the O_2 debt has eventually to be repaid, the yield of energy per unit volume of O_2 consumption, owing to anaerobic processes, is only half that achieved by aerobic processes. However, during recovery from sprint races, O_2 is abundant so that the advantage of the rapid availability of chemical energy outweighs the minor disadvantage of interest payment.

Lactic acid threshold

Whether sufficient O_2 is present in muscle cells depends on the speed and the extent of training of the horse. The anaerobic lactic-acid system in unconditioned TBs is probably untaxed until work rates exceed around 600 m/min (22 mph) on the flat (Williamson 1974). The anaerobic threshold of Standardbred trotters is said to be 300–400 m/min (11–15 mph) on the flat (Milne *et al.* 1976), although a striking increase in blood lactate of Standardbred trotters was not observed until speeds exceeded 600 m/min

(Lindholm 1974a), or 684–750 m/min (25–28 mph). Lindholm & Saltin (1974) and Williamson (1974) recorded similar blood responses in these breeds undertaking the same types of exercise.

The threshold of exercise-induced lactate accumulation is taken to be 4 mmol lactate/L in the plasma, and the velocity for a particular horse at which this occurs is termed V_{LA4} (Lindner *et al*. 2009). Anaerobic metabolism provides approximately 30% of immediate energy needs of Standardbreds running at racing speeds. Conditioning by repeated high-intensity exercise (92% $\dot{V}_{O2max}$) produced: increased muscle glycogen concentration before exercise; a higher V_{LA4}; increases in $\dot{V}_{O2max}$ and in speed at which $\dot{V}_{O2max}$ was achieved; speed at 115% of $\dot{V}_{O2max}$; and an increase in the duration at which 115% of $\dot{V}_{O2max}$ was maintained by Standardbreds (Hinchcliff *et al.* 2002). Oxygen consumption was greater and accumulated O_2 deficit was higher than it was before conditioning. O_2 deficit is defined by the O_2 equivalent of the difference between ATP supplied oxidatively from pulmonary $\dot{V}_{O2}$ and the ATP utilized in the exercising muscle. (N.B. TBs, as a breed, have a higher mean $\dot{V}_{O2max}$ and a greater ability to produce lactate than the Standardbred. Moreover, Ronéus *et al*. (1999) concluded that plasma lactate and ammonia concentrations in Standardbred trotters after racing were unrelated to current race performance.) Thus, conditioning caused an increase in both aerobic and anaerobic capacity.

However, there were lower muscle lactate concentrations after high-speed exercise, but similar blood lactate concentrations during exercise at $\dot{V}_{O2max}$ and recovery, so that conditioning enhances the rate of efflux of lactate from muscle, and high-intensity conditioning causes an increase in muscle lactate dehydrogenase (LDH). The lower muscle lactate and a raised intramuscular pH might well have reduced the energetic cost of muscular function. If so, the increase in O_2 deficit could be explained by this in the absence of demonstrated increases in the net energetic contributions of anaerobic glycolysis and/or phosphagen (ATP and PCr system) and O_2 stores (Poole *et al*. 2002). The authors concluded that high-intensity exercise resulted in reduced reliance on anaerobic metabolism at similar absolute work intensities.

This explanation is supported by the observation of other investigators that conditioning results in a reduction in respiratory exchange ratio (rate of CO_2 output/rate of O_2 uptake) during intense exercise (N.B. A decrease in the ratio occurs when there is an increase in the proportion of total energy derived from fat oxidation.) However, in association with a greater work output, the conditioning increased the calculated maximal O_2 deficit in horses. Horses with greater anaerobic capacities that can generate larger O_2 deficits will have a significant race advantage.

Snow & MacKenzie (1977b)showed that cantering for up to 22 km caused no stress on nutrient reserves in the blood. Immediately after the exercise, blood glucose, glycerol and FFAs, as well as pH, rose slightly, which indicated an adequate rate of their mobilization and a sufficient irrigation of muscle cells with O_2. Snow & MacKenzie (1977a) found that, at a maximum work rate of 864 m/min (32 mph) over 3×600 m, with two 5-min intervals between the gallops, anaerobic respiration was necessary in that blood glucose, glycerol, FFA and lactate were all elevated and there was a fall in pH. The increased fund of blood glucose was associated with a considerable rise in adrenocorticosteroid secretion. Training for 10 weeks before the gallops caused a further increase in blood glycerol and lactate with smaller changes in FFA and pH than was the case with the untrained horses. As corticosteroid secretion was even greater after training, training must confer a more efficient mobilization of reserve fat, so that both it and glucose are used to a greater extent by trained horses, resulting in faster times. The lesser fall in blood pH indicates that training also leads to better ventilation and oxygenation of the muscles.

TRAINING METHODS

Muscle type and breed

Skeletal muscles comprise 40–50% of the mass of the average horse and are formed of long multinucleate, cylindrical cells or muscle fibres. Approximately 17–19% of the horse's body comprises protein of which 55% is muscle and 30% is in connective tissue (Martin-Rosset *et al.* 1983). Long-distance riding requires a preponderance of muscle fibres capable of slow contraction but resistant to fatigue, whereas sprinting ability demands a higher proportion of fast-contracting fibres that also happen to be readily fatigued. These fast-contracting fibres are categorized as having myosin with high ATPase activity at pH 9.4 and a high glycolytic activity (type II fibres). They are subdivided into fast-twitch, low oxidative (FT) and fast-twitch, high oxidative (FTH) fibres. Fibres with low myosin-ATPase activity (type I fibres) are present in relatively higher proportions in the skeletal muscles of horses more suited to long-distance riding. These are known as slow-twitch, high oxidative fibres (ST). Standardbred trotters older than four years have 54% FTH, 22% FT and

24% ST fibres in the gluteus medius muscle (Lindholm & Piehl 1974). All muscles have about the same ratio of low oxidative to high oxidative fibres, but a mixture of anaerobic and aerobic training increases the proportion of FTH fibres and decreases the proportion of ST and possibly also of FT fibres, leading to an increase in the ratio of type IIa (hi oxidative) to IIb (lo oxidative) fibres (Lopez Rivero *et al.* 1991, 2002). Differences in muscle fibre type are evident at 158 days of gestation (Lopez Rivero 1997) and Dingboom *et al*. (2002) detected a change with time in fibre type of locomotory muscles that was uninfluenced by exercise in Dutch warmblood foals during their first year postpartum. Further muscle adaptation occurs, such as in oxidative capacity (increase in oxidative enzymes), number of mitochondria and blood supply. The mitochondria in horse muscle are in close association with blood capillaries in the sarcolemma (the plasma membrane which covers every striated muscle fibre) and their volume and density varies in different muscles from 2 to 24% (Hodgson & Rose 1994).

The purpose of training is to modify muscular action and indeed the whole metabolism of the horse so that it functions at maximum efficiency with minimum fatigue at the speed and over the distance at which the sights are set. Couroucé *et al.* (2002) demonstrated in French trotters that V_{LA2} and V_{LA4} and the corresponding heart rates HR_2 and HR_4, and the velocity for a heart rate of 200 beats/min (V_{200}), all improved with age between one and six years. Moreover, V_2, V_4, and V_{200} increased with training. HR at a given blood lactate content can improve with age, owing to an increase in the stroke volume of the heart and an increase in the arteriovenous oxygen content difference, brought about by a reduction in the mixed venous blood O_2 content. A low V_{200} frequently indicates an underlying orthopaedic disease, according to several groups of workers.

During exercise, feed energy is converted into work and Table 6.8 gives the approximate amounts of feed energy required by horses of different weights undertaking work of a variety of intensities. In all but one instance, the work covers a period of 1 h, although it is appreciated that the most strenuous effort could not be sustained continuously for such a period. Nevertheless, the method allows comparisons of different degrees of effort. Walking creates practically no further demand on maintenance requirements, whereas at the other extreme, racing – and more particularly lengthy endurance work – creates an energy demand that exceeds the capacity of the horse for the immediate replenishment of the losses through feeding. Glycogen and fat reserves are therefore heavily drawn upon.

Muscle hypertrophy

Adaptation to hard muscular work entails changes not only in the blood vascular system but also in skeletal muscle. Hypertrophy of muscle occurs during training, the extent of which varies with the breed of horse and type of work. This change is reflected in an increase in apparent nitrogen (N) retention in Quarter Horses (Freeman *et al.* 1988), although losses of N in sweat, which will have partly accounted for the apparent retention, were not measured,. The increased retention of N appeared to continue for a month of rest following the exercise, indicating a higher maintenance requirement for protein in such horses.

Training effects

As the intensity of training increases, the demand for energy release by a particular metabolic pathway rises, so increasing the need for the appropriate enzymes to catalyse the reactions. Sprint racing imposes greater demands on anaerobic metabolism while longer races call predominantly on aerobic processes. The latter implies a more intense use of the TCA cycle in the mitochondria. Training for sustained work therefore brings about an increase in the number of these cellular organelles and their associated enzymes, while training for sprint racing increases glycolytic activity and there may be up to a two-fold increase in the activity of the enzymes aldolase and alanine aminotransferase (ALT). The latter promotes the formation of the amino acid alanine (see 'Ammonia and the alanine vehicle', this chapter) from pyruvate, so lessening the conversion to lactate.

Exercise induces a transient rise in extracellular K^+ concentration, owing to a continuous loss of K^+ from contracting muscles. This loss affects the membrane potential that contributes to muscular fatigue. Re-uptake of K^+ by the cell is achieved with the action of the Na^+,K^+ pump (Na^+,K^+-ATPase). Suwannachot *et al*. (2001) demonstrated that short-sprint training of Dutch warmblood foals for five months increased the Na^+,K^+-ATPase concentration in gluteus medius and semitendinosus muscles. The effect persisted during six months of detraining. These responses reveal ways in which training can influence specific aspects of muscle function and ultimately performance (see also 'Sodium bicarbonate', this chapter, and 'Dehydration and potassium status', Chapter 11).

MUSCLE ENERGY RESERVES AND FEEDING BEFORE EXERCISE

Hormonal effects

A normal meal of grain leads to an elevation in plasma glucose and insulin concentrations. Insulin elevation can last for up to 8 h. It is an anabolic hormone promoting glucose and fat storage, whereas exercise requires the mobilization of energy reserves for combustion. An intense exercise bout 2–6 h after a meal, is associated with decreased NEFA availability, leading to a rapid fall in blood glucose concentration, as the contribution of fat to energy expenditure is decreased. For example, Lawrence *et al*. (1993) fasted Standardbred horses overnight and then gave them no grain (controls) or 1, 2 or 3 kg maize grain 2.5–3 h before a warm-up and intense exercise over 1600 m at a heart rate of 206 bpm. The controls maintained steady plasma glucose concentrations, whereas plasma glucose declined in those given any grain. Plasma NEFA were initially higher in the controls, but declined in these horses during the intense exercise. In the grain-fed horses plasma NEFA remained steady during exercise, but the concentration was always less than in the controls. Similarly Pagan (2000) showed that grain given 3 h before exercise (treadmill, total 60 min) decreased availability of NEFA and decreased blood glucose during exercise as a consequence of stimulation to insulin secretion, indicating decreased lipid oxidation during exercise (Lawrence *et al.* 1993, 1995; Stull & Rodiek 1995; Jose-Cunilleras *et al*. 2002), as insulin is a potent inhibitor of lipolysis and lipid oxidation. Increased concentrations of circulating NEFA and TAG before low intensity exercise (15% of $\dot{V}O_{2max}$ for 1 h) yield a lower respiratory exchange ratio (R) and subsequently raised plasma glucose (Orme *et al*. 1994). The feeding of grain during the hours immediately before exercise is ill-advised. Elevated plasma NEFA before low-intensity exercise spares plasma glucose during exercise; but the effect of a meal on repeated bouts of intense exercise during a day probably depends on the size of the meal, timing, exercise intensity, etc. Intense exercise stimulates epinephrine and norepinephrine (adrenaline and noradrenaline) secretion which overrides the effect of insulin on glucose and fat metabolism, so that fat mobilization would not be impaired during further bouts of exercise.

Blood distribution effects

Exercise in ponies at 7.8 m/s on a 6.3° incline at 75% HR_{max} for 30 min, 1.4 h after feeding, led to higher GI tract and skeletal muscle blood flows compared with those in fasted ponies (Duren *et al.* 1992). There was an increase in heart rate, cardiac output, stroke volume and arterial blood pressure, whereas accommodation might not have been possible with more intense work. This work is therefore normally delayed for 5–8 h after feeding, although the optimum time interval is influenced by the dietary proportion subject to fermentation and type of activity. (See also 'Exercise warm-up' and 'The vascular and respiratory systems', this chapter, and Figure 9.7)

Glycogen use and muscle type

The accelerated use of glucose is accommodated by a stimulation of glycogen deposition in the muscles of adequately fed and trained horses. This training can increase the glycogen capacity of TB muscles by a third. Blood glucose cannot, however, be maintained indefinitely during work, and endurance rides of up to 150 km result in a gradual decline and an exhaustion of muscle glycogen. In one 80 km (50 mile) ride, blood glucose on average fell by 40%, whereas NEFA rose eightfold (Hall *et al.* 1982). In trained horses, blood glucose concentration is not affected by endurance rides of 50 km (31 miles), but is decreased 23% during a ride of 100 km (62 miles) (Essén-Gustavsson *et al.* 1984). Over intense and long duration exercise type IIB can suffer complete glycogen depletion, but total glycogen reserves never fall below 400–500 mmol glycogen/kg freeze dried muscle (Essén-Gustavsson *et al.* 1991). Fatigue sets in before lipids are depleted as glycogen depletion in liver and muscle reduces pyruvate production, necessary for NEFA utilisation in the TCA cycle. For this reason, it is necessary to maintain reasonable concentrations of circulating fatty acids and glucose at endurance check points. The intravenous infusion of glucose at the rate of 6 g/kg BW (3 kg/500 kg) during the first several hours following glycogen-depletion extends the time to fatigue (Farris *et al.* 1995,1998; Lacombe *et al.* 2001). Lower rates (2 g/kg BW) given by nasogastric tube did not elevate glycogen resynthesis above controls (Nout *et al.* 2003).

The repletion of muscle glycogen and rehydration of the horse following exercise is important to subsequent performance (Snow *et al.* 1987d; Lacombe *et al.* 2004); but a starch-rich meal given 15 min. and 4 h after glycogen depleting exercise only minimally enhanced muscle glycogen replenishment after 8 h (Jose-Cunilleras *et al.* 2006). Subsequently the same group showed that a high starch diet given at 8 h intervals for 72 h (6.5 kg/horse daily, 77% cereal plus 21% hay) returned muscle glycogen concentration to baseline concentrations, whereas a diet of 66% hay plus 33% cracked maize did not.

Post-exercise glycogen repletion requires action in skeletal muscle of glucose transporter (GLUT) proteins. After a single bout of moderate exercise, GLUT-4 protein

was not increased (Nout *et al.* 2003), but after a 3-day regimen of strenuous, glycogen-depleting exercise GLUT-4 protein and/or GLUT-4 mRNA were increased; the mechanism controlling repletion is not yet understood (Lacombe *et al.* 2003; Jose-Cunilleras *et al.* 2005).

The net loss of muscle glycogen is extremely small on light work. Lindholm (1974) observed that Standardbred trotters lost 0.3 mmol glycogen/kg per min when trotting at 300 m/min (11 mph), whereas trotting at a maximum rate (750 m/min or 28 mph) led to a loss of 14 mmol glycogen/kg per min. Three aggregate minutes of maximal trotting were proved to cause a 48% decrease in muscle glycogen, but the decrement was not equally distributed among the fibre types. Maximal work causes a striking depletion of ST fibres in addition to a loss from the other two types. Slow trotting leads to a gradual depletion of ST fibres after which the FTH fibres become active and depleted. Thus, there seems to be a preferential fibre recruitment with increasing speed and duration.

Glycogen 'loading'

The stimulation of glycogen accretion has been considered from both safety and efficacy aspects. Starchy diets and regular exercise can increase muscle glycogen, but this increase is not maintained if the starch content of the diet is subsequently reduced. Harris & Hultman (1992a) found no difference between diets insofar as loading is concerned, although loading is probably more effective during rest on a high carbohydrate rather than a high-fat diet following intense exercise that depletes type II muscle fibres (fast twitch) of glycogen (Pagan *et al.* 1987a). By comparison with intense exercise, aerobic work is ineffective and glycogen loading may cause poorer performance (Topliff *et al.* 1985, 1987; Pagan *et al.* 1987a) and an increased risk of exertional rhabdomyolysis syndrome (ERS, or 'tying-up'), according to a widely held view. This condition might not, however, be associated with either lactic acidosis, or excessive dietary carbohydrate. Hodgson (1993) speculates that horses predisposed to tying-up exhibit a temporary failure in the control of intracellular [Ca^{2+}] (see ERS, Chapter 11).

A glycogen storage disorder, polysaccharide storage myopathy (PSSM) is recognized in Quarter Horses, but also in other breeds, e.g. Anglo-Arab show jumpers and Andalusian dressage horses with back pain (Quiroz-Rothe *et al*. 2002, see also Chapter 11).

Tissue fat as an energy source

Horses described as 'moderately fat' require more energy for maintenance than do those in 'moderate condition'. The reserves of fat in the latter state are adequate as a source of energy for exercise, indicating that a relatively lean condition is satisfactory.

Exercise warm-up

Horses have a high aerobic capacity and rapid kinetics of gas exchange compared with many other mammalian species. The maximum rate of O_2 uptake ($\dot{V}O_{2max}$ mL/min per kg) in racehorses is double that of human athletes and $\dot{V}O_2$ increases very rapidly at the onset of high-intensity exercise. The kinetics of gas exchange are affected by a warm-up prior to high-intensity exercise which results in horses reaching steady state $\dot{V}O_2$ faster. Tyler *et al.* (1996) found that a warm-up for 5 min at 50% $\dot{V}O_{2max}$ increased the aerobic contribution to total energy needs from 72.4 to 79.3% when they were run to fatigue (1–2 min) at 115% of $\dot{V}O_{2max}$. The maximal accumulated O_2 deficit was lower in the warmed-up horse, i.e. 34.7 *vs*. 47.3 mL O_2 eq/kg BW, evidence confirmed by McCutcheon *et al.* (1999) with a decreased glycogen utilization and longer running time to fatigue, emphasizing the value of a warm-up, allowing greater use of aerobic energy sources and providing the time necessary for the redistribution of the blood with an increase in circulation rate. Recovery from intensive exercise is promoted by post-race aerobic trotting, compared with no exercise, as clearance of lactic acid is accelerated.

Bone remodelling and training

The commencement of training causes a remodelling of the long bones to accommodate the stresses on the skeleton. This process involves the mobilization of bone salts and their redeposition. There is a risk of microfractures occurring if training is intensified too rapidly. Ca balance data indicate that bone density is low during the first two months of training and that Ca balance and bone density are still increasing after three to four months. When stalled four-month-old Quarter Horse foals were sprinted over only 82 m/day, five days a week for two months, there was an increase in the radiographic density of the third metacarpal (Hiney *et al*. 2002). (See also 'Acid–base balance', this chapter, and 'Ca and P in bone', Chapter 3.)

Causes of withdrawal from training

Causes of illness or fatal injury were analysed in 496 racehorses in California between 1990 and 1992 (Johnson *et al.* 1994). Musculoskeletal injuries accounted for about 80% of TB and Quarter Horse submissions, among which forelimb fractures were prominent. Poor mineral nutrition is likely to have played some role in this statistic.

THE ENDOCRINE SYSTEM

Examination of Figure 9.2 reveals that the changing demands of the horse for energy are monitored by a number of endocrine secretions. Where rapid changes are necessary, the signal for secretion by the appropriate glands is provided by the involuntary action of the autonomic nervous system reacting to environmental stimuli. This in turn brings about other essential changes in cardiac muscle and the smooth muscles of arteries, intestines, etc. Endocrine secretions mediate some of their effects by switching on and off enzymes that regulate energy metabolism.

Insulin

The insulin molecule attaches to receptors on cell membranes to stimulate cellular uptake of glucose and TAG 'clearance', so that their blood concentrations are decreased after a meal. Lipolysis in adipocytes is inhibited by insulin action on hormone-sensitive lipase. Horses accustomed to a high starch diet possess a high insulin sensitivity and therefore are more inclined to hypoglycaemic shock when subjected to a fast than horses given hay diets and accustomed to deriving blood glucose from other sources. Insulin sensitivity can be determined by measuring glucose and insulin responses to a given dose of starch or glucose. Low sensitivity and poor tolerance increase the areas under the plasma response curve of insulin and of glucose. Large doses of glucose in insulin resistant, diabetic or fasted animals cause glycosuria.

In TBs, blood glucose normally peaks about 2 h from the start of feeding. The peak value is 6.5–8.5 mmol/L (117–153 mg/dL) at 1.5–3 h. There follows a gradual decline to a post-absorptive level of about 4.6–4.8 mmol/L (83–86 mg/dL). Ponies on roughage diets maintain normal levels 1.4 mmol/L (25 mg/dL) lower than this. Blood insulin activity tends to fall slightly during work because of the catabolism of glucose, but some insulin is still probably required to ensure that glucose is available to the working muscle cell.

Glucagon

In order to sustain concentrations of blood glucose within normal limits, the influence of insulin is counteracted by that of glucagon. Whereas the former promotes uptake of glucose by all cells of the body (except brain cells), glucagon appears to focus its effects primarily on the liver and adipose tissue. It achieves an increase in blood glucose by stimulating those enzymes that cause a breakdown of liver glycogen (Figure 9.2) and encouraging gluconeogenesis. In this it works in concert with other hormones discussed below, a particularly important task in roughage-fed animals. These other hormones are the glucocorticosteroids, produced by the adrenal cortex, and epinephrine and norepinephrine secreted by the adrenal medulla.

Adrenal hormones (catecholamines and glucocorticoids)

The rapid initiation of intense work necessitates an immediate response of energy mobilization. This is brought about by sympathetic nervous activity, which not only causes splenic release of red cells but also stimulates the adrenal medulla to secrete epinephrine and norepinephrine (adrenaline and noradrenaline). The extent of this reaction depends on the intensity of the work – that is, the faster the horse is running the greater the secretion. The medullary hormones affect several tissues, increasing the mobilization of fatty acids from adipose tissue and stimulating the production of glucose with a rapid rise in the blood concentration by the breakdown of liver glycogen and by amino acid metabolism (Figure 9.2).

The glucocorticoids secreted by the adrenal cortex are somewhat slower in responding to work and their secretion depends on a hormonal signal from the anterior pituitary. Moreover, they stimulate a rise in blood glucose and the accumulation of liver glycogen by promoting gluconeogenesis through the inhibition of protein synthesis. They also provoke the breakdown of depot fats to fatty acids and glycerol. Synthetic analogues of these secretions, when given repeatedly, have a comparable effect and cause muscular wasting. They also cause bone problems through an inhibition of Ca absorption from the gut. The stimulation from glucocorticoids of amino acid mobilization is expressed by excitation of transferase enzymes, and raising the serum (Codazza *et al.* 1974; Sommer & Felbinger 1983; Essén-Gustavsson *et al.* 1984) and muscle (Guy & Snow 1977a) activities of ALT and AST (see Figure 9.2), observed after exercise.

In an analogous fashion to the response of glucagon, a reduction in blood glucose triggers the secretion of the glucocorticoids and their circulating level increases with agitation, trauma and psychological stress. Training leads to a greater adrenocorticoid response under such stress. This applies to sprint, endurance and other training so that normal concentrations of blood glucose are maintained more effectively in all circumstances. In contrast to the response of medullary hormones, amounts of plasma cortisol do not seem to be correlated with the intensity and speed of work. (N.B. Acute stress in the horse leads to an increase in circulating total cortisol, whereas chronic stress generally seems to decrease total plasma cortisol

concentration. This might be due to the production of ACTH-release inhibiting factor, which could be an endogenous opioid.)

THE VASCULAR AND RESPIRATORY SYSTEMS

The blood volume of horses is about 9.7% of body weight so that a horse weighing 560 kg would contain around 51.2 L (SG 1.06). Blood volume is important for both O_2 and heat transport so that a large plasma volume is accompanied by a considerable skin blood flow. This volume can increase by 30% over two weeks' training (Erickson *et al.* 1987). Other adaptations that of increasing blood supply to skeletal muscles. For example, resting glomerular filtration rate in TBs averages 3.3 mL/kg per min but, unlike the response in man, the value decreases by over 40% during exercise and the decrease is considerable even during a walk (Gleadhill *et al.* 2000). This decrease appears to be an adaptation to allow greater blood perfusion of muscle and skin.

The volume of blood discharged per beat (stroke volume) in a 560 kg horse at rest would be 1.2 L and as blood volume is proportional to body weight, cardiac output per ventricle ranges between 56 and 75 mL/kg BW per min at rest. The need for such a flexible system can be appreciated when it is realized that O_2 consumption by skeletal muscle can increase 100-fold in strenuous exercise.

The blood of the horse is a fluid containing, by volume, 40–45% red cells (erythrocytes), 1% white cells (leucocytes) plus platelets, and 54% plasma. The red cells have, as a major function, the transport of O_2 from the lungs to the muscles and other tissues (Snow *et al.* 1983b). To accommodate an increased O_2 demand, a reserve of red cells is held in the spleen. This splenic reserve is very large in TBs, so that they can increase the numbers of red cells in circulation by 30–60%. The adult TB can mobilize 6–12 L red-blood-cell-rich blood into the central circulation, and increase cardiac output increases from 30 L/min to amounts exceeding 300 L/min at the onset of maximal exercise.

Thus, in heavy work, the O_2-carrying capacity of the blood increases from rest by as much as 8.8 volumes %. Between rest and galloping at 700 m/min (26 mph), the O_2-carrying capacity changed from 15.9 to 21.4 volumes % in one study and from 16.35 to 25.19 volumes % (a 54% increase) in another study (Milne 1974) using TB, Quarter Horse, Arabian and Standardbred horses. The extent of change is increased by training and the effect of a change in capacity is augmented by a redistribution of the blood supply to skeletal and cardiac muscle and skin. The degree of splenic release tends to be proportional to speed; in one set of measurements the PCV rose 32% at 350 m/min (13 mph) and 55% at 700 m/min (26 mph) (Williamson 1974; for other observations see Figures 9.3 and 9.4). To assess the red-cell count and haemoglobin content of blood as affected by dietary inadequacies and other factors, the most consistent results are obtained after splenic release.

Blood and waste disposal

The supply of blood to muscles, which is increased during work, is the principal vehicle in which the waste products of energy metabolism, water, carbon dioxide and heat are removed from muscle cells. In the absence of their

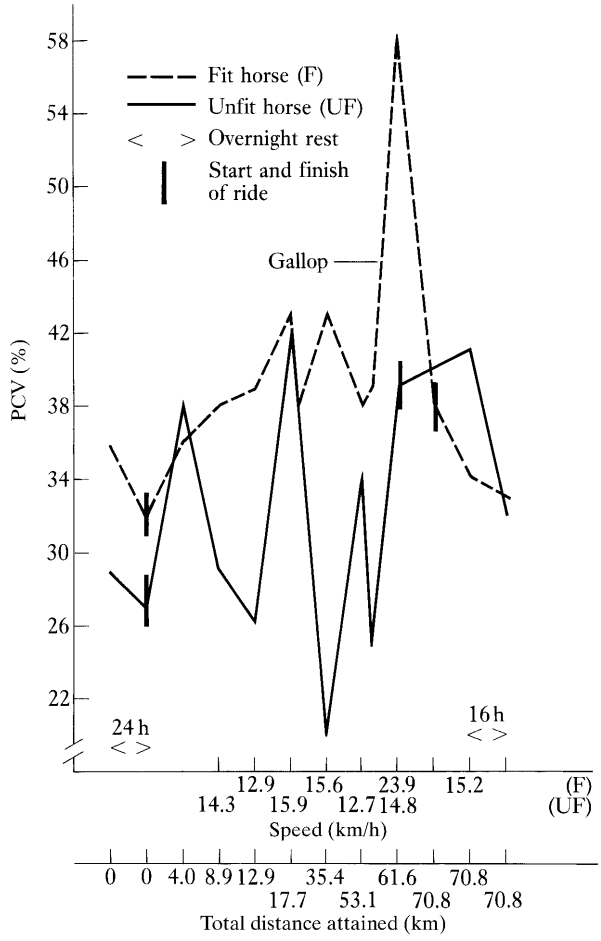


Figure 9.3 PCV of the blood during an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

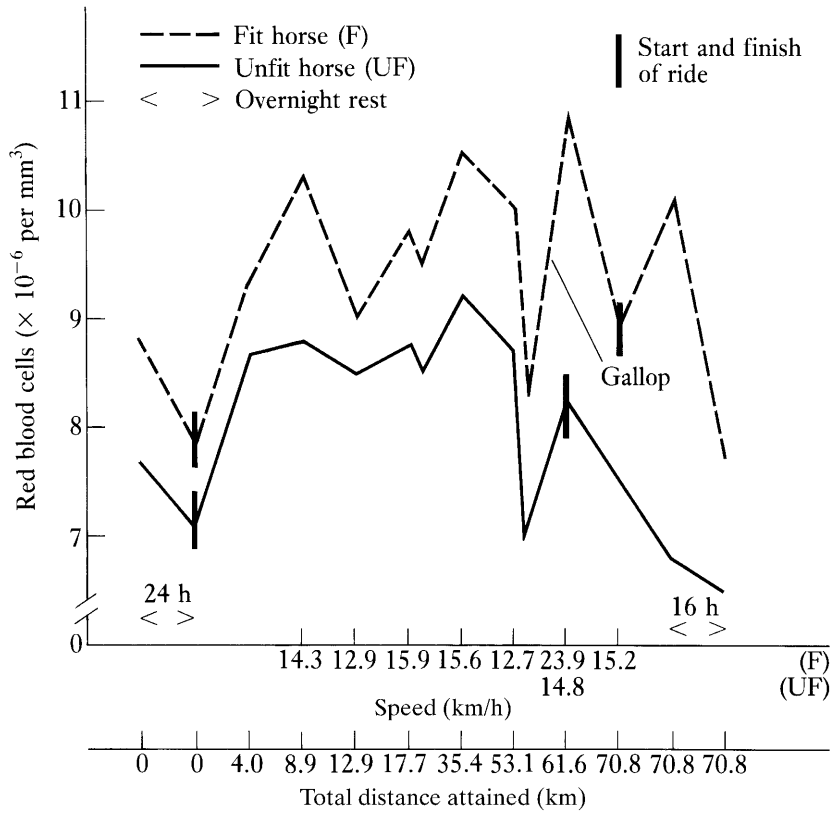


Figure 9.4 Changes in erthyrocyte count during an endurance ride in a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

efficient disposal, pathological cellular changes would result. To facilitate this disposal, the capillaries of skeletal and cardiac muscle become dilated during increased work, while those in the visceral region contract. This in turn diminishes the digestion and absorption of nutrients and gives credibility to the rule that horses should not be fed before hard work (see 'Muscle energy reserves and feeding before exercise', this chapter).

Body temperature

At high work rates, heat production is 40–60 times basal levels, and body temperature can rise appreciably (Figure 9.5). The mean muscle temperature of five Standardbred trotters increased from a normal of 37°C to 41.5°C during a race of 2100 m at a mean rate of 708 m/min or about 26 mph (Lindholm & Saltin 1974). Carlson (1983b) calculated that if a horse could work at a moderate intensity for one hour with an O_2 consumption of 30–40 L/min, the total waste heat would amount to 38 MJ. The principal mechanism for exhausting this waste heat from the body, when atmospheric humidity is not excessive, is the evaporation of sweat and of moisture from the surface of the lungs, with the greater proportion being dissipated through the skin, accompanied by electrolyte losses, accelerating the onset of fatigue. Dilatation of subcutaneous blood vessels diverts blood from skeletal muscle, contributing to a decrease in work capacity, at a cost influenced by the depth of subcutaneous fat (Webb *et al.* 1987b).

If circulatory adjustments fail to maintain heat balance, ventilation rate is increased (Figure 9.6), which induces respiratory alkalosis. Training is important, as the rise in core temperature during exercise is proportional not to exercise intensity, but to the percentage $\dot{V}O_{2max}$, which is increased by training, so retarding a temperature rise. Long low-intensity exercise on a treadmill at 30°C and 80% RH over 15 days gave horses an advantage in a three-day event at 30°C, 80% RH (Marlin *et al.* 1999). The advantage was assessed as a lower body temperature, lower metabolic

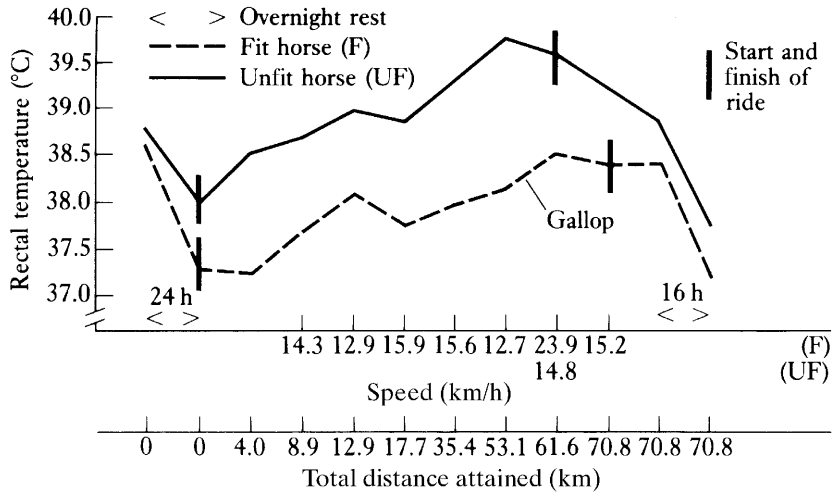


Figure 9.5 Effect on body temperature of an endurance ride by a fit Arab stallion and a less-fit pony gelding in cool weather (Frape *et al.* 1979).

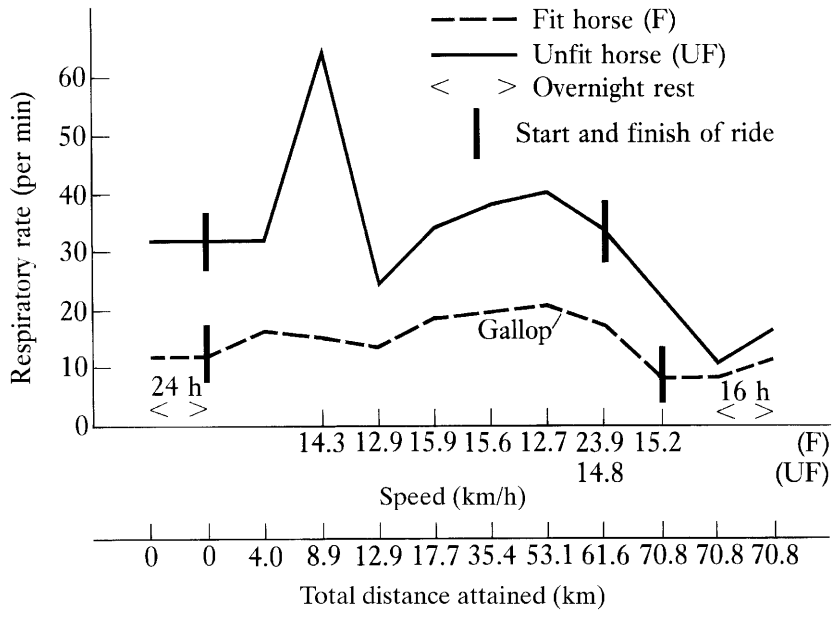


Figure 9.6 Effect on respiration rate of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

Feed consumption leads to:

- ↑ coeliac blood flow
- ↓ skeletal muscular blood flow, or
- ↑ cardiac work

Exercise leads to:

- ↑ skin blood flow
- ↑ water–electrolyte loss
- ↑ respiratory and heart rates
- ↑ pulmonary blood flow

Figure 9.7 Heat flow and blood redistribution as a consequence of eating and exercise (Frape 1994) (↑, increase; ↓, decrease).

heat production and decreased total fluid loss with sweating initiated at lower body temperatures. Rose *et al.* (1980a) recorded changes in metabolic parameters in horses during three-day event competition.

Following feed ingestion the amount of blood distributed to the GI tract increases, competing with the redistribution necessitated by a rise in heat production. Accommodation is achieved by augmenting cardiac output, redistributing regional blood flow, or by combining these mechanisms (Figure 9.7).

Blood as a buffering system

Red blood cells contain haemoglobin, which not only carries O_2 but also acts as a buffer to the lactic acid produced in contracting muscles. This is probably why the pH of blood falls only during galloping, whereas the haemoglobin content rises appreciably during only moderate exercise. As haemoglobin is a major buffer, blood pH is changed very little for a considerable change in bicarbonate content. However, in performing this function, haemoglobin carries less O_2. In extreme cases of acidosis the horse becomes cyanotic, or O_2-starved.

Blood glucose concentration

Blood glucose concentration is the expression of a dynamic balance of glycogen breakdown and glycogen synthesis and the production of glucose from other sources – amino acids, lactic acid and propionate (gluconeogenesis). The resting level is somewhat higher in horses in intensive training for sprint races. This state is brought about by the stimulation of the two systems leading to the formation of glucose and by increasing the efficiency of fatty-acid utilization, sparing glucose. Thus, blood glucose fluctuates throughout the day, rising in TBs from post-absorptive values of 4.7 mmol/L to a peak of approximately 6.5–7.5 mmol/L, 2–3 h after a large feed of oats, following moderate work (those fed after strenuous exercise may not show a plasma glucose response, Frape 1989). In contrast, blood glucose concentrations reached values of between 4.7 and 6.4 mmol/L in ponies after receiving a pelleted meal to appetite, whereas after 3 h of fast, the levels fell to between 2.8 and 5 mmol/L (Ralston *et al.* 1979). Plasma pyruvate also tends to increase with glucose, whereas fatty acids and glycerol are in lower concentrations.

The fluctuation between peak and resting levels of glucose varies with the type of diet, in that foods containing more grain and less roughage lead to higher peaks and lower troughs. Horses subjected to a rougher diet develop a greater faculty for gluconeogenesis and can therefore resist a depression in blood glucose more readily during a fast, but are unable to meet the demands of an excessive rate of exertion. Furthermore, breeds such as TBs, with higher insulin sensitivity, experience a greater fluctuation in blood glucose than do ponies given the same diet at similar times of the day.

Haematocrit, blood viscosity, blood volume and exercise-induced pulmonary haemorrhage (EIPH or 'bleeders')

Slight pulmonary bleeding seems to be a normal consequence of strenuous work and does not reflect any dietary abnormality, but simply reflects the physiological stress. Pulmonary capillaries necessarily have very thin walls to allow rapid exchange of respiratory gases across them and stress failure is greatly increased at high lung volumes. Capillary walls are not strong enough to withstand the stress of the high capillary pressures accompanying extremely high cardiac outputs during strenuous exercise. Low environmental temperatures (range of observations −10 to +17°C) can increase the risk (Lapointe *et al.* 1994) and the evidence of Derksen *et al.* (2009) indicated that repeated bouts of strenuous exercise lead to vein wall remodelling and pulmonary capillary hypertension and failure, with haemosiderin accumulation and lung fibrosis.

In order for blood to travel freely through the capillary bed of muscles, it is essential that it retains its fluidity, and where the PCV exceeds 55% there is an exponential increase in blood viscosity. Providing horses with additional red blood cells before races can therefore be

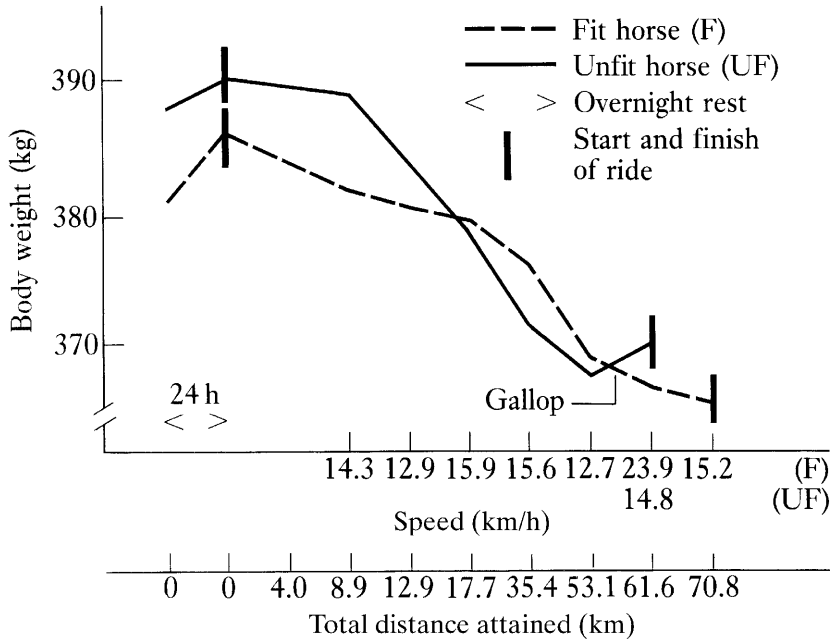


Figure 9.8 Effect on body weight of an endurance ride by a fit Arab stallion and a less-fit pony gelding in cool weather (Frape *et al.* 1979).

misguided. Optimization of blood flow to working muscles is dependent upon blood pressure, diameter and length of vessel, as well as blood viscosity. The high PCV of horses during exercise could be fatal in human athletes, but owing to blood's unique rheological properties that allow for optimal flow despite an elevated PCV during equine exertion, such a PCV is not fatal (McKeever 1998). However, changes in blood viscosity and other rheological factors contribute to the rise in pulmonary artery pressure and the onset of EIPH (Weiss & Smith 1998).

Performance is impaired by the haemodynamic response to exercise in Standardbred trotters with red cell **hypervolaemia** (RCHV) (Funkquist *et al.* 2000). Exercise-induced pulmonary haemorrhage could be caused by the increase in red cell volume. This results in the high PCV and high total blood volume of RCHV horses that contribute to their high blood pressure in the pulmonary and systemic circulation during exercise. The importance of a low viscosity is recognized in horses subject to dehydration in hot climates and during long-distance rides (Figures 9.8 and 9.9). For this reason it might not be by chance that the PCV of Arab horses (and that of racing dromedary camels) (Figure 9.3) is lower than that of other hot-blooded horses and that Arab horses are highly adapted to both hot climates and extended work. A voluminous splenic pool is therefore unnecessary for some purposes. On the other hand, there is evidence that a relatively greater increase in PCV and circulating red cell volume observed in a hot humid climate cf. a cooler one during racing confers a thermoregulatory advantage (Hargreaves *et al.* 1999). The critical feature is probably blood viscosity.

Plasma albumin, a reserve protein and a major contributor to blood viscosity, displays a decreased concentration during training for reasons that are understandable in this context. The dilutions achieved still provide adequate osmotic pressure and do not necessarily suggest a dietary protein deficiency.

Pulse, respiratory rate and fitness

Heart rate is linearly related to speed of the horse and varies between the approximate limits of 30 and 240 contractions/min. After a gallop at 700–800 m/min (26–30 mph) the rate is as high as 240 bpm with an output from each ventricle of 3–4 L/s. Heart rate, particularly after work, is a good indicator of fitness. In the pre-ride checks of endurance horses it is agreed that pulse rate should fall within 36–42 bpm (Figure 9.10) and respiration rate 8–14/min (Figure 9.6). Both are higher in unconditioned horses.

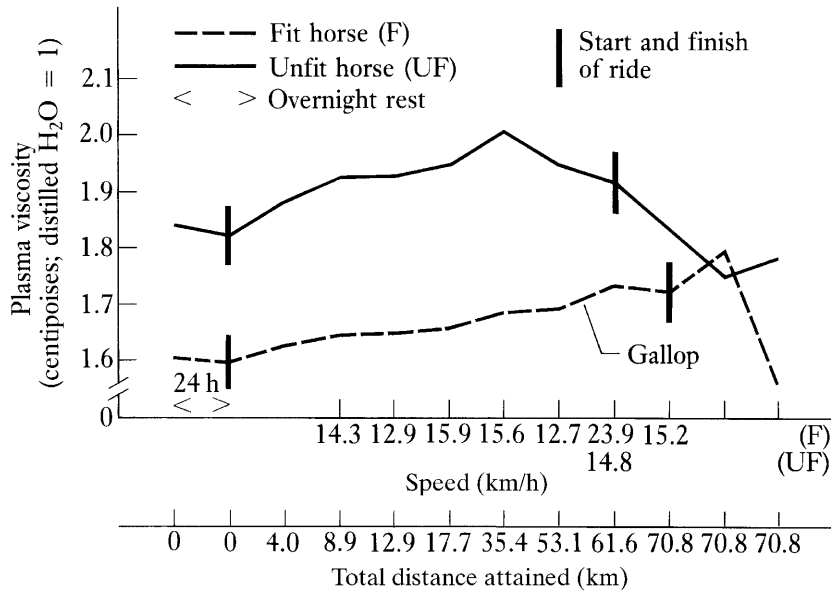


Figure 9.9 Effect on blood plasma viscosity of breed and of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

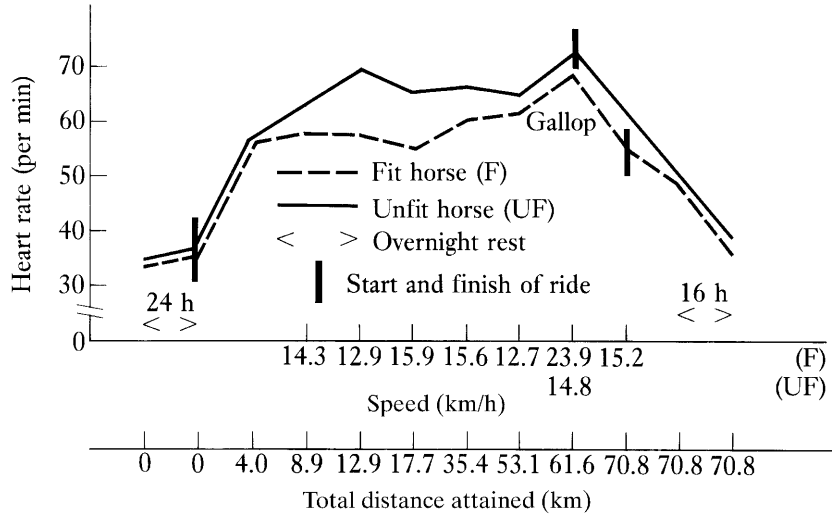


Figure 9.10 Effect on heart rate of an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979). (NB: raised heart rate of unfit horse at 53–61 km might have resulted from anxiety when partner galloped off.)

After an endurance ride and 20 minute rest the pulse rate should have fallen to less than 55 bpm and the respiration rate to 20–25/min. In exhausted horses, the rate of both these is greater (tachycardia and hyperpnoea) and the occurrence of muscular spasms more likely.

The ratio of pulse to respiratory rate should fall within the limits of 2:1 to 5:1. During heavy exertion and heat stress the pulse and respiration rates have been known to rise to 85 and 170, respectively, a ratio of 1:2, that is, an inversion of the pulse:respiration rate ratio. Poorer horses and those suffering from adrenal exhaustion tend to exhibit lower heart:respiratory rates both before and after exercise. After a 20 minute rest during an endurance ride, horses exhibiting heart rates exceeding 70 bpm or heart:respiratory rate ratios of less than 2:1 should be eliminated.

Hyperventilation simply reflects a shortage of oxygen and normal respiratory acidosis (not commonly seen in endurance horses), or it can indicate an increase in body temperature (easily measured per rectum) brought on by hot weather or inadequate training, or both (Figure 9.5). Alkalaemia normally follows a raised body temperature (see 'body temperature', this chapter).

RESULTS OF EXERCISE

Sweat and dehydration

A horse working at a moderate intensity for 1 h with an O_2 consumption of 30–40 L/min would need to dissipate 38 MJ waste heat (Carlson 1983b). The disposal of this quantity by evaporative processes alone would entail the loss of just over 15 L water. Although this is an oversimplification, it is a reasonable estimate of events in high environmental temperatures and low relative humidities. Sweat losses are quite modest in horses racing for distances of up to 3 km, but it seems that body-water losses in sweat (and urine), and from the lungs, during prolonged exercise can approach 10–12 L/h. Of the total fluid losses, 20–30% is lost by expiration and the remainder is largely sweat. Hence, a 500-kg horse can lose 30 L water in sweat, with a total loss of as much as 40 L during extended exercise.

Typically, body weight falls by 5–9%, principally from these evaporative losses (Figure 9.8), but the extent of loss depends on the level of fitness and the availability of water and electrolytes during the exercise. The evidence in Figure 9.8 and Table 9.2 clearly indicates a greater rate of body-weight loss and of sweat production (containing the fixed ions of Na, K, Cl, Ca, P and Mg) by an unfit horse than by a fit one, although in both horses the molar sum of cations approximately equals that of the anions in the sweat.

Water losses are more aptly compared with total body water, which, in a horse of 450–500 kg body weight, would amount to 8–14% of the total of about 300 L in extended exercise. Of this 300 L, about 200 L is in intracellular fluid (ICF) and 100 L is in extracellular fluid (ECF), made up of the water in blood plasma, interstitial fluid, lymph and contents of the GI tract.

Sweat, work, electrolytes and their losses

Na, K and Cl

The 100 L of ECF contain 14 000–15 000 mmol readily exchangeable Na, representing nearly all the exchangeable Na in the body, the total Na content of which is about 1 kg. Of this total, 40% is located in the skeleton, and during extended Na depletion this depot appears to be partially mobilized. The 200 L of ICF contain 20 000–30 000 mmol readily exchangeable K, i.e. most of the body's reserve. The bulk of the exchangeable Cl is present in the ECF, where it is the major fixed anion, but its concentration is substantially lower than that of Na, probably of the order of 10 000–12 000 mmol/100 L.

Ca and Mg

Horse sweat is hypertonic with respect to plasma, containing 2–6 mmol/L Ca and 1–6 mmol/L Mg, indicating a loss of 2–7 g Ca and 0.6–4.0 g Mg in 30 L. The sweat to plasma ratio ranges between 1:1 and 8:1 for Mg and 1:1 and 2:1 for Ca. However, Mg is mainly an intracellular cation and less than 1% is present in the ECF, so serum, or plasma, Mg is not a reliable indicator of body Mg status. Moreover, catecholamines are elevated during exercise. Raised blood adrenaline causes a net efflux of Mg from the intracellular space. Blood pH also influences the values of ionized Ca and Mg in plasma, as the extent of binding increases with increasing pH. Approximately 70–75% of plasma Mg is ultrafiltrable, of which the main portion (65% of total) is ionized and the remainder (8% of total) complexed with anions. Weiss (2000, personal communication) determined a decrease in total plasma concentrations of Ca and Mg following a trot. This decrease reflected the sweat loss, as urinary Mg is not increased in exercising horses (Meyer *et al*. 1991). Body stores of Ca and Mg are generally sufficient to replace sweat losses, but serum concentrations of Ca and Mg also reflect dietary supply and a liberal supply can prevent critical adverse clinical consequences of exercise (see also 'Hypo- and hypercalcaemia and stress tetany', Chapter 11).

Table 9.2 Electrolytes (mmol/L)[1] in evaporated sweat of horses participating in an endurance ride[2] and other data obtained from exercising horses.

Horse	Cl	Na	K	Ca	P	Mg	pH of venous blood
Fit at mid-ride[2]	910	710	215	—	—	—	—
Fit at finish[2]	1180	880	270	—	—	—	7.29
Unfit at finish[2]	3060	2120	780	—	—	—	7.36
Harris[3]	155	135	41	3	<0.3	2	—
Mean of other data[4]	231	173	49	—	—	—	—
Hoyt *et al*. 1995[1]	26	19	36	0.051	<0.083	—	—

[1]Hoyt *et al*. 1995a; data given as mmol/MJ DE consumed by miniature horses for work, above maintenance.
[2]Frape *et al*. 1979.
[3]Harris (1996), personal communication.
[4]Meyer *et al*. 1978; Carlson & Ocen 1979; Rose *et al*. 1980c; Snow *et al*. 1982.

Sweat losses during work

The primary route of fixed-ion loss by working horses is sweat produced to prevent an excessive rise in body temperature, so that dehydration through sweating entails a loss of both water and electrolytes with a contraction of the volume of body fluid. Hoyt *et al.* (1995a) found relatively greater losses of K than of Na and concluded that dietary needs during exercise were increased over the basal dietary supply by three-, seven- and six-fold, respectively for Na, K and Cl. It would be important to establish the validity of, or reason for, the differences in relative loss of Na and K.

Changes in the composition of the blood plasma depend on the proportions lost of each of these constituents and of water and on the movement of ions into and out of ICF. Extended exercise, with minimal water consumption, leading to dehydration, will normally precipitate a substantial reduction in the concentration of plasma Cl. Little change is frequently detected in the amounts of K and Na, although hypokalaemia is not rare. The explanation for the hypochloraemia is revealed by a comparison of the Na and Cl contents of sweat (Table 9.2) with their respective concentrations in blood serum (Table 3.1), demonstrating that a much greater proportion of Cl than of Na in body fluid is lost. Extended work, in hot dry weather, by a 450–500 kg horse expressing the above plasma changes yields losses of as much as 35 L of water, 3500 mmol Na, 1500 mmol K and 4200 mmol Cl (equivalent to 80 g Na, 59 g K and 149 g Cl). The loss of Na, for example, represents over 200 g of sodium chloride, that is much more than a horse would eat in a day. These values are at variance with the relatively higher values for K reported by Hoyt *et al.* (1995a), which are equivalent to 0.43 g Na, 1.41 g K, 0.93 g Cl, 2.03 mg Ca and 2.56 mg P/MJ DE consumed for work above maintenance.

The total concentration of electrolytes in sweat is higher than that in blood plasma, so a decline in the plasma concentration, despite considerable dehydration, is readily understandable. Most studies have revealed a decline in plasma electrolytes during endurance rides taking place in hot weather. For example, Carlson *et al.* (1976) reported reductions of 4.2 mmol Na/L, 0.9 mmol K/L, 10.3 mmol Cl/L and no change in Ca. On the other hand, the changes can be variable depending on such factors as relative losses of water to electrolytes, plasma pH changes and time of collection after the ride. In horses losing a considerable amount of water, Rose *et al.* (1977) detected increases of 6 mmol Na/L and 0.3 mmol K/L plasma, no change in Ca and a decrease of 6.8 mmol Cl/L plasma.

Glycogen stores are depleted after extended exercise. The administration during recovery of glucose solutions by nasogastric tube does not stimulate glycogen resynthesis, whereas a NaCl solution of 4.5 g/L or 9 g/L does (Vervuert *et al*. 1999). Moreover, NaCl was more effective in restoring plasma volume and normochloraemia (Coenen *et al*. 1999). Nevertheless, the rise in plasma Na^+ increases blood pressure and secretion of natriuretic peptide. During a second exercise test, 2 h following the recovery period there was a decrease in blood glucose and higher blood lactate in horses that had been treated with glucose, whereas blood glucose remained unchanged and there was a pronounced FFA response in those that had been treated with electrolytes (Vervuert *et al*. 1999). Electrolyte supplementation is common during endurance competition, although oral hypertonic electrolyte administration by the normal procedure is associated with an exacerbation of

Table 9.3 Composition (mmol/L) of various electrolyte solutions for i.v. use[1](Rose 1981).

	Na	K	Ca	Mg	Cl	Glucose	Bicarbonate	Lactate	Acetate	Gluconate	Propionate
0.9% NaCl	154	—	—	—	154	—	—	—	—	—	—
5% Dextrose	—	—	—	—	—	278	—	—	—	—	—
Hartmann's solution	131	5	2	—	112	—	—	28	—	—	—
Ringer's lactate	130	4	3	—	109	—	—	28	—	—	—
Normosol R[2]	140	5		1.5	98	—	—	—	27	23	—
Dilusol R[3]	140	5		1.5	98	—	—	—	27	23	—
Normosol M[2]	40	13		3	40	278	—	—	16	—	—
Balanced electrolyte solution[4]	137	5	3	3	95	—	—	—	27	—	23
Solution to treat acidosis[5]	137	20	—	—	97	—	60	—	—	—	—
5% $NaHCO_3$ (hypertonic)	600	—	—	—	—	—	600	—	—	—	—
5% Dextrose saline (hypertonic)	154	—	—	—	154	278	—	—	—	—	—

NB: Intravenous solutions should contain a bicarbonate precursor (lactate, acetate, gluconate or propionate), as the sole use of fixed bases such as chloride can cause metabolic acidosis, hypokalaemia and hyperchloraemia.

[1]Give only 1–2 L/h of solutions containing 5% dextrose, but up to 3–5 L/h for other solutions, all of which should be at 37°C.

[2]Abbot Laboratories, Illinois, USA.

[3]Diamond Laboratories, California, USA.

[4]Merritt (1975) in Rose (1981).

[5]Rose (1979) in Rose (1981).

gastric **ulceration** (Holbrook *et al.* 2005). Exercise itself causes a lowering of gastric pH and is a risk factor for ulceration of the gastric squamous epithelium. Pretreatment with a proton pump inhibitor may be helpful in reducing this adverse reaction.

Thirst, recovery, drinking and blood osmotic pressure

Thirst is in part controlled by the osmotic pressure of the blood and therefore it is frequently necessary to rectify electrolyte loss (Tables 9.3 and 9.4) before dehydrated horses will drink adequate quantities of water. Electrolyte mixtures are widely available commercially. If the horse is also acidotic, then plasma K could rise, despite a K deficit, as intracellular K is exchanged for H^+ ions. Subsequently, renal losses of K increase, ultimately causing severe K depletion (see Chapter 11, assessment of K status).

Horses should be allowed to drink frequently during extended work and at least 2 min should be allowed on each occasion. If the weather is very hot, then a drink at least every 2 h is desirable. Hard work diverts much of the blood supply to the skeletal muscles away from the splanchnic bed of blood vessels serving the GI tract. It is thought that this inhibits the efficient absorption of water, so that the amounts consumed on any one occasion should be relatively small. The consumption of large volumes should be avoided because of the large difference between

Table 9.4 Composition (g/kg DM) of mixtures for administration by stomach tube and as a daily supplement.

Mix*	Glycine	Sodium chloride	Monopotassium phosphate	Magnesium sulphate	Potassium chloride	Calcium carbonate	Calcium gluconate	Sucrose or glucose*
(1)	470	270	190	13			57	
(2)		325			325	175		175
(3)		170			70			100

N.B.: Mixes (1) and (2) can be squirted as a thick slurry to the back of the mouth *after* water has been given, to rectify a water deficit and thirst. Mix (3) is a suggested simple mixture to be given in the amounts shown daily as a dry supplement mixed with bran or other palatable dry material to a 500 kg horse during periods of extreme effort in hot weather. Fresh water must be freely available. (NB: It is usually more 'palatable' dry than as a solution.)

In order to provide approximately isotonic solutions, add 230 g mix (1) to 6 L water, or 120 g mix (2) to 6 L water, given every 2–3 h for a 500 kg horse.

*Can be replaced by molasses.

it and blood in osmotic pressure. After work, very dehydrated animals should be given about 4.5 L (1 gallon) every 15 min, preferably containing 30 g electrolytes. If the horse will not drink, administration by stomach tube is sometimes necessary. Dehydration is frequently accompanied by coldness and fatigue, muscular tremors, colic, thumps, lack of appetite and a low pulse : respiration-rate ratio. Severely dehydrated animals are sometimes given a 5% glucose–electrolyte solution i.v., while heart rate is monitored, when their ability to absorb fluid from the gut is in doubt (Table 9.3).

Where there has been a contraction of blood volume, the administration of electrolytes has only a transient effect on increasing that volume. Nevertheless, rectification of losses and of the acid–base balance must be considered. The total osmotic pressure of the blood depends to a large extent on the colloids it contains, the principal ones being proteins, more especially albumin. However, protein loss during work will be minimal and reflects only its metabolism as an energy source apart from very slight losses through pulmonary haemorrhages. The rebuilding of energy reserves, particularly in respect of muscle glycogen, will take several days after extended hard work.

Fatigue and interval training

Fatigue during extended exercise partly results from a decline in blood glucose concentration. The rate of this decline is affected by velocity, by the extent of glycogen stores, by dietary manipulation that spares glycogen mobilization and by training that promotes a greater use of both fat and glycogen during maximal exercise (Snow & Mackenzie 1977a). Fatigue is first exhibited by a decrease in speed during the course of running. Contributory factors to fatigue include a loss of muscle ATP, depletion of muscle glycogen, metabolic acidosis and blood lactate and ammonia accumulation. Fatigued horses can still possess a mean of 400–500 mmol glycogen/kg muscle tissue, but the muscle fibres used predominately in submaximal extended exercise, that is, the type I fibres (slow-twitch oxidative) are almost depleted and the type IIa fibres (fast-twitch oxidative) show a reduction in glycogen content.

To obtain a large reserve of glycogen, there is an advisable limit set for sprint training. Excessive training leads to an increase in the time over 1200 m. This is associated post-exercise with an increase in blood lactate, a decrease in plasma cortisol concentration and PCV and decrease in body weight and V_{200} (Hamlin *et al*. 2002). Tapered training (maintaining the intensity but reducing the volume of work before a race) over seven days before a 2400 m individual timed trail reduced fatigue and improved performance in comparison with constant training. The introduction of interval training to the tapered system increased the risk of injury (Shearman *et al*. 2002).

The object of interval training is to increase the volume of work accomplished in a single training session by providing exercise in bouts separated by recovery periods. This system allows some recovery of the muscle glycogen reserves and a reduction in heart rate and plasma lactate concentration during rest periods. The reduction in plasma lactate will be accelerated by trotting the horse between bouts, rather than by walking or cantering. Repeated high-intensity interval training has caused loss of performance

Table 9.5 Effects of racing 1900–2500 m on concentrations of blood lactate and acidity and their relationship to exhaustion (Krzywanek 1974).

	Blood lactate (mEq/L)			Blood pH		
		After race			After race	
	Before race	Immediately post	15 min post	Before race	Immediately post	15 min post
Exhausted	0.58	23.14	24.37	7.379	7.086	7.105
Not exhausted	0.63	17.99	16.92	7.379	7.164	7.213

capability, body condition and weight and so should be carefully monitored.

Measurement of fitness and exhaustion

A medium-paced canter is normally accomplished with aerobic respiration so that the ratio of lactate to pyruvate in the blood is unchanged. However, during galloping, the ratio rises sharply, and an effect of training is to induce smaller changes in blood lactate and in the lactate : pyruvate ratio. Blood lactate concentration is nevertheless unrelated to racing speed, but as it is quite strongly acidic, it tends to be correlated with blood pH. Blood lactate concentration is partly an expression of adaptation to training. Persson (1983) proposes that the estimation of blood lactate at a work-induced heart rate of 200 bpm could be used to gauge fitness in training, where lower lactate concentrations down to 2 mmol/L would reflect greater fitness (McGowan 2008a). Heart rate would be measured by telemetry on the track and the results would be unaffected by track conditions that affect speed.

In addition to dehydration and electrolyte depletion, blood acidity is a dominating factor in determining exhaustion (Table 9.5) and serum levels of the enzyme CK provide an indication of the severity of exercise and metabolic acidosis. In one study, where horses raced at the rate of 700 m/min (26 mph) serum AST rose 50%, serum Ca rose 13%, but serum CK rose 227% (Williamson 1974). It is thought that the increase in serum concentration of muscle enzymes after exercise is explained by an increase in the permeability of muscle cell membranes, owing to hypoxia. Thus, inadequate training, or severe work loads, will induce a greater increase in the serum concentration of CK.

Age and training

Amongst 12- and 22-year-old mares subjected to two graded exercise tests plasma lipid hydroperoxide (LPO) concentration was positively correlated with plasma lactate concentration (Williams *et al.* 2008). Total glutathione concentration increased during acute exercise in both groups, but its concentration was greater after the first exercise test than after the second. Apoptosis of white blood cells during exercise was less in the younger mares. The authors concluded that age did not affect oxidative stress, as measured by LPO, that training helps horses adapt their glutathione system for intense exercise, and that greater white blood cell apoptosis indicates that exercise can compromise the immune system of older horses. This subject is reviewed by Kirschvink *et al.* (2008).

Ammonia and the alanine vehicle

The accumulation of ammonia (NH_3) in the blood of exercised horses probably contributes to fatigue, although the experimental evidence is weak. Ammonia causes pyruvate to accumulate, which in O_2-limited states, is converted to lactate, resulting in a decrease in muscle pH. Blood ammonia concentrations have been decreased by dietary monosodium glutamate (MSG) supplementation. Plasma ammonia and uric acid concentrations rise and attain maxima during recovery (Harris *et al.* 1987; Miller-Graber *et al.* 1987; Miller-Graber & Lawrence 1988), signifying an increased rate of adenine nucleotide cycling. A greater demand for ATP is partly met by the myokinase reaction ($2ADP \rightarrow 1ATP + 1AMP$) (Figure 9.11; Frape 1994). Deamination of AMP, which supplementary $NaHCO_3$ can restrict, produces NH_3 and IMP, yielding uric acid (Harris *et al.* 1987) and alanine as the ammonia vehicle. Additional dietary protein may or may not aggravate this (Figure 9.11).

Summary of fatigue

The first requirement of exhausted dehydrated horses is water, followed closely by electrolytes. Bodily energy resources must then be rejuvenated, and, if the weather is cold, the horse must be kept warm but not hot. Metabolically some ATP is reformed by the myokinase reaction in the presence of pyruvate, but generally the fuels for ATP synthesis are depleted. After normal hard work a horse should be cooled by gentle exercise of the muscles through

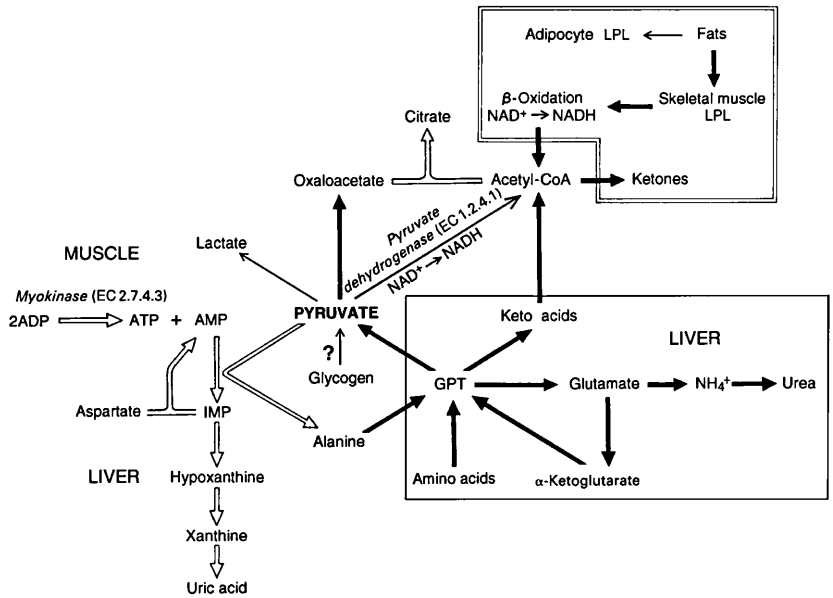


Figure 9.11 Key: Proposal for reactions stimulated (➡), not measurably influenced (⇨) or suppressed (→) by high protein or high fat diets during equine exercise of moderate intensity in the post-absorptive state. Reactions proposed to be stimulated by dietary fat (▣) and by dietary protein (□). GPT, glutamic-pyruvic-transaminase (EC 2.6.1.2); LPL, lipoprotein lipase (EC 3.1.1.34); IMP, inosine monophosphate. (Frape 1994.)

walking, ridding the muscles of waste products, but access to light grazing or hay should not be ruled out. After this relaxation of 1–1.5 h, tepid water should be given before a light meal of concentrates. It can be concluded that exhaustion during long-distance work is an expression of nutrient depletion whereas during sprint work it is principally the result of raised blood lactic acid and a consequential fall in blood pH.

BLOOD ACID–BASE BALANCE

What is an acid?

An acid is a substance, such as lactic acid, that yields hydrogen ions in solution. The acidity of blood or other solutions is expressed as the pH (the negative logarithm of the H^+ ion concentration). Acids and bases are produced during the metabolism of nutrients, and abnormalities in the acid–base balance result from dysfunction, or overloading, of general metabolism and respiration. The normal pH of arterial blood is 7.5 and that of venous blood 7.4. The blood carries CO_2 to the lungs, partly in the form of weak carbonic acid (H_2CO_3), one of the principal acids of fizzy drinks. Carbonic acid and haemoglobin act as the principal buffers in blood; that is, they prevent the pH from shifting appreciably and so prevent death. In the plasma, CO_2 reluctantly and slowly forms H_2CO_3, but on diffusing into the red cells this reaction is accelerated 13 000-fold by the presence there of the enzyme carbonic anhydrase. Despite the accelerated change, only 1 part in 800 of CO_2 forms H_2CO_3. This is described in Figure 9.12, which shows that nearly all this H_2CO_3 dissociates into H^+ and bicarbonate (HCO_3^-) ions. The former are partly buffered by haemoglobin and the latter to a large extent diffuse back into the plasma so that around 20 times as much CO_2 is carried as HCO_3 as remains in the form of dissolved gas. The dissociated form of H_2CO_3 in blood solution is in a constant (K) proportion to the undissociated form, as shown below:

$$\frac{[H^+]\times[HCO_3^-]}{[H_2CO_3]} = K$$

If acid is produced during muscular activity, or during intestinal colic (see Chapter 11), H^+ ions in the numerator increase and these react with HCO_3 forming CO_2 and water (Figure 9.12). In this process the HCO_3 concentration falls, but the H^+ ion concentration does not rise as much as it would in the absence of the HCO_3 buffer, and so the ratio $HCO_3 : CO_2$ governs the pH of the blood.

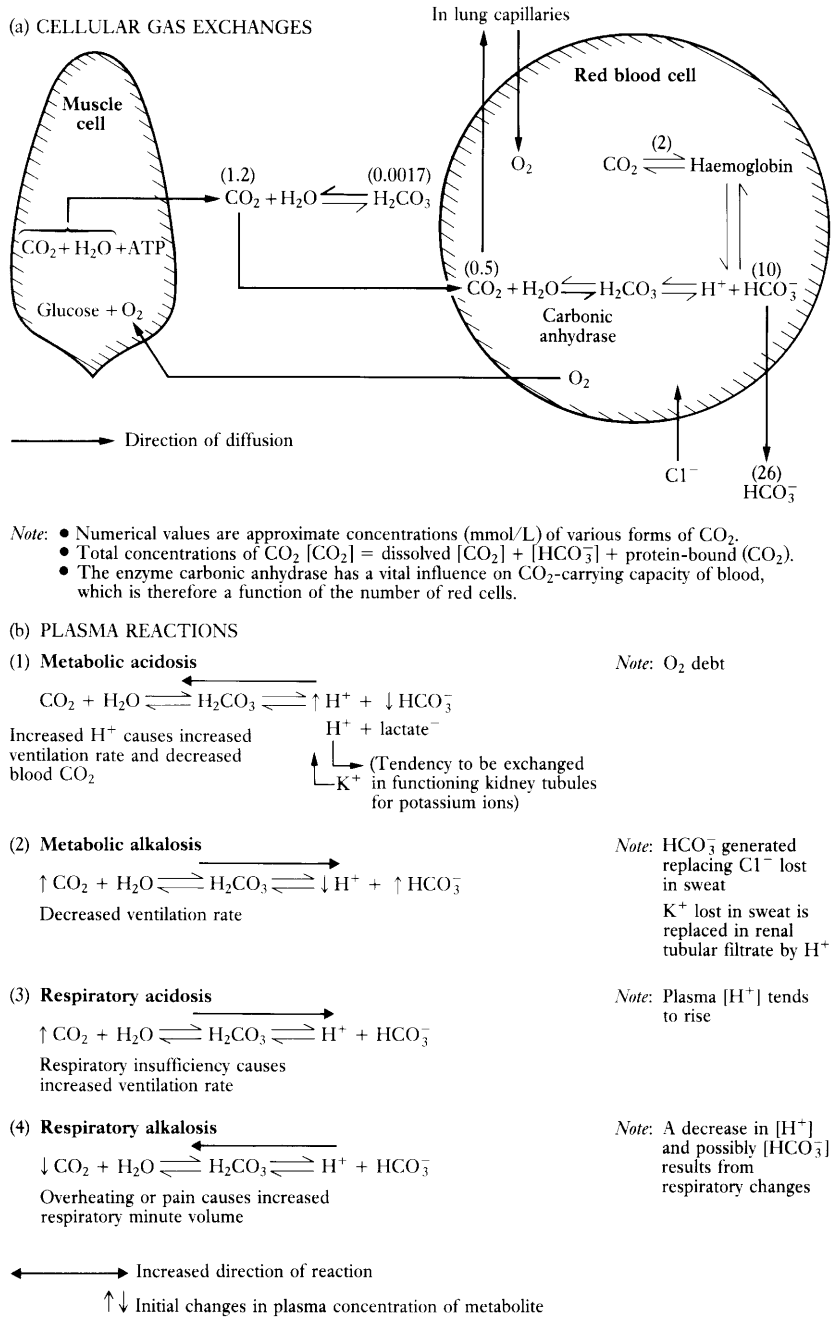


Figure 9.12 Effects of metabolic state on carbonic acid in blood plasma and associated relationships in muscle cells and red blood cells.

Acidosis and alkalosis

Fatigue during exercise is associated with a deviation of blood pH from the ideal range, causing metabolic acidosis or, with overheating, respiratory alkalosis. In lactic acidosis there is increased production of H^+ ions owing to an O_2 debt. With overheating there is increased loss of CO_2, resulting from a high **respiratory minute volume** (Frape 1994). Metabolism for all important functions occurs most efficiently when arterial pH approaches the normal value of 7.5. Deviations are associated with losses of important ions through the kidneys and intestinal tract, causing a burden on metabolism which has eventually to

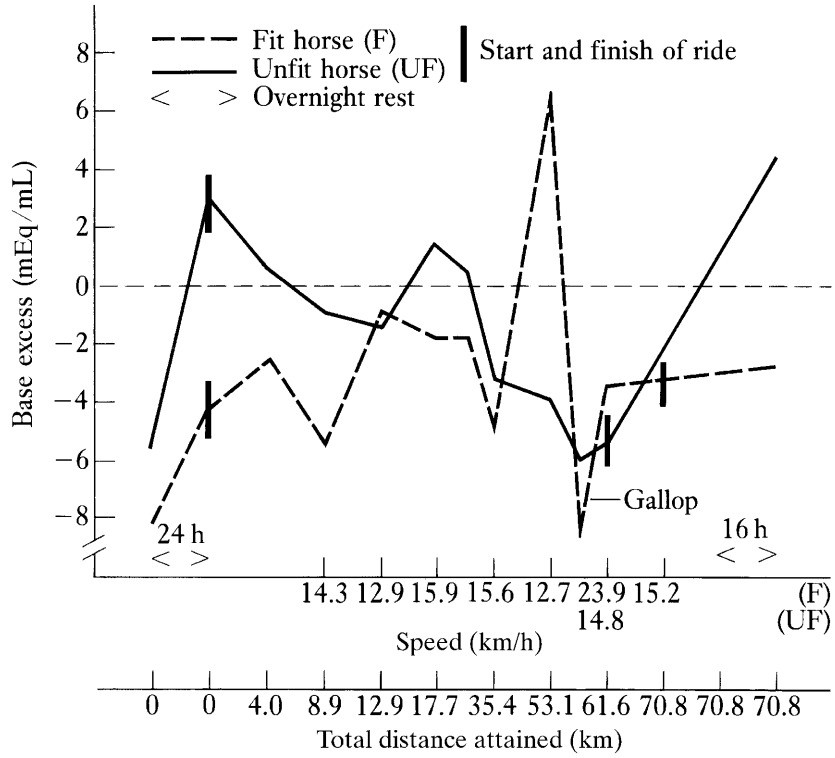


Figure 9.13 Changes in plasma BE during an endurance ride by a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979). BE of the unfit horse declined from the start. Its respiration rate was consistently the higher, which could imply that a higher ventilation rate was removing more CO_2 from blood and carbonic acid reaction was moving to left (Figure 9.12b '(4) Respiratory alkalosis' equation). Lap eight was run at lowest speed (Figure 9.10) and, although respiration rate of the unfit horse did not fall (Figure 9.6), the low rate of the other horse immediately allowed the BE to rise. This lap was followed by a rest period before the fit horse was galloped.

be rectified. A lowering of pH may result from an excessive rate of acid production during exceptional work or from disease states of the intestinal tract and associated organs, kidneys and lungs in particular. Organic acids produced in metabolism have only short-term consequences as they should be ultimately disposed of by metabolism and respiratory compensation. Fixed acids and bases (those that cannot be metabolized to CO_2 and water and exhaled) absorbed from digesta have a longer term influence and attention has been focused on the fixed cations and anions present in diet and their deducible effects on acid–base balance. Bone acts as a buffer to prolonged dietary imbalances of this kind. An acid diet, containing an excess of fixed anions, leads to bone resorption and osteoporosis so that renal excretion of those anions can proceed.

Base excess

A quantitative measure of the acid–base status of a horse, as affected by metabolism, health and diet, is known as the base excess (BE) (Figure 9.13). This is the base content of the venous blood measured by titration with a strong acid to a pH of 7.4 at normal CO_2 tension. Base deficit is the same as negative BE and its measurement requires titration with a strong base, again to a pH of 7.4. At that pH, BE is zero and plasma bicarbonate equals approximately 22–25 mEq/L. In the normal horse, bicarbonate should be in the range of 24–27 mEq/L and the BE 2–5 mEq/L.

Function of the lungs and kidneys

The lungs have a vital short-term role in the acid–base balance by providing a route for the excretion of carbonic

acid (Figure 9.12). Although the kidneys also function in this role, quantitatively they are insignificant, as the lungs dispose of 200 times more of the acid. However, the kidneys fulfil a long-term role in disposing of nonvolatile acids and bases from the diet (see 'Dietary BE and 'fixed' DCAB', this chapter). In this balance, and in that between acids and bases, an important principle is disclosed. This is the principle of *electroneutrality*, in which all urinary and lung excretion and in all movement across normal cell membranes in the horse, no electrical charge can accumulate, as in a battery. The number of anions has to correspond with the number of cations moving in the same direction. This leads to pH changes of various bodily fluids and the concept forms a basis for an understanding of the issues involved in acid : base and cation : anion balance.

Like most laws, this one is only approximately true as it conflicts with that of **iso-osmolarity**, in which all bodily-fluid compartments (between which water is exchangeable through semipermeable membranes) approach isotonicity. In achieving this, a small voltage gradient occurs as, for example, between ICF in the muscles and ECF. These principles play a central part in muscular ailments, such as 'tying-up' (see Chapter 11). In so far as their acid–base status is concerned, horses are subject to four types of abnormal metabolism. Only those conditions relating to physical work will be discussed here.

Metabolic acidosis

In strenuous exercise, an O_2 debt, with a tissue accumulation of lactic acid, occurs to varying degrees. It is also possible that this form of acidosis exists in combination with a B-vitamin deficiency, causing an incomplete metabolism of pyruvic acid, but this proposition has not yet been substantiated. The build-up of H^+ ions causes hyperventilation and thus an increase in respiratory minute volume with a fall in blood partial pressure of carbon dioxide (pCO_2). This shifts the carbonic acid equation to the left (Figure 9.12), which brings about some respiratory compensation of the acidosis and prevents an excessive fall in pH. In an analogous situation, the raised H^+ ion concentration and pain of laminitis will have a similar respiratory effect so that in its advanced state, respiratory alkalosis supervenes. Sodium bicarbonate, at a rate of 250–300 g (3000–3600 mEq) over 24 hours is sometimes given as a therapy for metabolic acidosis, but its unconsidered use can produce untoward effects. Moreover, the feeding of excessive sodium chloride (NaCl) will exacerbate an exertion-induced acidosis (Hinchcliff *et al*. 1993).

Metabolic alkalosis

Alkalosis can occur in chronic laminitis as well as from exhaustion after long-distance cross-country work in hot weather. A reduction in ventilation rate in the worst-affected horses, however, induces slight acidosis. During extended work a depletion of body potassium and chloride develops in varying degrees. H^+ ions are excreted as a substitute for the depleted potassium, and bicarbonate fills the anion gap after loss of chloride. Tetanic spasms sometimes result *in extremis* from a decreased availability of ionized calcium brought on by alkalosis. Unfit horses present signs of adrenal exhaustion. The consequent failure to secrete aldosterone precipitates an excessive urinary loss of sodium with potassium retention and a further decline in well-being. Sodium bicarbonate therapy would severely aggravate alkalosis, and the provision of a balanced electrolyte and glucose solution containing sodium, potassium and chloride (see also Chapter 11) is recommended. The want of potassium is not truly attested to by its plasma level, as a consequence of a shift from ICF to ECF space. Electrolyte losses and hypocalcaemia, related to a raised pH in exhausted endurance horses, are associated with a condition known as synchronous diaphragmatic flutter, in which heart and respiratory contractions coincide. In addition to electrolytes, calcium gluconate solutions are given i.v. (Table 9.3).

Respiratory acidosis

This is the build-up of blood CO_2 (hypercapnia) which arises with the respiratory insufficiency of sprint work. The condition may also occur in several diseased states of the lungs, including respiratory allergy, and the resulting decline in oxygen tension (hypoxia) in the blood causes metabolic acidosis.

Respiratory alkalosis

Hard work in hot weather brings about overheating which directly affects the respiratory centre of the brain, causing rapid breathing. Rapid ventilation rate flushes CO_2 from the blood. The pain of colic and laminitis also accelerates respiration rate with similar consequences. The effect is to induce a slight rise of blood plasma pH (Figure 9.14), although an absence of change in endurance horses reflects a concomitant fall in both CO_2 and HCO_3.

Factors controlling plasma pH

Plasma $[H^+]$ is regulated by several independent variables, especially:

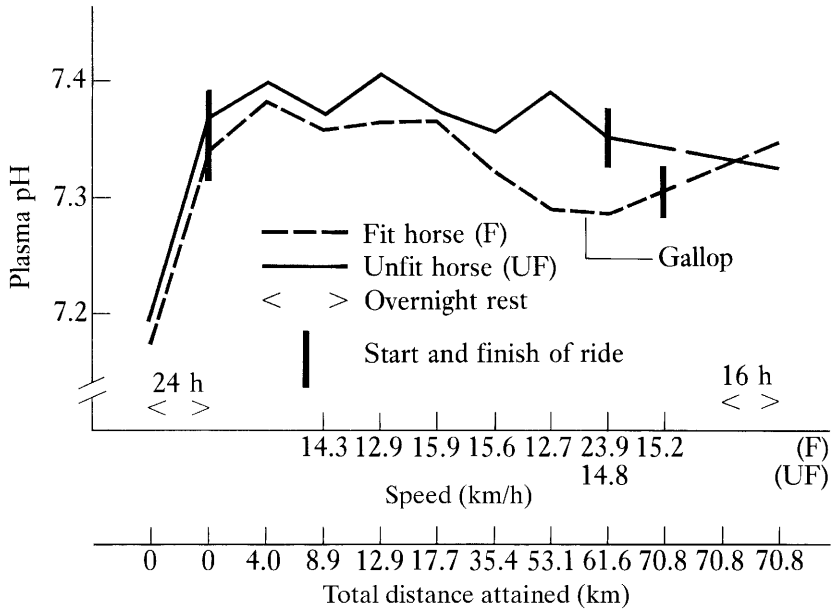


Figure 9.14 Effects of an endurance ride on venous blood plasma pH in a fit Arab stallion and a less-fit pony gelding (Frape *et al.* 1979).

- partial pressure of CO_2, i.e. pCO_2, controlling $[H^+]$ and HCO_3^-;
- strong ion difference (SID), i.e $[Na^+] + [K^+] - [Cl^-] - [lactate^-]$; and
- weak electrolytes $[A_{total}]$, represented by [albumin].

A decrease in plasma SID is normally associated with a decrease in plasma pH. This points to an increase in plasma $[H^+]$ implying there is insufficient HCO_3^- available with which it can combine to maintain the constant K in the bicarbonate equation (see 'What is an acid?', this chapter). (N.B. The function of dosing with $NaHCO_3$ is to provide that bicarbonate ion. Also, the Na^+ ion balances the lactate$^-$ ion, the concentration of which does not necessarily decline in plasma.)

DIETARY BASE EXCESS AND 'FIXED' DIETARY CATION–ANION BALANCE

Dietary BE

Mineral nutrition has a role in acid–base balance. This follows from the premise that excess base, or acid in feed is estimated as the difference between the sums of mineral cations and anions, approximated by:

$$\text{Dietary BE (mEq/g)} = (\text{Na} + \text{K} + \text{Ca} + \text{Mg}) - (\text{Cl} + \text{P} + \text{S})$$

The effect that diet and metabolic acid production have on plasma BE is shown in Appendix D. Typical horse diets have a dietary BE of 200–300 mEq/kg, estimated from the fixed-ion content. Thus, for a horse consuming 10 kg feed/day, the BE would amount to approximately 2500 mEq, which is similar to that provided by 200 g sodium bicarbonate – a quantity frequently given in therapy over a period of 24 h. Horses with a serious base deficit might require double the amount contained in a normal ration over that period. The dietary optima assume that the protein content is probably higher than that provided for the average adult horse. For a diet containing 10% crude protein, the dietary BE might be of the order of 30 mEq/kg less.

The absorption of Ca, Mg and P is limited and variable so that DCAB is frequently measured in a simplified form as mEq/kg dietary DM:

$$\text{Dietary BE (mEq/kg)} = (\text{Na} + \text{K}) - (\text{Cl} + \text{S})$$

(N.B. In some studies, the dietary S, normally providing 70–80 mEq/kg, is ignored.) On this basis, by including 10 g $CaCl_2$/kg in the 'medium' diet DM (Table 9.6) to give the 'low' diet and 13 g $NaHCO_3$/kg to give the 'high' diet, the urinary and blood pH were also affected.

Table 9.6 The effect of dietary BE on urinary and blood pH.

	Low	Medium	High
mEq/kg diet	22	202	357
Urinary pH	5.38	7.69	8.34
Blood pH	7.368	7.400	7.402

In the GI tract, H^+ ions are exchanged for Ca^{2+} and Mg^{2+} ions on consumption of the 'low' diet. Urine is excreted in an electrically neutral state and, during exercise, immature horses given this diet could experience a considerable loss of urinary HCO_3^- and Ca^{2+} (hypercalciuria), accounting for metabolic acidosis and a negative Ca balance, demineralization and a weakening of the skeleton. Cooper *et al.* (1995) found raised urinary Ca^{2+} and Cl^- values in horses given a diet with a DCAB of −25.7 and relatively greater urinary losses of P, Na^+ and K^+ in horses given a diet with a DCAB of 370.4. Diets with a DCAB > 200, as indicated above, reduce the risk of metabolic acidosis. Lower blood pH, pCO_2 and $[HCO_3]$ values are found at rest (Baker *et al.* 1992) and subsequent to anaerobic exercise (Stutz *et al.* 1992) with a dietary (Na + K − Cl) balance of 5–21 cf. 107–125 or more mEq/kg. Horses receiving 107 mEq/kg, or less, recovered normal blood glucose more slowly after exercise than did those receiving 201 mEq/kg, or more. To maximize the transitory buffering effect of diet, the optimum time for anaerobic exercise is 3.5–4.5 hours after feeding a light meal (Stutz *et al.* 1992), despite an increased fluid load (Meyer 1992). Popplewell *et al.* (1993) recorded faster times over 1.64 km and lower heart rates after racing 2–4 h after a meal with a balance of 295 cf. 165 mEq/kg DM, despite higher blood lactates. Moreover, a balance of 354 mEq/kg DM achieved a greater Ca balance cf. 223 mEq/kg (Wall *et al.* 1993). The optima for balance and total mineral ion load is, however, likely to differ between types of work – sprint *vs*. extended.

Dietary protein

The effects of dietary protein level on performance and metabolism during exercise are discussed in this chapter under 'Dietary protein requirements and exercise'. The effects of excess dietary protein, or that inevitably oxidized, on acid–base balance have not been addressed. The effects are small, but the basis for drawing conclusions should be summarized.

Only absorbed products of protein digestion can influence cation–anion balance. Many native proteins are rich sources of P, such as phosphorylated amino acids, e.g. phosphoserine, and so contribute to the fixed anions. The oxidation of neutral amino acids has no effect on the acid load, but three classes of these nutrients do.

(1) the basic (cationic) amino acids (lysine, arginine and histidine) yield neutral end products plus a proton (H^+) and thus are strangely acidogenic;
(2) the sulphur-containing amino acids (methionine, cystine and cysteine) are also acidogenic, as they generate sulphuric acid when oxidized; and
(3) the dicarboxylic amino acids (aspartic and glutamic acids) are anionic, but metabolizable, and consume protons when oxidized, and so reduce the diet's acid load.

Protons can be excreted in the urine but primarily as the ammonium ion $H^+ + NH_3 \rightarrow NH_4^+$. Glutamine is the principal amino acid involved in renal ammonium ion genesis, and although not an essential dietary nutrient, it is used up in this process (see 'Protein assimilation', this chapter). Horse diets frequently have lysine and threonine as first and second dietary essential amino acids and consequently there is interest in adding lysine-HCl to make good any limitation. When supplied as their Cl^- salts they are a fixed anionic, or acid, source. However, if 0.1%, or 1 g lysine-HCl/kg diet is added, it provides only 5.5 mEq acid/kg (see 'Dietary protein requirements and exercise' this chapter).

The effect on acid load of the rapid metabolism of amino acids during intense exercise are important at the margin, but the amounts of acid produced are dwarfed by that produced through carbohydrate metabolism. Moreover, it has not been established whether the rate of acid production from protein is markedly influenced by the dietary level of protein.

Effect of DCAB on digestibility and Ca and Mg retention

The DCAB has been artificially adjusted by additions of calcium chloride, ammonium chloride, potassium citrate and sodium bicarbonate, giving a range in balance of 20–400 mEq/kg diet. There is an increase in DM digestibility with an increase in positive balance. Hypocalciuria, promoting an increased retention of Ca and Mg, can also result from an increase in the balance, reducing the chronic risk of osteoporosis. A low DCAB induces metabolic acidosis, causes hypercalciuria, decreased Ca and Mg balances, and when horses are exercised within 4 h of feeding, it leads to poorer work output and a slower recovery, owing to a poorer dietary buffering effect (Popplewell *et al.* 1993).

Supplements

Cofactors

The activity of enzymes depends on the presence of the necessary cofactors and an increasing demand for these enzymes implies an increasing need for the cofactors. These cofactors include magnesium and zinc together with forms of the vitamins thiamin, riboflavin, niacin, pantothenic acid, pyridoxine, biotin and vitamin B_{12}, all of which play major parts in carbohydrate and/or fat metabolism. The horse derives these B vitamins from its diet and by microbial synthesis in its intestine. As the intensity of work increases, the composition of the diet and the amount of feed consumed change as a consequence of the increased consumption of starchy cereal grains. This will alter not only the dietary supply of B vitamins, but also the intestinal synthesis of the vitamins, and it is an open question whether the rate of their absorption is exceeded by tissue demand when horses are in intensive training.

The microbial fermentation of starch cf. fibre yields a higher proportion of propionate in the VFAs. Metabolism of this acid requires adenosylcobalamin (vitamin B_{12}), as the coenzyme of methylmalonyl-CoA mutase, and work with ruminants has revealed that such diets create a dietary requirement for this vitamin (Agricultural Research Council 1980), the lack of which causes an accumulation of propionate, depressing appetite. Unpublished observations by the author of horses in training have shown that their blood concentrations of vitamin B_{12} are lower than in many other horses and that the palates of those with flagging appetites might be whetted by supplements of the vitamin. A reasonable inference is that an increased consumption of cereal grains by horses increases propionate production and hence the dietary requirement for vitamin B_{12}. An analogous argument is put for thiamin, functioning as cocarboxylase in the cleavage of pyruvate. Studies by Topliff *et al.* (1981) suggested that exercising horses have a thiamin requirement double that of non-working horses. TBs in training have low serum folate concentrations, but whether this simply reflects a lower potency of training diets has not been established. Thus, there is no conclusive evidence concerning the effect of work on the requirement for B vitamins functioning as enzyme cofactors.

Amino acids

BCAAs stimulate protein synthesis for which growth hormone is a mediator. Oral supplementation of athletes with BCAAs (0.2 g/kg BW daily for one month) cf. controls led to a raised plasma concentration of growth-hormone-binding protein, slightly elevated growth hormone and lower plasma lactate concentrations (De Palo *et al.* 2001). The author is unaware of any similar evidence in horses.

Water and electrolyte loading

Despite normal variation in the levels of each of the dietary electrolytes, adaptation serves to maintain the pH of body fluids in the normal physiological range. Outside this normal range, tissue pH is altered through an overload of compensatory mechanisms. Excretion of excess fixed ions requires water as a solvent, increasing water demand. Complete water restriction for 20 h before exercise has been shown to reduce the intestinal water content by 10% at the start of protracted exercise, and exercise reduces it by a further 15–20% through sweat losses, regardless of whether drinking had been allowed. Horses that are severely dehydrated are exceptionally exhausted (Carlson *et al.* 1976), and are reluctant to drink. Even during a ride, spontaneous drinking might not occur when water is offered if there has been an iso-osmotic loss in sweat, unless the fall in plasma volume exceeds 6% (Sufit *et al.* 1985) and especially if electrolytes are not given. Water and balanced electrolytes should therefore be given frequently during a ride.

Sodium chloride feeding before a ride could encourage water intake *then* and so should improve water balance during a subsequent extended ride. Loading the horse with electrolytes tends to increase their temporary accumulation, and that of water, in the large intestine (Slade 1987). This could act as a reserve of water, Na^+ (Meyer 1992) and of Cl^- (Coenen 1992a) for extended work. The daily ileocaecal flow of water and electrolytes per kg BW is in the range of 100–140 mL water, 300–420 mg Na^+, 50–70 mg K^+ and 100–140 mg Cl^-. Absorption along with water from the large intestine, during ingesta fermentation, is estimated to be 75–95% for Na^+, over 90% for Cl^- and 30–55% for K^+ (Meyer 1992). These nutrients can revive tissue depleted of water, Cl^-, Na^+, K^+ and Ca^{2+} through sweating (Rose *et al.* 1977). The benefit of such an electrolyte reserve is promoted by the presence of fermentable material in the hind-gut, as this allows the continuous absorption of the reserve ions during a ride.

The amounts of dietary electrolytes necessary to keep a horse in electrolyte balance have been assessed to be 1.3–1.8 g Na, 3.1–3.9 g Cl, 4.5–5.9 g K, 8.5 mg Ca and 10.7 mg P/Mcal DE (0.3–0.4 g Na, 0.7–0.9 g Cl and 1.1–1.4 g K, 2 mg Ca and 2.6 mg P/MJ DE). Thus, the requirements for Na, K and Cl during exercise are increased over maintenance needs by three-, seven- and six-fold, respectively, according to Potter's group in Texas (Hoyt *et al.* 1995a).

Natural feed, given after rides, is likely to contain much more K than Na. There should therefore be about twice as much Na as K and 1.2 times as much Cl as Na in supplements given with these feeds (Table 9.4). Organic anions can make up the residue. Small amounts of Ca and Mg can also be included.

Sodium bicarbonate

Anaerobic exercise causes a rise in plasma K^+, released from the contracting muscle fibre. A failure of its reuptake results from an inhibition of the Na–K pump of the muscle fibre membrane, owing to decreased ATP availability (Harris & Snow 1988) through inadequate buffering of H^+ ions within active fibres (Harris & Snow 1992). The loss of intracellular K^+ leads to an altered transmembrane potential that can contribute to fatigue during exercise.

To counter this, oral sodium bicarbonate ($NaHCO_3$), which increases plasma SID, results in a smaller rise in plasma NH_3 through a lower ATP loss and IMP formation (Greenhaff *et al.* 1991b), HCO_3^- accelerating H^+ removal (the bicarbonate system is the major proton acceptor in the body). A positive effect seems to occur only where the duration of exercise is 2–3 min, accounting for its particular use with Standardbreds engaged in races over 1.6–2.4 km. However, the use of alkalizing agents before a race is discouraged, or leads to disqualification, in many racing jurisdictions. No effect of $NaHCO_3$ was observed over 1 km (Greenhaff *et al.* 1991b). Lawrence *et al.* (1987a, 1990) reduced race times by 1.1 s over 1.61 km with Standardbreds treated orally with 0.3 g $NaHCO_3$/kg BW mixed with 20 mL corn syrup and 10 mL water, cf. powdered confectioner's dextrose and salt mixed with corn syrup and water, 2.5 h before exercise. Treatment increased both blood pH and lactate disappearance rate after racing. Harkins & Kamerling (1992) treated TBs with 0.4 g $NaHCO_3$/kg BW in 1 L water, cf. 1 L water only, 3 h before a 1.61 km race, increasing venous HCO_3^- and pH. After the race, there was an increase in venous blood pH and lactate in the $NaHCO_3$ group, with no change in race times, or in venous pCO_2. (N.B. The lactate ion $[Lac^-]$ is neutralized by Na^+ and does not determine the pH. Alternatively, it can be argued that the increase in $[Na^+]$ reduces plasma $[H^+]$ through the maintenance of electroneutrality, despite an increase in plasma $[Lac^-]$. This increase in $[Lac^-]$ is probably the result of increased efflux of Lac^- from muscle cells caused by extracellular alkalosis, so reducing fatigue.) The optimum dose and time are 0.4 g $NaHCO_3$/kg BW (in 1 L water) 2–4 h prior to work, as assessed by blood pH and HCO_3^- (Greenhaff *et al.* 1990b; Corn *et al.* 1993), although doses of 1 g/kg BW have achieved higher values with a peak 4 h after administration.

Analysis of venous blood from Standardbred pacers before racing has revealed HCO_3^- values in excess of 40 mmol/L, indicating higher doses than 0.4 g/kg. A dose of Na equivalent to 20% of the body's total exchangeable Na should increase plasma volume, which could have an opposite effect on sprint performance to that of a buffer, and accounts for variable results following $NaHCO_3$ administration. Moreover, the value of the large intestine as a source of Na is modulated by acetate production (Argenzio *et al.* 1977), which varies with the interval from, and nature of, the last meal. Lloyd *et al.* (1993) administered 1 g $NaHCO_3$/kg BW, cf. a similar molar dose of NaCl or water only. The $NaHCO_3$ extended exercise to exhaustion on a treadmill and increased blood lactate, but a comparison with the two untreated groups, where all horses had access to water, indicated poorer endurance with $NaHCO_3$, possibly from a higher fluid load. Yet Hanson *et al.* (1993) gave horses, with and without free access to water, 1 g $NaHCO_3$/kg BW in 4 L water and found no significant difference in plasma volume. The response is, however, more complicated, as alkalosis, caused by the 1 g dose/kg, led in both studies to hypercapnia and some hypoxaemia through respiratory compensation. This ventilatory depression was not thought to have affected performance, and was probably associated with reduced tidal volume because of the tendency for a 1 : 1 linkage of respiratory frequency with stride. Moreover, intracellular $[H^+]$ exchanges for extracellular K^+, causing hypokalaemia after administration of 'milk shakes' of sodium bicarbonate. Both plasma K^+ and Ca^{2+} concentrations decline with alkalosis so that cardiac and skeletal muscle contractions may be disrupted. This contributes to synchronous diaphragmatic flutter, and other signs, after exercise.

Thus, the optimum dose, method of administration and overall effects of $NaHCO_3$ and water have yet to be determined. Nevertheless, some value results from treatment with 0.4 g $NaHCO_3$/kg BW (in 1 L water) 2–4 h prior to gallops lasting 2–3 min.

Calcium carbonate and sodium chloride

Frey *et al.* (2001) compared iso-osmolar amounts of $NaHCO_3$ (500 mg/kg BW) and $CaCO_3$ (595 mg/kg BW) with water, and demonstrated that $CaCO_3$ had no effect on blood pH or bicarbonate HCO_3^- level. On the other hand, 488 mg CH_3COONa/kg BW (pKa 4.8) produces a similar degree of metabolic alkalosis to an isocationic dose of 500 mg $NaHCO_3$/kg BW (pKa 6.1), when they are

administered 3 h before a fast mile (Frey *et al*. 1999). By comparison, it was previously noted that NaCl decreases arterial blood pH, inducing slight acidosis (Hinchcliff *et al*. 1993).

Carnosine (β-alanyl-L-histidine)

Excess production of lactic acid and H^+ is managed by an increased respiration rate. A more enlightened approach to combating the rise in intracellular [H^+] is to alter the intracellular concentration of the imidazole dipeptide buffers carnosine and its N_2-methyl derivative, anserine. Carnosine scavenges reactive oxygen species and contributes 30% of the buffering in equine skeletal muscle (Harris *et al.* 1991a). In type IIb fibres (prominent in equine muscle), it can account for up to 50% at pH 6.5–7.1, with a concentration of 188 mmol/kg DM muscle (Sewell *et al.* 1991a,b; Sewell 1992b). However, dietary supplements of histidine have not given convincing responses in delaying fatigue.

Fat supplements and exercise

Horses participating in competitive long-distance rides are required to make effective use of body-fat reserves as a source of energy to conserve glucose sources, as a severe depression in blood glucose is an indication of fatigue. Good-quality dietary oils, fats and medium-chain TAGs are all well utilized (McCann *et al*. 1987; Hollands & Cuddeford 1992; Potter *et al.* 1992b; Jackson *et al*. 2001). Medium chain triglycerides (carbon-chain lengths of 6–12) are rapidly absorbed, followed by portal transport to the liver. They are independent of mitochondrial transport enzymes and, with hepatic metabolism to ketones, are oxidized in the presence of O_2. Vegetable fats are, however, generally more readily digested than fats from animal sources.

Fats given to man and to the horse seemingly delay gastric emptying of carbohydrate, and so improve glucose tolerance, by lowering the post-meal peak plasma glucose response. Fats are not subject to microbial fermentation and their greater use decreases the risks of colic and laminitis and promotes intramuscular and hepatic fat catabolism, increasing performance at submaximal and intense rates. Nevertheless, the rate of endogenous *de novo* fatty-acid synthesis might be reduced. A diet supplemented with soya-bean oil reduced fasting plasma TAG concentrations (Orme *et al*. 1997; Geelen *et al*. 2001). The authors concluded that this was caused, at least in part, by a reduction in fatty-acid synthesis, indicated by decreased activities of hepatic acetyl-CoA carboxylase (EC 6.4.1.2) and fatty acid synthase activities.

Work in Texas with Quarter Horses used for cutting indicates that the benefits of a 10% fat supplement are adaptive and take 3–4 weeks to materialize (Julen *et al.* 1995). However, fatigue is not associated with depletion of citric acid cycle intermediates with oil supplementation (McCue *et al.* 2009). Fat supplements can delay the decline in blood glucose during endurance rides, accelerate the rate of recovery of resting pulse and respiration rates (Hintz *et al.* 1978a,b; White *et al.* 1978) and promote the recovery of resting blood glucose, reducing the risk of fatigue-related injuries. They can also reduce concentrate and total feed intake of exercised horses (Besancon *et al*. 1999) but the practical problems of adding large amounts of fat to the diet have to be addressed.

Fat yields less CO_2/mole ATP generated, decreasing plasma pCO_2. Therefore, relative to the effects of a carbohydrate diet, there is a small increase in plasma pH brought about by a fat supplement in exercised horses with decreases in heart rate, plasma [H^+] and lactate concentration, and so an increase in venous SID, with a delay in fatigue (Kennedy *et al*. 1999; Taylor *et al*. 1999). Horses given high fat diets have a lower respiratory exchange ratio, R, and lower rates of output and uptake, (CO_2/O_2) cf. those given a high carbohydrate diet (Pagan *et al.* 2002); but Zeyner *et al.* (2002b) recorded lower blood pH, raised blood pCO_2 values, higher blood glucose in blood samples before exercise, a reduction in urine volume and reduced feed and water intake in horses given a high fat diet. It is speculated that the unexpected blood pH and pCO2 responses may have resulted from lower metabolic CO_2 yield causing moderate alkalosis, overcompensated for by reduced ventilation, and/or reduced lung perfusion caused by a lower plasma volume (indicated by increased plasma protein in the high fat group).

Hard training (Hambleton *et al.* 1980) and fat supplementation with anaerobic (Pagan *et al.* 1993b) and extended aerobic (Pagan *et al.* 1987c) exercise are followed by an elevation in plasma FFAs, whereas resting FFAs are lowered by supplementation (Harkins *et al.* 1992). Thus, there could be a stimulation to β-oxidation, or to both fat mobilization and metabolism (Figures 9.15 and 9.16), sparing glycogen. In this way, triacylglycerols do not accumulate in plasma (Kennedy *et al*. 1999). These responses result from increases in muscle LPL activity and in citrate production which inhibits phosphofructokinase, one of the rate-limiting enzymes of glycolysis. This in turn results in an accumulation of glucose-6-phosphate, inhibiting glucose phosphorylation and sparing glucose oxidation.

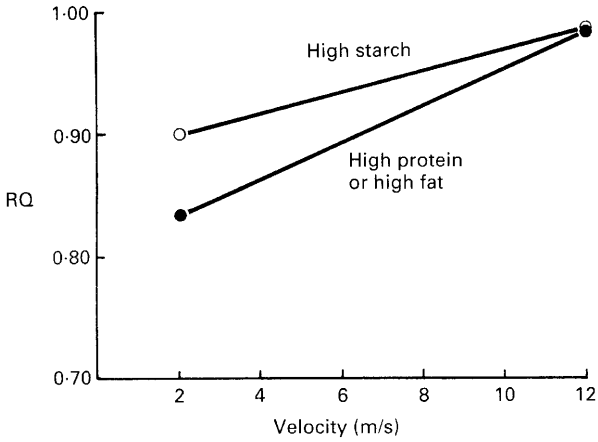


Figure 9.15 Relationship between RQ and velocity of horses given high starch, high protein or high fat diets. At high velocity there is no difference in RQ because high rate of energy expenditure demands glycolysis. At low to moderate velocity, fat and protein are used (Frape 1994).

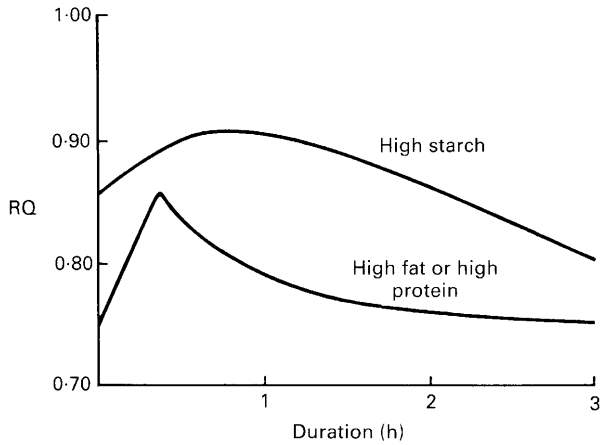


Figure 9.16 Relationship between RQ and time during aerobic work (low to moderate velocity, 4–6 m/s) in horses given high starch, high fat or high protein diets. On high fat or high protein diets, fat mobilization becomes predominant earlier, thereby conserving muscle glycogen (Frape 1994).

Evidence from Orme *et al.* (1997) and Geelen *et al.* (2001) indicated that while hepatic acetyl-CoA carboxylase and fatty acid synthase activities were reduced, fat supplementation (with pentosanpolysulphate) raised plasma total lipase, skeletal muscle citrate synthase (EC 4.1.3.7) and carnitine palmitoyltransferase-I (EC 2.3.1.21) activities. The authors concluded that postprandial plasma TAG clearance was promoted and both transport of fatty acids through the mitochondrial inner membrane and the oxidative capacity of highly aerobic muscles were enhanced by fat feeding. The addition of 100 g corn oil to the diet of sprint-trained Arabian horses (Taylor *et al.* 1993, 1995) caused, at fatigue, greater plasma glucose and lactate (11 *vs.* 8 mEq/L) concentrations, offset by increases in plasma [Na^+] and [K^+] and decreases in plasma [Cl^-]. Thus, the fat raised SID, minimized the decrease in pH of plasma and could reduce PCV in exercised horses in hot weather, possibly indicating greater reserves of water in the ECF (Mathiason-Kochan *et al.* 2001).

Three points to note concerning fat supplementation are:

(1) increased plasma lactate may cause fatigue, independent of an effect on plasma pH;
(2) the increase in lactate in Arabians is less than that observed in TBs. This could be associated with the higher proportion of slow-twitch, oxidative muscle fibres (type I), and greater oxidative enzyme activity in Arabians; and
(3) the greater rise, after exercise, in plasma lactate (and alanine, a precursor of pyruvate and glucose) with fat supplementation possibly results from the accelerated rate of glycogenolysis coupled with a reduction in the activity of the pyruvate dehydrogenase (PDH) complex and decreased oxidation of pyruvate. PDH is a key regulator of fat and carbohydrate metabolism (Figure 9.11). There can in fact be a synergistic effect of the combined treatment with fat and $NaHCO_3$, leading to even higher plasma lactate levels (Table 9.7). There is a lower rate of pyruvate conversion to acetyl-CoA, as β-oxidation increases production of acetyl-CoA. However, there is evidence that horses run at a constant velocity show lower plasma lactate accumulation with dietary fat supplements (see Table 9.10). This points to a substitution of β-oxidation for glycogenolysis, conserving glycogen stores.

During sprinting, glycogenolysis is required as an anaerobic high-power source to complement the greater oxidation of fatty acids, a low-power source, associated with fat supplementation. Although plasma lactate can be increased, there is not necessarily a decrease in plasma pH, or SID, if there are increases in plasma Na^+ and K^+ concentrations and a decrease in plasma Cl^- concentration.

Heat increment

The increased energy density and decreased intestinal residue achievable with dietary fat supplements could be the essential characteristic of fat (Hiney & Potter 1996). When 10% fat replaced starch, heat production fell from 77% of available DE to 66% and available NE rose from 16% of DE to 36% during work (Scott *et al.* 1993), reducing thermal stress (McCann *et al.* 1987), regardless of body fatness and in both temperate and hot weather (Potter *et al.* 1990). Waste heat production during exercise is very large. Approximately 80% of stored energy utilized for movement is lost as heat. The decrease in heat production achieved by fat supplementation in the main reflects diminished microbial fermentation in the hind-gut.

Table 9.7 Blood [Lac^-] during sprints in horses adapted to either a control or high-fat diet and administered either water or $NaHCO_3$ before exercise (Ferrante *et al.* 1994b).

	Blood [Lac^-] (mmol/L)
Control/water	5.73 ± 0.12
Control/$NaHCO_3$	6.26 ± 0.21
Fat/water	7.01 ± 0.20
Fat/$NaHCO_3$	9.47 ± 0.32

Muscle glycogen

Several reports indicate no difference in resting muscle glycogen concentration between high starch and high fat diets (Hintz *et al.* 1978a; Pagan *et al.* 1987a; Topliff *et al.* 1987; Orme *et al.* 1997). However, both Pagan *et al.* (1987b) and Geelen *et al.* (2001), equalizing DE intake between treatments, and Greiwe-Crandell *et al.* (1989) providing higher energy intakes with fat supplements, found lower concentrations. Most other reports describe increased resting muscle glycogen following vegetable, or animal, fat additions at approximately 10% of the diet, to provide equal DE or ME intakes (Hambleton *et al.* 1980; Meyers *et al.* 1987, 1989; Oldham *et al.* 1990; Harkins *et al.* 1992; Jones *et al.* 1992; Scott *et al.* 1992; Julen *et al.* 1995) (Table 9.8) (N.B. Post-exercise muscle glycogen concentration may be no higher as glycogen utilization could be promoted by its higher muscle concentration).

Effects of fat on hepatic glycogen capacity, which is 10% of that in skeletal muscle, are equivocal, as marginal increases (Hambleton *et al.* 1980) and decreases (Pagan *et al.* 1987b) are reported. Although fat concentrations of up to 20% of the dietary DM have been used without digestive upset, or any reduction in utilization, dietary fat levels of 15% have decreased glycogen storage compared with controls. A diet clearly needs to contain enough starch from which the storage is derived. Meyer &

Table 9.8 Resting muscle glycogen[1] as affected by added dietary fat in diets of differing energy densities but generally given to horses to equalize DE intake (Frape 1994).

Added dietary fat (g/kg diet)	0	20–30	50–60	80–100	140–150	SE	Reference
	Muscle glycogen (mmol/kg wet tissue)						
Animal fat	81	—	—	78	—	—	Hintz *et al.* 1978a
	94	—	109	143	—	10.5	Meyers *et al.* 1989
	88	—	—	127	—	2.6	Oldham *et al.* 1990
	93	—	—	145	—	2.1	Scott *et al.* 1992
	Muscle glycogen (mmol/kg DM)						
Vegetable oil[2,3]	—	200	255	292	240	—	Hambleton *et al.* 1980
	680	—	—	—	580	100	Pagan *et al.* 1987a
	198	229	—	—	—	12	Harkins *et al.* 1992

[1] *Gluteus medius*, *biceps femoris* or *quadriceps femoris*.

[2] Fat-added diet replaced maize grain giving diets of different energy densities but constant daily energy and protein intakes.

[3] Fat-added diet contained less roughage but more fat, starch and protein and was fed to provide equal DE intakes.

Table 9.9 Relationship of added dietary fat to RQ in horses exercised on treadmills (Frape 1994).

Speed (m/min)	Slope (°)	Time (min)	Added dietary fat (g/kg diet) (SE) 0	50	100	150	Reference
					R	Q	
180	9	20	0.910	0.860	0.870	—	Meyers *et al.*1989
300	0	90	0.830 (0.012)	—	—	0.750 (0.022)	Pagan *et al*. 1987b
360*	0	2	0.887 (0.019)	—	—	0.827 (0.038)	Pagan *et al*. 1987b
480*	0	2	0.890 (0.031)	—	—	0.873 (0.030)	Pagan *et al*. 1987b
600*	0	2	0.977 (0.027)	—	—	0.957 (0.043)	Pagan *et al*. 1987b

*Step-wise work test.

Sallmann (1996) found that when 2 g fat/kg BW daily was given, as much as 0.4 g fat/kg BW was transferred to the hind-gut, with a risk of disturbing caecal microbial metabolism. Hence, an optimum dietary concentration of approximately 100 g/kg exists.

Respiratory quotient and metabolism of carbohydrate, protein and fat

The respiratory quotient (RQ) is the ratio, at STP, of the volume, or moles, of CO_2 eliminated, to the volume, or moles, of O_2 utilized, in oxidation, i.e. CO_2/O_2 for carbohydrate = 1, fat = approx. 0.7 and amino acids = approx. 0.85. RQ rises with increasing speed (Pagan *et al.* 1987b), is lowered by training (Meyers *et al.* 1987) and is either not affected by dietary fat (Meyers *et al.* 1989) or is lowered by additional protein or fat during submaximal exercise (Pagan *et al.* 1987b) (see Table 9.9; Figures 9.15 and 9.16). A lower RQ implies a lower rate of CO_2 production.

A decreased pCO_2 could moderate a decrease in blood pH (through maintenance of the dissociation equilibrium of carbonic acid), so offsetting fatigue. RQ is positively correlated with muscle glycogen reserves during mild aerobic exercise and it declines as submaximal exercise progresses (Pagan *et al.* 1987b) (Figure 9.16), indicating a sparing of glycogen. Higher stores of glycogen, with fat supplementation, accelerate their mobilization rate during anaerobic exercise (Oldham *et al.* 1990; Jones *et al.* 1992; Scott *et al.* 1992; Julen *et al*. 1995). Yet a lower rate (Greiwe-Crandell *et al.* 1989), no difference (Hintz *et al.* 1978a) and a marginally greater loss of glycogen, with lower initial reserves (Pagan *et al.* 1987b), have all been reported during aerobic exercise with fat supplements (Figures 9.17 and 9.18).

No clear picture emerges that fat particularly benefits exercise in which extended aerobic metabolism dominates. Metabolic adaptation to a fatty diet could take as long as 6–11 weeks (Custalow *et al.* 1993) and some studies might not have allowed sufficient time for this, so that their outcome could have depended on design details and horse temperament. The interpretation is complicated by the expression of harder anaerobic work (Webb *et al.* 1987a) and faster speeds, with no greater glycogen loss, at a constant heart rate (Oldham *et al.* 1990) by fat-supplemented horses. As fat yields energy only by oxidation, mimimal glycogen sparing would be expected during maximal exertion, when its value is other than for conservation of glycogen.

Some workers report similar (Worth *et al.* 1987) or lower (Meyers *et al.* 1989) blood glucose concentrations with added fat during aerobic exercise, but the majority (Hintz *et al.* 1978a; Hambleton *et al.* 1980; Webb *et al.* 1987a; Oldham *et al.* 1990; Harkins *et al.* 1992; Scott *et al.* 1992; Custalow *et al.* 1993) observed higher values during and after exercise of all types (Figure 9.17 and Table 9.10), even with increased work effort (Webb *et al.* 1987a; Harkins *et al.* 1992).

Lower heart rates and a more rapid recovery of resting rates (Meyers *et al.* 1987), lower blood lactic acid concentrations during and subsequent to submaximal and strenuous standardized exercise tests (SETs) in fat-supplemented horses (Pagan *et al.* 1987a,c; Webb *et al.* 1987a; Meyers *et al.* 1989; Pagan *et al.* 1993b) (Table 9.10), a higher velocity (m/s) at which venous blood lactate attains 4 mmol/L (V_{LA4}) (Pagan *et al.* 1993b) and lactate speed threshold (Custalow *et al.* 1993) possibly reflects a slightly lower RQ (Table 9.9, Figure 9.15), but an absence of blood pH measurements complicates fatigue assessment. Higher lactates during some higher speed SETs (Ferrante *et al.* 1993; Taylor *et al.* 1993) with fat cf. starch might represent a tendency for the response curves to cross over at these speeds (Custalow *et al.* 1993).

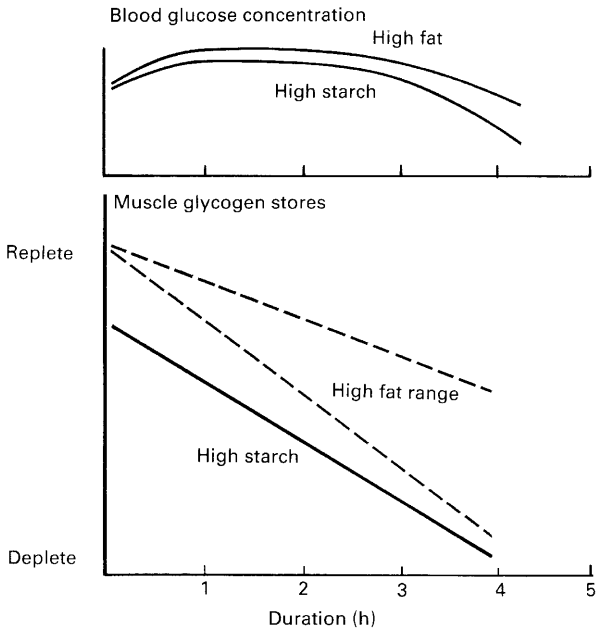


Figure 9.17 Generalized relationships, with time, of blood glucose and muscle glycogen concentration in horses of moderate fatness during extended aerobic work (Frape 1994).

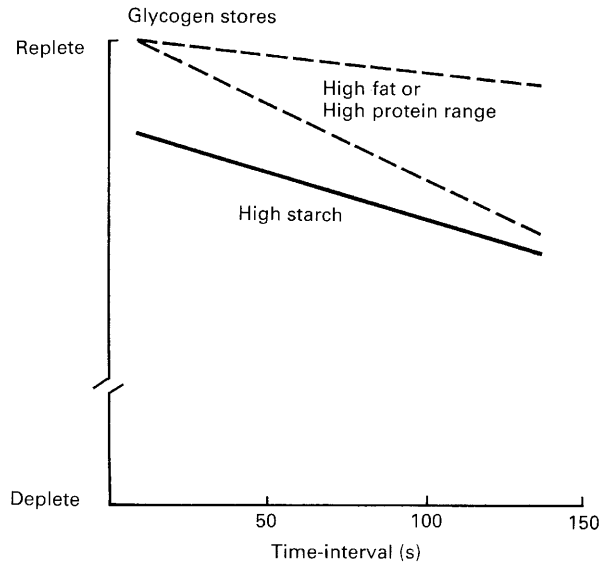


Figure 9.18 Generalized relationship between glycogen stores and time-interval during intense anaerobic work (>600 m/min, >190 beats/min) (Frape 1994).

Table 9.10 Effect of fat supplementation on blood plasma lactate and glucose concentrations during exercise at constant velocity (C) or at uncontrolled velocity (UC) and after post-exercise rest. Data averaged over the sources used for each comparison of carbohydrate control and added fat (Frape 1994).

Added dietary fat (g/kg)		Plasma lactate (mmol/L)		Plasma glucose (mmol/L)		Reference
		Work	Rest	Work	Rest	
0	C	2.30	1.98	5.42	5.32	Hambleton *et al.* 1980; Meyers *et al.* 1987, 1989;
100	C	1.89	1.47	4.93	5.23	Webb *et al.* 1987a; Worth *et al.* 1987
0	C	9.90	—	2.52	3.80	Pagan *et al.* 1987c
100	C	6.70	—	2.82	3.70	
0	UC	2.31	2.25	5.32	6.02	Hintz *et al.* 1978a; Harkins *et al.* 1992
100	UC	2.12	2.09	6.11	6.25	
0	UC	15.79	2.76*	6.57	5.29*	Webb *et al.* 1987a; Oldham *et al.* 1990
100	UC	18.61	1.87*	7.05	6.02*	Scott *et al.* 1992

*Data from Webb *et al.* 1987a only.

The effects of high protein on blood lactic acid can be more prominent than those of high fat (Pagan *et al.* 1987a,c). Observations in human subjects indicate that high protein/high fat diets increase the activity of exercised skeletal muscle LPL (cf. *adipose* tissue LPL activity), whereas the activity is unchanged with high carbohydrate diets (Jacobs 1981). Increased energy generation from fat oxidation with high fat and protein diets is due to the combined effects of increased muscle LPL hydrolysis of plasma TAG and increased use of plasma FFA (Figures 9.16 and 9.17), accounting for lower plasma lipids during aerobic SETs (Meyers *et al.* 1987). No certain conclusions can, however, be drawn from changes in venous blood FFA values (Frape 1993), but it is suggested that high-fat diets increase activity of muscle LPL (and possibly of TAG lipase), reciprocally reducing adipose tissue LPL activity, in contrast to the effects of starch (Figure 9.11). As plasma TAG concentration tends to decline with fat supplementation, the increase in muscle LPL activity would seem to exceed the reciprocal decrease in adipose tissue LPL activity.

A dietary increase in either protein or fat normally results in decreased dietary starch, reducing 'heating', anxiety, heart rate and excitability. Mixed fats contain lecithins, a component of which is choline. Holland *et al.* (1996) reported that vegetable oil, or especially oil enriched with additional soya lecithin, reduced the spontaneous activity and excitability of horses when the diet contained 100 g of this supplementary oil/kg. Choline is used in the synthesis of acetylcholine, a neurotransmitter found in parasympathetic nerve synapses and in voluntary nerves to skeletal muscles. The potential advantages and disadvantages of fat are shown in Table 9.11 (also see 'Polyunsaturated fatty acids', Chapter 5 and specific amino acids, later this chapter).

Table 9.11 Provisional conclusions on effects of high fat diets given to exercising and other horses compared with diets of normal fat concentration providing similar amounts of dietary fibre, protein and DE but more starch (Frape 1994, NRC 2007).

Advantages

(1) Lower RQ during submaximal exercise (promoting fat catabolism) potentially extending endurance and lower metabolic heat production.
(2) Possible decrease in adipose tissue LPL activity (EC 3.1.1.34) and increase in muscle LPL activity.
(3) Increase in muscle glycogen stores, but possibly no effect on rate of glycogen use.
(4) Glucose-sparing and sustained blood glucose concentrations during extended exercise.
(5) Possibly delayed lactic-acid accumulation during anaerobic exercise (but lactic-acid accumulation is proportional to the rate of glycogen expenditure, when other conditions are constant); mitigation of acidaemia.
(6) Reduction in gut fill, which can benefit work at >200 bpm, but which can compromise endurance.
(7) Reduced excitability of hot-blooded horses and a possible reduction in risks of colic and laminitis in all types of horse.
(8) Fats contain lecithins, a component of which is choline, a precursor of the neurotransmitter acetylcholine. Lecithin feeding is associated with reduced excitability.
(9) Fish oils, *n*-3 fatty acids cf. *n*-6 fatty acids, lower heart rate and blood cholesterol, improve exercise tolerance and seem to reduce measure of the inflammatory response.

Disadvantages

(1) In excess of 10 % of DM intake as dietary fat reduces fibre digestion. NRC (2007) recommends an upper limit for dietary fat of 0.7 g/day per kg BW.
(2) High cost of high-grade fat.
(3) Wide availability of poor-quality feed-grade fat and difficulty of assessing quality.
(4) Lack of stability of large fat supplements in mixed feed and practical problems of administration to the horse.
(5) Refusal of high-fat diets, or delay in acceptance of equivalent intakes, i.e. lower palatability.
(6) Lower large intestinal fluid reserves for endurance events.

Polyunsaturated fatty acids and 3-thiobarbituric acid reactive substances (TBARs)

Linoleic acid is an essential dietary polyunsaturated fatty acid (PUFA). Oils especially rich in PUFAs have no notable benefit for ponies given a diet deficient in PUFAs for seven months (Sallmann *et al.* 1992). However, chain length, or degree of unsaturation, may influence the exercise response (Pagan *et al.* 1993b). PUFAs in cell membranes are susceptible to attack, with the removal of an H atom with its electron, leaving a radical subject to attack by O_2, yielding peroxyl radicals. Chain reactions, the breakdown of the cell membranes and several products that include malonyldialdehyde (MDA), *n*-pentane and ethane are a consequence. MDA can be measured

colorimetrically with thiobarbituric acid (TBA). Strenuous exercise causes increased plasma thiobarbituric acid reactive substances (TBARs) and breath *n*-pentane per kg BW (McMeniman & Hintz 1992). However, the peroxidative stress of 3% corn oil was accommodated in exercising ponies given 42 iu vitamin E/kg dietary DM, through increased plasma glutathione peroxidase and superoxide dismutase activities and increased ascorbic acid concentration, despite higher muscle TBARs and regardless of vitamin E concentration.

n-3 fatty acids

More recently, evidence has been published for oils containing *n*-3 fatty acids and exercise. Exercise induced a decrease in erythrocyte membrane fluidity, associated with changes of blood oxidant/antioxidant balance in eventing horses given a (*ω*-3)-vitamin supplement. The (*ω*-3)-vitamin supplementation improved the oxidative balance poorly, but it delayed the exercise-induced decrease of erythrocyte membrane fluidity and increased the plasmatic *ω*3/*ω*6 fatty acid ratio (De Moffarts *et al.* 2007). Although vitamin E status decreased in both groups, GSH concentration decreased only in the control group. Post-exercise, Janiak *et al.* (2009), found an elevated plasma concentration of GSSG and a significantly lower GSH/GSSG ratio, indicating that the production of ROS can exceed the capacity of antioxidative defenses in immature trotters. During an incremental exercise test, horses that had received fish oil for 9 weeks had a lower heart rate and lower serum NEFA, glycerol and cholesterol concentrations compared with those receiving corn oil (O'Connor *et al.* 2004). O'Connor *et al.* (2004, 2007) compared the effects of fish oil with those of corn oil, each given at the rate of 324 mg/kg BW/day, in TB and Standardbred horses. The fish oil cf. corn oil, lowered the heart rate during exercise, lowered serum NEFA during the initial stages of exercise, lowered both serum glycerol and cholesterol and lowered plasma glucose during recovery from exercise, but plasma lactate was unaffected. These effects were associated with higher serum concentrations of EPA, DHA, C22:0, C22:1 and C22:5 fatty acids in the fish oil-fed horses. Overall, these horses had a decrease in serum concentrations of *n*-6 fatty acids, an increase of *n*-3 fatty acids and lower serum TAG than the corn oil-fed horses.

DIETARY PROTEIN REQUIREMENTS AND EXERCISE

A number of investigations have demonstrated that dietary protein concentration can influence running ability in horses. However, it is estimated that protein catabolism accounts for no more than 5–15% of the energy consumed during exercise, yet in both extended work (Rose *et al.* 1980b) and exercise in the post-absorptive state following high protein meals (Miller-Graber & Lawrence 1988), plasma urea is elevated.

Protein assimilation

Protein assimilation of tissue occurs during work following rest (Meyer 1987), but its scale is unclear. Johnson *et al.* (1988) detected no increase in N balance of working ponies. Patterson *et al.* (1985) found that 1.9 g digestible protein/kg $BW^{0.75}$, equivalent to 5.5% of dietary protein (maize soya protein), was adequate for intense exercise (cf. 7% and 8.5% dietary protein), as measured by plasma total protein, albumin and urea N. Orton *et al.* (1985a) trotted growing horses for 12 km daily at 12 km/h, on either a 12–14% or a 6–8% protein diet. Exercise increased the appetite, and consequently feed and protein intakes, of horses on the low protein diet, so as to equal that of those receiving more protein. The greater appetite provided protein, surplus to exercise requirements, which was utilized for growth. In a survey of racing TBs, Glade (1983a) found that protein intake (confounded with DE intake) was positively correlated with time to finish, implying that excess protein depressed speed. It is concluded that there is little justification for greatly increasing the daily protein intake of exercising horses to meet some putative increase in chronic requirement. On the other hand NRC (2007) estimates that daily protein requirements increase by 44% when exercise is increased from light to very heavy (Tables 2.2, 6.7).

The relationship between dietary protein and extreme performance is, however, far from clear. Despite the seemingly high NRC (2007) estimates, actual intakes are still higher. An excess of 56% over the estimates among racing Standardbreds (Gallagher *et al.* 1992a), and of 21% for racing TBs (Gallagher *et al.* 1992b) could reflect the natural protein content of palatable high-energy feeds. RQ was lower in horses given a high protein, cf. control, diet and exercised at high speed (Pagan *et al.* 1987b), implying a stimulation to protein or fat metabolism in the post-absorptive state (Figure 9.15), increasing urea yield (Frank *et al.* 1987) and water needs. Apart from this increased need, Hintz *et al.* (1980) observed no detrimental protein effect in horses during distance riding, in which dehydration causes fatigue. Miller-Graber & Lawrence (1988) recorded a higher plasma urea N, 16–19 h after an 18.5% cf. 12.9% protein meal during 15 min work at 170–180 beats/min; but plasma NH_3 rose to the same extent in both groups, jugular lactate concentration increased less, the

Table 9.12 Some metabolic responses of untrained, exercised horses to dietary protein intakes well in excess of N balance, compared with responses at approximately N balance with diets providing similar amounts of starch.

- Increase in blood urea concentration.
- Decrease in venous blood ammonia concentration after exercise.
- Decrease in RQ during aerobic exercise.
- In the range 240–600 m/min
  decrease in venous blood lactate concentration.
  decrease in hepatic lactate concentration.
  decrease in venous blood, lactate : pyruvate, ratio.

increase in plasma glutamine was marginally less and that of plasma alanine significantly less in the high protein group ($P < 0.05$). Others have also observed lower plasma lactic-acid concentrations during intense (Pagan *et al.* 1987b,c) and less intense (Frank *et al.* 1987) exercise with high protein diets (24.6% and 20%, respectively, *vs.* 14.6% and 10% protein, respectively), decreasing heart rate and glycogen catabolism at high speed (Pagan *et al.* 1987b). High protein reduced the post-exercise rise in plasma NH_3 only in untrained (Frank *et al.* 1987) and not trained (Miller-Graber & Lawrence 1988) horses. Observations in the fasting state have excluded an adverse effect of excess protein. Thus, Miller-Graber *et al.* (1991) performed the test 3 to 4 h after a meal, when the difference between 9% cf. 18.5% dietary protein was inconsequential for hepatic, or intramuscular, glycogen use, or for venous blood lactate concentration. Nevertheless, 5 min after exercise, venous blood lactate : pyruvate ratio was higher with the 9% diet, possibly indicating a higher pyruvate dehydrogenase (EC 1.2.4.1) activity, or decreased NADH : NAD ratio, with that diet.

It is concluded that high protein diets, above the need for N balance, confer some metabolic advantages to working horses (Table 9.12 and Figures 9.11, 9.15, 9.16 and 9.18). Nevertheless, excess protein is oxidized, resulting in the production of urea, heat and acid. Graham-Thiers *et al.* (2001) compared diets containing 7.5% and 12% protein, but with a similar DCAD (meq/kg, Na + K-Cl-S). At rest the higher protein diet led to a lower blood pH, owing to a difference in SID ($Na^+ + K^+$-Cl^--lactate$^-$). Both exercise and a higher protein diet can contribute to acidosis, so such a diet can compromise acid–base balance during exercise, in addition to creating additional waste heat. Moreover, increased urea production in the stable could increase environmental ammonia, contributing to respiratory stress. (It is of interest that glutamine drinks, taken after exercise by long-distance human athletes, reduce the frequency of respiratory infections. The reason seems to be that, during extreme exertion, glutamine sources are depleted and glutamine is an essential fuel for the functioning of the immune system – the equine possibilities have not been examined, to the author's knowledge.)

Specific amino acids

A 7.5% protein diet supplemented with 5 g/kg lysine and 3 g/kg threonine, cf. a 14% protein diet (low- and high-protein diets containing 6.1 g and 6.9 g L-lysine and 5.2 g and 5.9 g L-threonine per kg, respectively) led to lower plasma and urinary urea and uric acid, similar plasma albumin levels and a moderated acid–base response to sprints when compared with the effects of the high diet in exercising Arabians (Graham-Thiers *et al.* 1999, 2000). Higher plasma creatinine concentrations with the lower protein diet indicated to the authors that the amino acid supplement was supporting additional muscling. Valine and isoleucine are glycogenic and readily oxidized to provide energy. Supplements, including these branched-chain amino acids, have resulted in a lower plasma lactate accumulation and lower heart rates, when given 30 min before exercise, but have not improved performance over 1600 m in Standardbreds (Casini *et al.* 2000). After short intense exercise in trotters the plasma concentrations of isoleucine, leucine, alanine, aspartate, glutamate, lysine and taurine increased, those of arginine, asparagine, citrulline, glutamine, glycine, histidine, methionine, serine, tryptophan and 3-methylhistidine decreased (Hackl *et al.* 2009), while those of valine, ornithine, threonine, tyrosine and phenylalanine remained constant. Intense exercise markedly decreased tryptophan and increased taurine concentrations. These changes could reflect shifts between the free amino acid compartments, but indicate muscle catabolism. An amino acid mixture given orally to horses within 1 h of exercise increased the intramuscular amino acid pool (van den Hoven *et al.* 2009). Amino acid supplementation of sporting horses could be of significance for maintaining muscle integrity and for improving post-exercise recovery, without having a direct influence on performance.

FEEDING METHODS

Feed sequence, protein utilization and plasma amino acids

The true digestibility of protein in the equine small intestine ranges from 45% to 80%. At high rates of protein

intake, more will be degraded to NH_3 in the large intestine. Utilization of this by gut bacteria is 80–100% (Potter *et al*. 1992c). Excessive protein intakes must inevitably increase the burden of unusable N, either in the form of inorganic N or as relatively unusable bacterial protein. This burden is influenced by feeding sequence. The provision of a concentrate feed 2 h after roughage, cf. simultaneous feeding, caused higher levels of plasma free, and particularly essential, amino acids, 6 and 9 h later, respectively (Cabrera *et al.* 1992), indicating improved nutritional value derived from delaying the concentrate. Plasma urea did not rise with this dissociated feeding, but it rose continuously for the 9 h after mixed feeding, implying there was a large caecal flow of digesta. Belko *et al*. (1986) found that the thermal effect of food in exercising men increased with the protein content 150–270 min postprandially. No similar measurements of heat production in horses are available, although Frank *et al*. (1987) measured no difference in heart rate, or body temperature, between horses trained on diets containing 10% and 20% protein. The feeding sequence referred to above should influence heat production, as deamination and urea synthesis are associated with additional waste heat. The optimal amount and preferred feeding practice for dietary protein are thus not established, but optima for both might exist in exercise. The French evidence (Cabrera *et al.* 1992) indicates that the normal feeding sequence should be reversed. Improved husbandry allows the achievement of maximal effects without a burden of amino acid degradation products. Deamination of AMP is clearly prominent during brief maximal exercise (Miller-Graber *et al.* 1987) (Figure 9.11). Alanine is the principal vehicle for shuttling NH_3 from muscles to liver, but whether this is stimulated by extra protein given during rest has not been established.

Processing of cereals and precaecal digestion

The extent to which cereal starch provides glucose, or VFAs, depends upon its precaecal and even its pre-ileal digestibility. Kienzle *et al.* (1992) reported that the pre-ileal digestibility of oat starch was higher than that of maize starch, with similar degrees of processing. Grinding of whole grain led to high pre-ileal digestibility (%) for oats of 98.1% and for maize 70.6%, while rolling, or breaking, had little effect (whole oats 83.5%, rolled oats 85.2%, whole maize 28.95%, broken maize 29.9%). Starch gelatinization enhances its small-intestinal digestion, but at moderate, or high, rates of intake only. At low rates (<0.4% BW per meal) most sources of starch are digested in the small intestine (Potter *et al.* 1992a). Thus, end products, gut fill, and possibly the optimum time interval between feeding and exercise, are all influenced. A change in the proportions of fatty acids to glucose in the end products, which can be influenced by processing, could modulate exercise performance (Frape 1994).

'Hotting-up', the heat increment of feed

In Chapter 6 the phenomenon of waste heat generated during the digestion and metabolism of feeds was outlined and a mechanism for the 'hotting-up' effects of certain feeds was proposed earlier in this chapter. There is a reluctance to use energy-rich cereals such as maize and barley because of the alleged risks in this connection. However, the explanation here makes clear that where alternative feeds are rationed at rates that provide the same amounts of net energy, then energy-rich cereals will generate *less* rather than *more total* heat over a period of 12 h, or more. Measurements in polo ponies maintained at a constant body weight support the conclusion that energy-rich feeds do not necessarily exacerbate a heating effect of feed, but body temperature and metabolic rate increase after feeding (Figure 9.19). The ponies received equal amounts of net energy from maize and lucerne hay, or oats and timothy hay. No significant differences in response owing to diet, either before or after exercise, were noted (Wiltsie & Hintz in Hintz 1983). If anything, the maize-fed animals were less 'hotted-up'. Energy-rich feeds possess other advantages for sprint horses – for example, causing less gut-fill. Where unnecessary problems of 'hotting-up' have arisen, it is partly the consequence of a lack of appreciation of the differences between cereal grains in their energy content and bulk density described in Chapter 5 and quantified in Table 5.5. The cooking of cereal starch reduces extended 'hotting-up' by promoting digestion and thereby decreasing microbial fermentation.

In contrast to total heat production, the rate of fermentation, and of heat evolved, from indigestible and 'spill-over' starch is more rapid than that from fibre, and an increase in metabolic rate is caused by the greater peak blood glucose level achieved by digestible cereal starch cf. roughage. This also increases the rate of heat production. This increase in metabolic heat production rate is the principal cause of the heat increment of food in humans and it is greater with foods yielding large glucose and insulin responses and an enhanced glucose storage as glycogen. These rates can be controlled by the amounts of feed and the way this feed is presented (see Chapters 5 and 6). This elevated 'hotting-up' is of shorter duration than that of the total heat increment attributable to a feed and its effects

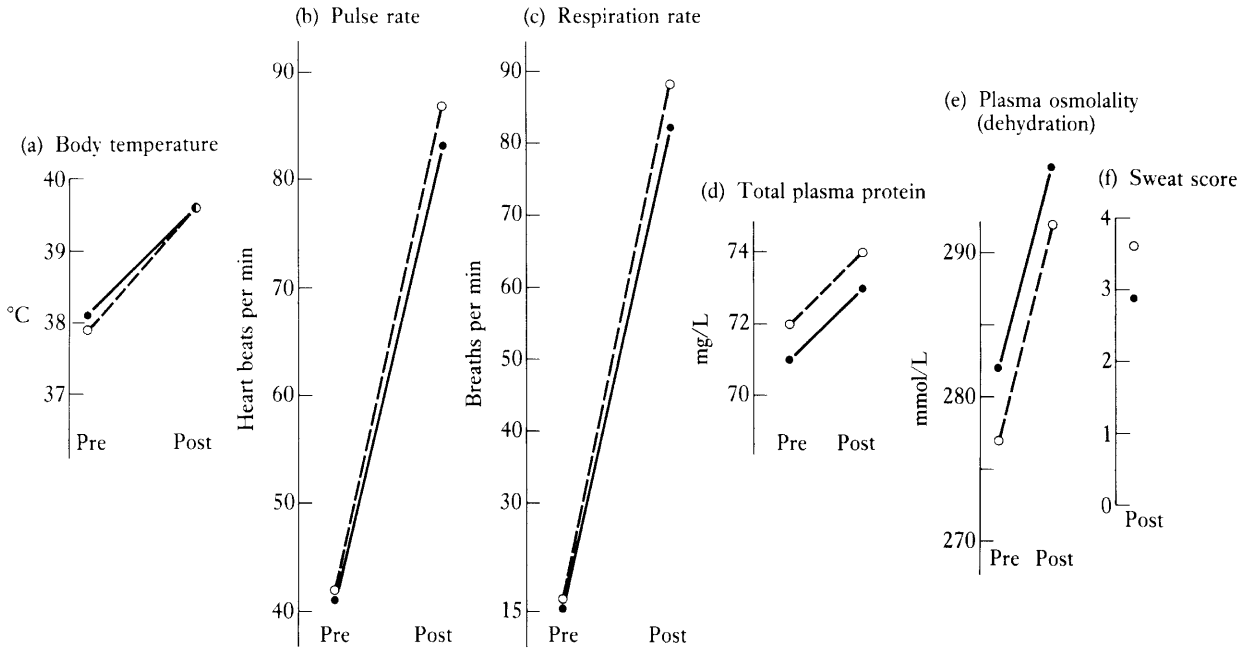


Figure 9.19 Pre-exercise and post-exercise values for polo ponies given 6.8 kg lucerne hay plus 3.2 kg maize (•) or 8.2 kg timothy hay plus 4.0 kg oats (○) continuously over 4-week periods of a reversal experiment to maintain constant body weight (Wiltsie & Hintz in Hintz 1983).

should have passed before horses are subjected to strenuous effort, say 5 h later, following small feeds.

Gut fill and speed

Processing of roughage can accelerate rate of passage and reduce gut fill, but decrease hind-gut utilization (Wolter *et al.* 1975, 1977, 1978). The extent to which feed is fermented will influence the weight of ingesta. Slade (1987) measured the speed of Quarter Horses galloping at up to 19.6 m/s (44 mph) over 137 or 229 m from a running start. Speeds differed according to digestibility of feeds, as reflected by differences in gut fill.

Feeding before endurance rides

Meyer (1987) concluded that endurance horses (cf. sprint horses) should be fed larger amounts of roughage (6–8 kg/day) to dilate the large intestinal volume, increasing water and electrolyte reserves. Poor-quality roughage should be avoided, as it causes a higher post-exercise plasma lactate concentration and more extreme hypoglycaemia than found with good-quality roughage. However, Warren *et al*. (2001) provided an afternoon feed, either high in fibre (54% NDF, 31% ADF), with a 15% greater GI-tract volume, or low in fibre (31% NDF, 19% ADF). The morning feed was withheld and subsequent exercise with dehydration caused a greater loss of ECF with the high-fibre feed. However, the decline in plasma volume was similar for both, indicating to the authors that the extra GI-tract fluid with the high-fibre feed had replaced circulatory losses more effectively.

The maximum postprandial increase in caecal volume is 8–14 kg/kg DM ingested, depending on the fibre content, with 130–135 mmol Na/L flow from the ileum. The average Na content of the large intestine is 40 mmol/L. Meyer (1987) concluded from previous evidence (Meyer *et al.* 1982a) that the horse should be fed at least 5 h before an endurance race, depending on the feeding sequence (see under 'Feed sequence'), as most of the residue will then have passed the ileocaecal orifice. Meyer (1987) compared two rations: one containing 2 kg concentrates and 3 kg hay, providing 11.5 g Na and 80 g K, and the other containing 2 kg oats, providing 1 g Na and 10 g K. Four hours postprandially the retention of water, Na and K was 5.8 kg, 9.3 g and 48 g for the first ration and 0.6 kg, 0.2 g and 1 g for the second, respectively. In support of Meyer, Ralston (1988) found that horses failing to complete 160 km

races had received mixtures containing less hay and more grain, and had been trained for greater distances each week (83 cf. 61 km). The energy intake per km trained was less in those that failed. Some samples of roughage contain excessive K which can stimulate diuresis, and so loss of water.

Glucose solutions given before prolonged exercise may increase glycogen degradation rate, through decreased fatty-acid mobilization, induced by the antilipolytic effect of elevated plasma insulin. Glucose given during hard exercise does not stimulate insulin secretion, but hyperinsulinaemia is protracted in TBs following a meal (Stull *et al.* 1987; Frape 1989), the timing of which is critical before extended exercise. Ponies given a fluid providing 5.4 MJ DE following exercise daily exhibited lower heart rates and blood lactate concentrations during and following subsequent exercise (Lindner *et al.* 1991). Clearly, some high quality roughage given 5 h before endurance exercise is beneficial. Moreover, performance was shown to be greater amongst those possessing a high glutathione reductase capacity and/or a lower plasma TBAR concentration, indicating that redox status modulates performance (Gondin *et al.* 2009).

Table 9.13 Factors influencing optimum interval between last meal and subsequent exercise.

- Weight of ingesta
  Size of meal
  Rate of passage
    proportion of roughage
    degree of grinding of roughage
- Dietary cation : anion balance
  ≈250 mEq/kg 3–4 h before sprint
- Potential for yielding glucose or VFA
  Digestibility of starch
    insulinaemia
    hind-gut volume and fluid/electrolyte retention
  Fermentability of fibre
    Hind-gut volume and fluid/electrolyte retention

Conclusions

- Type of meal: more roughage for aerobic exercise than for anaerobic.
- Optimum interval: sprint 3–7 h, extended aerobic exercise 5–8 h.

Feeding before sprints

In contrast, for short races there is some justification for a light concentrate ration 4–5 h before the start, although timing is rather critical as the race should not coincide with elevated plasma insulin. A large meal would extend the time over which plasma insulin is elevated, and forage intake should be strictly limited. Rice *et al.* (2001) conditioned TB horses to 10.1 kg (ad libitum) or 4.3 kg (restricted) grass hay during the three days before intense exercise (feed and water were removed 4 h before exercise), and found that restriction reduced body weight by 2%. Mass-specific $\dot{V}O_2$ was higher and both accumulated O_2 deficit and peak plasma lactate were lower in the restricted group, implying lower fatigue. Hay feeding the night before a gallop also causes a pre-exercise increase in PCV, as fluid migrates to the intestinal lumen and heart rate is increased during a gallop (Pagan 1999).The factors influencing the optimum interval between a meal and exercise are summarized in Table 9.13.

Meeting the increase in energy needs

The build-up of feed and of training before races takes 8–12 weeks and, in the case of event horses, a typical build-up starts with 5 kg concentrated feed daily and finishes with 8–8.5 kg two months later. The ration should be distributed among four daily feeds, and the hay, one-third of which should be given in the morning feed and two-thirds in the evening, should be reduced to 3.5–4 kg/day for a horse of average size during the last part of training. (The optimum sequence of concentrate and hay in the last days before an event is reversed, see 'Feed sequence, protein utilization and plasma amino acids', this chapter.) The protracted and intensive training for dressage (Plate 9.2) places emphasis on mental attitude and alertness, and it is important to achieve an appropriate energy intake at each meal. Additionally, lecithin supplements may assist the mental composure of dressage horses (Holland *et al.* 1996).

Feed allowances and work intensity

In all cases, feed intake should be increased as work rate increases and concentrated feeds should be severely restricted if work rate is reduced for any reason, whether this be of short or longer duration. On rest days, a horse that would normally receive 8 kg concentrates should then receive a maximum of 4 kg distributed in three feeds, but with a greater allowance of hay, up to 5–5.5 kg for the average-sized horse.

Either underfeeding or overfeeding leads to inferior performance. A horse should not be fed in order to fortify it with reserves for future events, but rather the rates of feeding should be consistent with immediate needs.

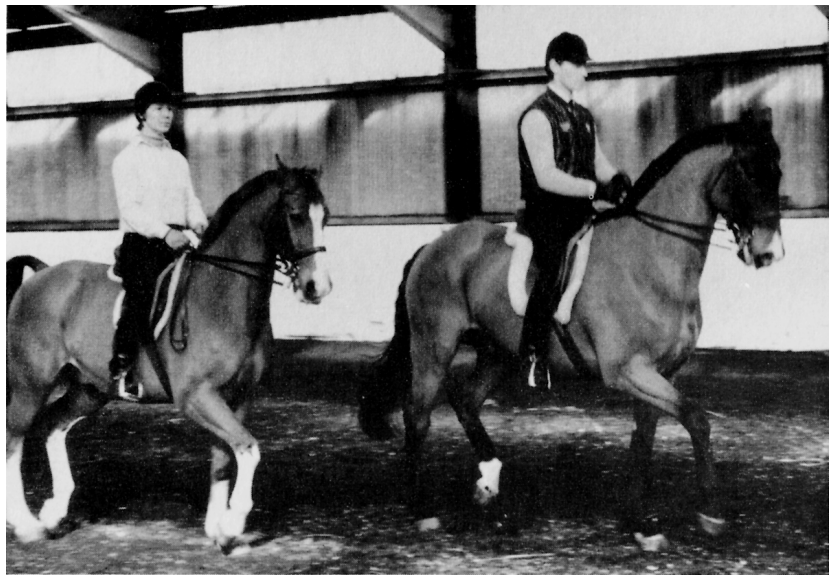


Plate 9.2 Wily Trout, a 16-year-old gelding, ridden by Chris Bartle and Pinocchio, a 15-year-old, ridden by Jane Bartle in 'Passage' during dressage training for the 1984 Olympic Games.

Overfeeding will create fatness, causing a greater burden on the heart, and interfere with the dissipation of heat. The synthesis of fat from excess carbohydrate, protein and dietary fat does not encourage those enzymes that participate in the breakdown of fat so necessary during work. Furthermore, overfeeding can lead to 'stocking up' (oedema) of the legs, hives (bumps under the skin), forms of colic, founder and exertion myopathy. Nevertheless, fat is the most abundant energy source in the body. McMiken (1983) estimated that a 500-kg horse with 5% body fat would have almost 10 times more energy stored in fat than in glycogen. The body fat content of mature horses in moderate condition can exceed 15 %, whereas in competitive endurance horses it was estimated to be <11%, in elite harness racing horses approximately 8% and in cutting horses 12% (NRC 2007).

Concentrated feed should be given at a minimum of three meals per day with some hay, ample water and salt supplements available, in the morning, at noon and with ample hay in the evening. Many horses in training do not take sufficient salt from licks, hampering progress, and a feed source providing 60 g daily is recommended.

Feed intakes in practice

Observations both in the US and the UK show that horses racing on the flat from two years of age, and weighing 470–530 kg, consume 13–18.5 kg total feed daily, which amounts to between 2.7 and 3.7% of BW (Mullen *et al.* 1979; Hintz & Meakim 1981; Glade 1983a; the author, unpublished data). Of this, concentrates with, for example, cereals, nuts, bran and linseed, amount to 30–60% of the ration. In one American study of 171 horses (Glade 1983a) concentrates provided 43–59% of the DE and 39–64% of the CP of the total ration. Moreover, the total ration provided 163 MJ DE/500 kg BW and 1686 g CP, 113% and 168%, respectively, of the NRC (2007) estimate for very heavy exercise. In the UK, daily rates of protein intake among both flat and National Hunt horses amount to between 1000 and 1400 g/day (the author's measurements, a range that just includes the NRC's 2007 estimate for very heavy exercise). These figures are well below the average of the American horses, largely as a result of the lower protein content of horse hays produced in the UK. Respondek *et al*. (2003) compared the feeding regimes of 29 French stables with the regimes published for similar stables in the US and Australia (Table 9.14). Far less hay is offered in Australia than in the other two countries.

Appendix B gives examples of dietary compositional errors encountered by the author in practice.

Transportation

The method and extent of the transport of horses before events could be critical to their subsequent performance. Van den Berg *et al*. (1998) showed that TB mares

Table 9.14 Mean daily concentrate and roughage intakes by Thoroughbred (TB), Standardbred (SB) and FrenchTrotter (FT) horses reported for commercial stables in France, Australia and the USA (Respondek *et al.* 2003).

	Total kg DM/day, per 100 kg BW	Concentrate kg DM/day	Hay kg DM/day
TB, 448 kg BW			
France, Respondek *et al.* (2003)	3.0	7.6	7.4
Australia, Southwood (1993a)	2.2	7.8	3.3
USA, Gallagher (1992)	2.4	5.6	6.6
SB or FT, 447 kg BW			
France, Respondek *et al.* (2003)	2.5	6.8	6.0
Australia, Southwood (1993a)	2.7	7.7	4.1
USA, Gallagher (1992)	3.2	5.1	9.3

transported 600 km over 8 h in South Africa ate but did not drink during transportation; potassium intake was decreased, so that water and electrolyte balances were affected. The stress of transport in mature horses over journeys of as little as 2 h in France (Goachet *et al.* 2003) and of 24 h in California (Stull *et al.* 2003) led variously to increases in white blood cell (WBC) count, neutrophil : lymphocyte ratio, blood cortisol, heart rate, rectal temperature, and faecal moisture content and weight. Plasma cortisol concentration increases in sport horses during a jumping competition, but trained experienced jumpers have lower basal concentrations of free triiodothyronine throughout competition than trained inexperienced jumpers, or than both inexperienced and experienced trained jumpers that have been transported before the competition. The concentration of the stallion's circulating thyroid hormones can be used to assess the effects of transport stress (Fazio *et al.* 2008, 2009). Stull *et al.* (2008) showed that the stress of 24 h continuous road transport was reduced by two 12-h periods of transport separated by off-loading, resting and feeding the horses for 12 h. Transport increased the horses' cortisol concentrations, neutrophil counts and neutrophil : lymphocyte ratios, and decreased numbers of all the lymphocyte subpopulation cell types. The inclusion of a 12-h rest-stop interrupted the transport-related decline in the lymphocyte subpopulations, allowing them to recover. This procedure might not only improve subsequent performance, but reduce transmissible disease risks.

STUDY QUESTIONS

(1) How would you feed a horse from 96 hours before (a) a sprint event, (b) a major dressage event or (c) a long-distance event?
(2) How should you manage a fatigued horse?
(3) How would you introduce fat-supplement feeding to a stable of working horses?

FURTHER READING

Eaton, M.D. Evans, D.L., Hodgson, D.R., & Rose, R.J. (1995b) Maximal accumulated oxygen deficit in Thoroughbred horses. *Journal of Applied Physiology*, **79**, 1564–8.

Williams, C.A., Gordon, M.E., Betros, C.L. & McKeever, K.H. (2008) Apoptosis and antioxidant status are influenced by age and exercise training in horses. *Journal of Animal Science*, **86**, 576–83.

10
Grassland and Pasture Management

... but that grass which grows on wet grounds, or the winter-grass, abounds with little or no spirit, wherein a great deal of the true nourishment consists, and therefore it must needs beget a viscid and indigested chyle, which must also render those horses that are fed with it sluggish, dull and unactive.

W. Gibson 1726

GRASSLAND TYPES

In humid temperate climates, natural succession favours the replacement of grassland by scrub and then woodland and forest. To sustain high-quality 'permanent' pasture requires perseverance in land management, through the grazing of domesticated animals and through the treatment of grassland as a crop to be cultivated. These pastures constitute the greater proportion of grazing and they contrast with the uncultivated areas of mountain, moorland, heath and downland, where wild grazing and browsing animals contribute to the distribution of plant species.

Fertility and grass species

The most fertile temperate pastures can theoretically support annually five, 500 kg barren or pregnant mares per ha from grazing and conserved forage. The most productive swards contain more than 30% perennial rye grass (*Lolium perenne*), a proportion of rough meadow grass (*Poa trivialis*), and the remainder of grasses consisting mainly of cocksfoot (*Dactylus glomerata*), timothy (*Phleum pratense*), other meadow grasses, Yorkshire fog (*Holcus lanatus*), species of bent grass (*Agrostis*) and fescue (*Festuca*). The proportion of white clover (*Trifolium repens*) depends very much on the use of nitrogenous fertilizers and the seasonal grazing pattern, but can amount to 25% of the cover.

Other broad-leaved plants vary in abundance according to management. An extensive survey of grasslands in England and Wales (Hopkins 1986) (Table 10.1) indicated that *Lolium perenne, Agrostis* spp. and *Holcus lanatus* were numerically the most important species, contributing 35%, 21% and 10% of the cover, respectively (of those swards exceeding 20 years of age the proportions were 22%, 27% and 14%, respectively). The palatable *L. perenne* decreased in proportion with time while the unpalatable *H. lanatus* increased. The fescues (*F. arundinacea, F. rubra*) are also highly palatable to horses and generally their persistence requires a lower fertility than does perennial rye grass. In England, potential stocking density of all grazing animals is generally correlated positively with the contribution perennial rye grass makes to the sward.

Poor drainage, low fertility and plant species

Where poor drainage has not been rectified, creeping bent (*Agrostis stolonifera*), Yorkshire fog, rough meadow grass and creeping buttercup (*Ranunculus repens*) thrive better than ryegrass. Other less-productive grasses which invade swards of this class in significant numbers include meadow foxtail (*Alopecurus pratensis*), couch (*Agropyron repens*), crested dog's-tail (*Cynosurus cristatus*) and barley grass (*Hordeum murinum* and *H. pratense*), together with red clover (*Trifolium pratense*) and bird's-foot trefoils (*Lotus* spp.). However, the decline in ground cover by sown species over the years occurs on all but the most fertile land, regardless of the excellence of drainage. About 20% of cover by sown species is lost after 5–8 years and a further 10% is lost during the next 4–12 years. Poorly drained soils provide a less suitable initial habitat for sown species and their proportion decreases throughout the pasture's life. Improvement by heavy treatment with fertilizers, drainage and intensive management yields swards of open texture, subject to poaching in wet weather. Lush pastures of this description, with little bottom, are unsuited for grazing by horses without great care and experience.

Table 10.1 Factors favouring species distribution in pasture* (after Hopkins 1986).

	Drainage	Soil nutrient status	Fertilizer N inputs	Grazing intensity	Hay cutting	Sward age	Elevation	Optimum pH
L. perenne	Good	good	high	hard	—	young	low	6–7
T. repens	Moderate to good	good	low	hard	no	younger	—	7–7.5
H. lanatus	Poor	low	low	moderate	yes	older	NS	5–6
F. rubra	Moderate	low	low	moderate	—	older	high	5–6
P. trivialis	Moderate	high	high	low	yes	—	—	6–6.5
Rumex spp.	Poor to moderate	fair	—	—	yes	—	—	—
Agrostis	Moderate	low	low	low	—	older	NS	5–6

*It should be recognized that these assessments relate to mixed swards, as pastures of single species only exist as such immediately after sowing.

NS, no significant correlation between elevation and frequency of the species.

In many river valleys, rushes and sedges appear in *Agrostis* pastures where drainage is impeded, or the land is otherwise neglected. The fertility is potentially quite high, but in the more degenerate and derelict land, even in lowland areas, purple moorgrass or flying bent (*Molinia caerulea*), bracken (*Pteridium aquilinum*) and gorse (*Ulex europaeus*), quite useless for horses, sometimes appear. On better-drained slopes of acid soils, between altitudes of 100 and 350 m (350–1100 ft) under annual rainfalls of 90–120 cm (35–45 in), fine-leaved fescues and bent grasses dominate pastures, with a scarcity of clovers in the latitudes of 50–57°N of maritime climates.

The specific distribution within these ranges depends on soil pH, latitude, aspect, soil drainage and grazing. These areas merge into the uncultivated rough and hill grazings in which there is the invasion of bracken fern and gorse at lower altitudes along with the fine-leaved fescues. On moorland, matgrass (*Nardus stricta*) and purple moor grass, rushes (*Juncus* spp.), heather (*Calluna vulgaris*) and bell heather (*Erica cinerea*) may occupy a larger proportion of the area. Fertilizers will generally encourage nutritious grass species, whereas excessive grazing by ponies of the better fine-leaved fescue and *Agrostis* areas lead to their suppression and the encroachment of economically useless *Nardus*, bracken, etc. In addition, where the pH is low, poor bone development occurs in young horses. The spread of shrubs and useless weeds largely depends on drainage, the extent of cutting and the presence or absence of cattle.

Poor upland grassland is generally considered to be of marginal value for horse production. Nevertheless, in France, upland pastures composed of *Nardus stricta, Festuca ovina* and including *Vaccinium myrtillus* (bilberry, blaeberry) are grazed successfully in summer by heavy breed mares (1.5 ha per mare and foal), during which time the mares gain in condition (Micol & Martin-Rosset 1995).

Herb strips

Herbs can be defined as broad-leaved plants with non-woody aerial parts and so could, of course, include clovers. Like clovers, many other herbs are rich in protein, minerals and trace elements relative to the common grasses; some are relished by horses and espoused by enthusiasts. The DM of nettle, for instance, contains nearly 6% of lime, 5% of potash and 2% of phosphoric acid, but the fresh plant is usually not sought by horses and ponies. A few relevant chemical values of herbs are given in Table 10.2. Herbs may be especially useful on marginal land, in which the upper layers are frequently leached of nutrients by excessive rainfall, and many tend to stay green in winter, so they provide a succulent winter bite, although their regrowth is protracted.

When herbs are present in abundance they depress total yield per ha of major nutrients, but this is less likely to be a critical issue in horse paddocks. In any case, their establishment in pasture as part of a normal grass and clover seed mixture is uncertain. Herb seeds are rather expensive, but many of them inevitably become established in permanent pastures through natural agencies. Herb strips are frequently sown on the headlands of fields. Table 10.3 gives suggested seed mixtures, which include some relatively noncompetitive grass species, although these are not essential. Chicory (*Cichorium intybus*) is a successful herb for strip-seeding in temperate pasture areas. It can reduce nutrient losses and yields DM rich in K, P, Ca, Mg and Na, although poor in N. However, the economic worth of herb mixtures is unproven for horses in any general way.

Table 10.2 Mineral contents (g/kg DM) of perennial rye grass and red clover at early maturity (Thomas *et al.* 1952; Worden *et al.* 1963) and of herb species (Hopkins *et al.* 1994) as means of four harvest dates from pasture sward.

N	P	K	Ca	Na	Mg	
Perennial rye grass						
Head	22.0	4.2	17.0	2.3	—	1.3
Leaf	21.0	3.2	23.0	8.7	—	1.7
Stem	8.0	2.7	17.0	3.0	—	0.9
Red clover						
Head	37.0	4.1	21.0	11.0	—	2.8
Leaf plus petioles	45.0	2.9	17.0	21.0	—	3.4
Stem	16.0	1.5	17.0	11.0	—	2.4
Chicory (*Cichorium intybus*)	26.2	5.4	26.1	19.0	12.2	3.5
Yarrow (*Achillea millefolium*)	26.4	5.8	33.2	12.1	13.0	2.5
Dandelion (*Taraxacum officinale*)	21.3	3.3	29.3	7.8	10.4	2.1
Ribwort plantain (*Plantago lanceolata*)	23.6	4.5	17.8	20.5	10.0	2.2
Grass/clover	24.2	3.5	19.3	9.9	3.9	1.6

Table 10.3 Suggested herb mixtures (kg/ha), including some grasses, for sowing as a strip 8–10 m wide in horse paddocks.

	Based on	
	Davies 1952	Archer 1978a*
Chicory (*Cichorium intybus*)	3	2.2
Ribwort plantain (*Plantago lanceolata*)	3	1.1
Burnet (*Sangiusorba minor*)	4	2.2
Yarrow (*Achillea millefolium*)	1	0.6
Cat's-ear (*Hypochoeris radicata*)	2	—
Dandelion (*Taraxacum officinale*)	—	0.3
Sheep's parsley, wild parsley (*Petroselinum crispum*)	1	0.6
Meadow fescue (*Festuca elatior*) or creeping red fescue (*F. rubra*)	3	(13)
Timothy (*Phleum pratense*) or smooth meadowgrass (*Poa pratensis*)	3	(7)
Crested dog's-tail (*Cynosurus cristatus*)	—	(7)
White clover (S100) (*Trifolium repens*)	3	—
Total	23	7 or 34

*Without the inclusion of grass seed the mixture should be introduced into an existing paddock by direct seeding if ground is well harrowed and the sward cut short.

Grass breeding

The Institute of Grassland and Environmental Research (IGER), in Aberystwyth, Wales, has crossed early- with late-flowering perennial rye grasses to produce a more even DM production through the season. Rye grass × fescue (*Lolium multiflorum* × *Festuca gigantea*) hybrids have been bred to increase pasture persistence and resist drought. An Italian rye grass containing 44% more Mg than standard Italian rye grass may be useful for hay production in studs. These ventures might have a value in

equine husbandry. Where grass is grown specifically for silage, a higher yield potential exists in some more exotic grass species, such as brome grasses. (For endophytes in seed production see 'Plant disease control', this chapter.)

PASTURE AS AN EXERCISE AREA

The production by pasture of digestible nutrients for horses and ponies is clearly of economic importance. Its critical role is starkly revealed by the historical evidence of the delay of European military campaigns until the spring flush in May, by armies dependent upon horses. A number of factors play a significant part in the selection of pastures, or their management, for horse husbandry. The thick cushion found in old pastures is better for exercise than is the open texture of heavily fertilized leys, in which numerous stones, upturned during ploughing, contribute to leg injuries.

Many horses needing rest and gentle exercise between periods of hard work, barren and pregnant mares and one-to-three-year-old growing stock are turned out to subsist on pasture. In each of these cases, leys or highly fertilized permanent pastures would initiate rapid and unwanted fat deposition. This creates unnecessary problems in the early stages of subsequent work, in late pregnancy and early lactation, or contributes to leg abnormalities in growing horses and to the incidence of laminitis and colic. Therefore, a high degree of skill is needed in evolving pastures for horses and ponies that provide useful grazing and also yield saleable and reliable stock. The thick, matted turf of well-drained old pasture resists poaching in wet weather and can, therefore, provide exercise and maintenance areas for out-wintered stock. However, not only is the total annual yield of digestible feed generally lower in these pastures, but, especially where drainage is poor, the season of herbage growth is shorter, a fact probably of much greater economic significance. The length of the grazing season is generally greater the higher the fertility of the land.

NUTRITIONAL PRODUCTIVITY OF PASTURE

Within any pasture, the nutritional quality varies from area to area. Therefore, the feeding value of the whole pasture will depend on the stocking density and the amount of the most attractive herbage at any one time. In temperate grassland areas – excluding acid land with very high rainfall – the protein content of pasture is directly correlated with rainfall and inversely with soil temperature during the growth period. Pastures grazed by horses in northern temperate latitudes tend to produce the greatest yield of DE and protein during May and June, after which there is a precipitous decline in productivity from July to August when grasses flower. Clovers and other legumes, if encouraged, prolong pasture growth and extend the summer grazing season.

Where persistent leafy strains of grasses have been established on fertile soils, this mid-summer fall in productivity is much less noticeable. More fertile deep soils are less inclined to dry out and the leafy strains of grasses continue vegetative growth much later into the summer. By grazing these pastures, the formation of seed heads is delayed or avoided, and tillering encouraged, so that their productivity is further enhanced.

Regrowth of succulent leafy material occurs in early autumn, but work with sheep indicates that the ME of autumn grass is utilized 40% less efficiently than that of spring grass of the same crude chemical composition. This poorer value should be recognized when foals are weaned in the late summer on pasture without supplementary feeding (see Chapter 7).

Minerals

Excepting horses confined to tropical grasses, a Ca deficiency is unlikely among grazing horses, even when the grazing and browsing are desiccated. In a parched terrain, horses and ponies are deprived, first, of water (Table 7.5), energy and protein and, second, of P. However, stock can become deficient in Ca, P and Mg if they are confined to wet acid soils covered by poor-quality, fine-leaved grasses. Many ponies coming off such hill land present signs of 'big-head' and other consequences of bone demineralization. Horses seem to be less prone to grass tetany caused by Mg deficiency than are cattle, but a fall in serum Mg is possible when lactating mares are grazing on low-Mg soils and it has been suggested that part of the effect is through excessive amounts of K in lush herbage. Leafy material contains far more K than the horse requires in normal circumstances. The needs for Na and Cl are likely to be met in horses dependent upon pasture in temperate latitudes.

Vitamins

Green leafy material is a rich source of folic acid, and comparisons made by the author (unpublished observations) between horses in training for flat racing, given a cereal-based diet supplemented with folic acid and vitamin B_{12}, and grazing in-foal and barren mares, foals and yearlings, indicated a 23% lower concentration of serum folate and a 33% lower concentration of serum vitamin B_{12} in the horses in training. Several other water-soluble vitamins are equally adequate in cereal-based and grazing diets.

There are normally large stores of vitamin A in the liver resulting from the consumption of green herbage rich in β-carotene, but after an extended drought there can be a clinical vitamin A deficiency as a result of protein and Zn deprivation, coupled with the scarcity of green herbage. It is unlikely that a deficiency of any of the other fat-soluble vitamins D, E and K would occur among horses confined entirely to pasture. However, a few isolated pasture species not found in the UK (see 'Vitamins D_2 and D_3', Chapter 4) can cause vitamin D toxicity, bone demineralization and soft-tissue calcification.

Trace elements

Australian evidence (Langlands & Cohen 1978) suggests that general pasture improvement increases the uptake by grazing animals of Cu, Zn, Mn, P, Ca and Mg; an excessive use of N fertilizers decreases the concentration of several trace elements in the sward, but with complex relationships (Burridge *et al.* 1983). Improvement in the drainage of waterlogged soils tends to increase Se and Zn availability, but reduce the availability of Fe, Mn, Co and Mo. The effect of drainage on Mo availability is advantageous, as peaty, poorly drained soils found in parts of Somerset and Ireland precipitate Cu-deficiency problems in ruminants through low soil availability of Cu and high availability of Mo, particularly where the soil pH is also high (see Chapter 3). These soils (pH in excess of 7.6–7.7) also tend to be deficient in available Mn and Co. Some soils contain more than 20 mg Mo/kg and an increase of Mo by 4 mg/kg depresses Cu availability to grazing ruminants by 50%. The effect is seasonal and an excessive uptake by plants of Mo and sulphate in the absence of generous amounts of available Cu leads to deficiency signs in cattle and sheep. Hypocupraemia in horses occurs less widely, but it exists in several parts of the UK and particularly in Ireland. On the other hand, the horse is much less susceptible to the effects of Mo and sulphate than cattle as, in ruminants, ruminal microorgansims synthesize thiomolybdate that reacts with Cu, decreasing its availability.

Soils subject to a high rainfall, waterlogging and a low soil pH are prone to Se-deficient herbage, as occurs in hill areas and on sands and gravels, in, for example, Newmarket, which is associated with low blood concentrations of Se in horses. By contrast, seleniferous soils containing very high levels of Se are a cause of toxic signs in grazing animals, for example on glacial lake deposits in Ireland. Shale, mudstone and clay soils contain higher concentrations of Se than chalk, limestone and sandstone soils (Thornton 1983) and many mountainous areas. Seleniferous soils are notorious in various regions of the world where accumulator plants store toxic amounts of soil Se. These accumulators leave Se residues which are apparently more readily absorbed by the roots of other plants, leading to alkali disease in grazing stock. (See selenium toxicity, Chapter 3)

Some inland continental areas, and even alkaline soils in central England, can induce signs of I deficiency in the young stock of grazing mares. When seaweed is used in excessive quantities as a source, signs of I toxicity, similar to those of deficiency, have been observed (see Plate 3.1). Deficiencies of Fe, Mn, Co and some other more exotic trace elements have not been recorded and are unlikely among grazing horses and ponies.

The correction of trace-element deficiencies by application of minerals to the soil is unsatisfactory for some elements, as the uptake is scant and repeated treatment is necessary. Better absorption is generally achieved with foliar sprays, but these are expensive and translocation is slight so that frequent treatment is unavoidable. Injections of Se have proved successful in grazing horses, but these are relatively expensive and repeated treatment at intervals is again necessary. When horses are held for extended periods on grazing lands, supplementary feeding with relatively concentrated sources of trace elements seems at present to be the most practical solution.

NUTRIENTS REQUIRED FOR PASTURE GROWTH AND DEVELOPMENT

Ultimately, nearly all life on the planet depends on sunlight and the fixing of atmospheric carbon by the action of chlorophyll, present in bodies called chromoplasts in seaweeds and chloroplasts in higher plants (see also 'Photosensitization' this chapter) (some deep-sea bacteria derive energy from the oxidation of Fe). Chlorophyll is green, although the colour is masked by other pigments in some species. Chlorophyll is somewhat similar to haemoglobin, but contains Mg in place of Fe. It absorbs red, orange and blue parts of the spectrum and uses this radiant energy to combine water with CO_2 in a reduction reaction, producing hexose sugar and oxygen, summarized as:

$$12H_2O + 6CO_2 \rightarrow C_6H_{12}O_6\ (\text{hexose}) + 6O_2 + 6H_2O$$

It is clear that not only light and water are required, but the process also needs warmth, so pasture plant growth accelerates to a maximum in mid-summer, when given adequate rainfall.

In addition to C, H and O, present as carbohydrates and fats, plant tissues contain a range of elements used in the

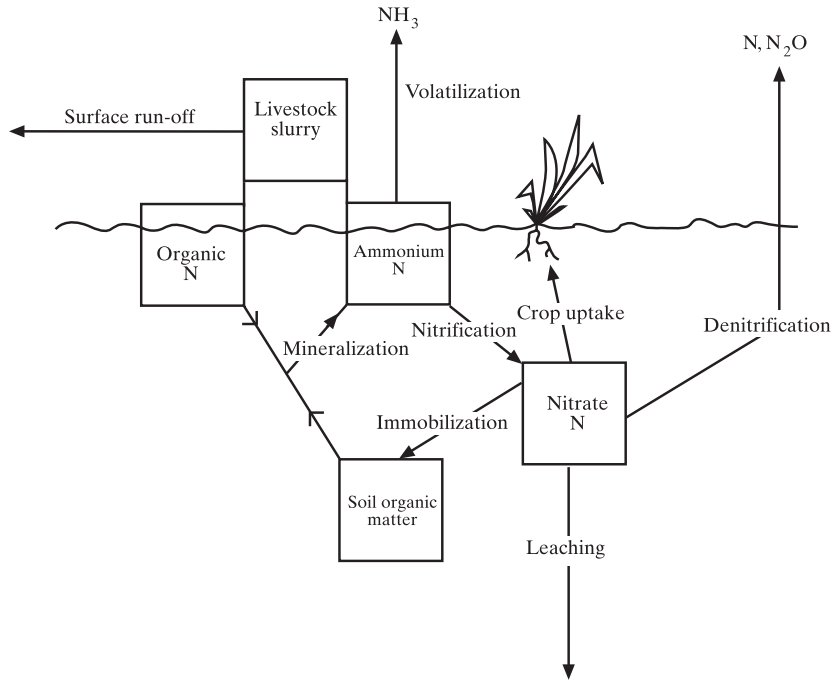


Figure 10.1 Fate of slurry N in the soil. N can be lost as a result of surface run-off, volatilization of ammonia, denitrification and leaching (Anon 1986).

synthetic process and present as components of proteins and many other tissue compounds. All these elements are present in many soils, but in most situations some are unavailable in optimum quantities for maximum plant growth, although in the height of summer water supply is frequently the limiting factor to growth. The critical elements are:

- typically N, P and K, as provided in chemical fertilizers;
- the other major elements – Ca, S and Mg; and
- essential minor elements including Fe, Mn, Cu, Co, B, Mo, and Zn, but also Na, Cl, Al and Si.

Leaching

Where fertilizers are applied to many soils, N passes into drainage water, when it is not absorbed by plant roots, or used in microbial growth. A comparable fate occurs with P, depending on soil pH, fixation and other factors. Concern exists over the pollution of streams, rivers and drinking-water sources with nitrates leached from the soil (Figure 10.1). It has been estimated that only 8–16% of N entering pastures leaves farms in the UK as meat or milk; rye-grass swards recover 65–90% of N applied, but much is then recycled in dung and urine and ultimately lost (Figure 10.2). Apart from nitrates leached out, gaseous N is lost as ammonia (NH_3), nitrogen (N_2) and nitrous oxide (N_2O), a potent greenhouse gas. Less N is lost when fertilizer is applied as ammonium sulphate than when it is applied as ammonium nitrate. Less still is lost when atmospheric N is fixed in root nodules of legumes by the bacterium *Rhizobium trifolii.*

In the West and in rapidly developing countries, for instance China, the overuse of P in fertilizers can cause serious eutrophication of lakes, ponds, rivers, streams etc. In the UK, almost all these show signs of eutrophication. In Sub-Saharan Africa the opposite problem – an acute shortage of P – is a more common occurrence. Eutrophication firstly reveals itself as excessive weed and algal growth, such as a thick layer of duckweed floating on the surface of the water, blocking out light, especially in slow moving canals and sheltered ponds. In extreme cases, there may be blooms of Cyanobacter, or blue-green algae, that can release powerful toxins, poisonous to land and aquatic animals and humans. Agriculture has been the dominant source of these polluting nutrients. Excess P is nearly always the main cause and P concentrations as small 10 to 25 parts per billion (ppb, 10^{-9}) in water can

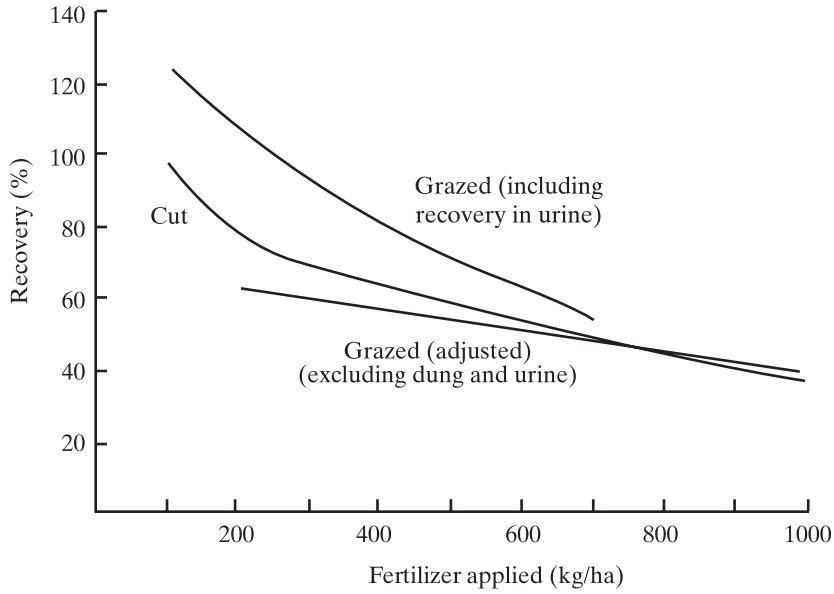


Figure 10.2 Apparent recovery of N from cut and grazed swards in National Grassland Manuring trial GM24 during 1982–84 in the UK and apparent recovery from grazed swards when N inputs are adjusted for returns in dung and urine.

trigger eutrophication. It is usually the 'limiting nutrient', meaning that even if N (usually as nitrate) enters water, it will only be used by aquatic plants for growth if sufficient P is already available (Brookes *et al*. 2010). (See also, water pollution, later this chapter.)

Chemical composition of herbage and equine health

Amounts of crude protein, soluble sugars and nitrogen-free extractives (NFEs) in the DM of herbage are highest during the period of rapid leaf growth in the spring, next highest during regrowth in the early autumn, lower during the period of flowering in mid-summer, and normally poorest during the winter when there is extensive dying back of the aerial parts of herbaceous plants. The months of the year when these phases occur in northern latitudes depend on the latitude, the lateness of the spring, rainfall, soil type and temperature. After the grazing of herbage, the first regrowth contains, per unit of DM, the highest protein, lowest CF and highest NFEs. These values change progressively as growth proceeds. For example, a study over 80 years ago (Fagan 1928) showed that, in Italian rye grass (*Lolium multiflorum*), from the second to the tenth weeks of growth, the CP composition of the aerial parts declines from 19% to 7%, the CF increases from 20% to 25% and the NFE increases from 44% to 60%. The changes can largely be explained by a rapid shift in the proportions of leaf to stem and leaf to flowering head (Tables 10.2 and 10.4).

The horse digests fibre less easily than domesticated ruminants so that shorter grass containing a higher proportion of leaf is a more valuable feed than herbage approaching maturity. Temperate grasses contain large amounts of water-soluble carbohydrates such as sucrose, fructose, glucose and fructans (oligo- and polyfructosyl sucrose). Fructans cannot be digested in the small intestine of mammals, but are rapidly utilized by microbial species in the large intestine. If large amounts of starch and/or fructans reach the hind-gut there is a rapid change in the microbial population, associated with the release of toxins and the onset of laminitis in susceptible horses. Moreover, cellulose fermentation *in vitro* with a pony faecal inoculum produces no lactate, whereas grass fermentation does, with high-fructan grass producing more lactate than low fructan (Longland & Murray 2003). It is important to establish the daily and seasonal variation in the fructan content of grasses. During one year in Wales, Longland *et al.* (1999) detected the highest content of fructans in leaf of *Lolium perenne*, var. Aurora and

Table 10.4 Effect of growth of timothy (*Phleum pratense*) on the ratio of leaf to stem and chemical composition (% DM) (Waite & Sastry 1949).

Sampling date	Leaf/stem	Crude protein		Ether extract		Ash		Crude fibre		NFE	
		Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem
20 May	2.57	21.7	14.1	3.8	2.9	7.1	9.9	19.1	23.5	48.3	49.6
2 June	1.30	17.2	11.4	4.7	2.5	6.5	7.9	23.8	29.7	47.8	48.5
16 June	0.39	18.5	7.6	4.1	2.6	8.0	6.6	26.1	32.6	43.3	50.6
30 June	0.35	12.3	4.4	3.3	1.7	8.8	5.0	26.9	31.7	48.7	57.2
14 July	0.20	11.1	3.4	3.2	1.3	9.0	5.0	30.6	32.4	46.1	57.9

Table 10.5 Mineral contents of grazed sward in stud paddocks at Newmarket, Suffolk (composition of DM) (data collected by author).

		Found	Normal range in UK
Calcium	(%)	0.34–1.6	0.3–1
Phosphorus	(%)	0.2–0.54	0.15–0.45
Potassium	(%)	1.5–2.5	1.6–2.6
Magnesium	(%)	0.12–0.2	0.11–0.27
Sodium	(%)	0.03–0.34	0.1–0.6
Sulphur	(%)	0.22–0.43	0.15–0.45
Molybdenum	(mg/kg)	0.9–2.8	0.1–5
Copper	(mg/kg)	4.5–12.3	2–15
Selenium	(mg/kg)	0.025–0.049	0.02–0.15
Zinc	(mg/kg)	21–34	12–40
Manganese	(mg/kg)	44–220	30–115
Cobalt	(mg/kg)	2.9–7.6	0.03–2

Perma from May to September at midday and during early afternoon (24–42% of DM mid-afternoon, but a considerably lower proportion the following year), whereas the highest content in the stems occurred in the evening (see also Chapter 11, 'Laminitis and other diseases of the hoof'). Short lush grass is, therefore, more valuable only when it is used knowledgeably. Table 10.5 gives some mineral values for herbage found by the author in stud paddocks at Newmarket. Although these values change with stage of growth, their digestibility is less affected by maturation than is that of energy.

White clover: its use and control and its relation to grazing intensity

Clovers tend to have deeper roots than most grasses, especially the less-productive grass species. Clovers draw moisture and minerals from lower horizons in well-drained soils, so that they remain green during summer drought when grasses have gone to seed, they cause less N-pollution of drainage water and they can rectify an imbalance of trace elements between the upper and lower horizons of the soil profile.

Research interest has refocused on making better use of white clover (or other legumes in non-temperate climates) in permanent pastures and leys. A white-clover sward can fix up to 200 kg N/ha annually, and use of this source can reduce N losses, mostly in winter, to a quarter of those resulting from equivalent fertilizer applications to a rye-grass sward. Although growth rate is slower where reliance is placed on clover N, this can benefit management of equine pastures.

Pastures on acid upland soils may carry no clover. White clover should then be sown as seed inoculated with *Rhizobia* of the appropriate strain, otherwise it will fail. In the UK, these strains are available from the IGER. Clover also fails through lack of available phosphate, or excess ionic aluminium (Al), fixing phosphate in acid soils. Liming at 3–5 tonnes/ha is therefore to be recommended (Table 10.6). Also slugs, weevils or eelworms (soil nematodes) cause damage.

It is advised that the clover content of pasture be held in balance with that of grasses. Clover growth in the UK reaches a maximum in July, when it can represent the dominant weight of leaf in the pasture. The growing points of clover plants are farther above the soil surface than are those of grasses. Grass shoots, or branch stems, grow as 'tillers' (stolons on the surface) and as rhizomes (subsurface) which originate at the nodes of the original stem, from which nodal leaves also originate. In perennial grasses, only a few of the stems are flowering stems, the remainder are vegetative stolons that, in various grass species, are above ground, at ground level, or, in fact, are rhizomes below the soil surface. Grazing, especially by

Table 10.6 Effect of lime in seed bed with an initial pH of 4.7 and superphosphate in seed bed annually or in alternate years on annual DM yield (tonnes/ha) over 3 years* (Sheldrick *et al.* 1990).

Lime (t/ha)	Total* superphosphate application (kg P/ha)				
	0	3 × 15	2 × 30	3 × 30	1 × 120
3	5.19	6.22	5.54	7.35	7.50
6	6.82	7.38	7.09	7.45	6.38

*15 kg annually, 30 kg in years 1 and 3, 30 kg annually, or 120 kg in seedbed.

sheep, will remove relatively more leaf and growing points from clovers than from grasses. Therefore, repeated grazing can reduce the proportion of clover in the sward should this be required. In contrast, cutting for silage, liming and phosphate fertilizer application will tend to promote clover and 100–120 kg N/ha, applied per silage cut, will create a predominance of grasses.

White clover can be introduced by strip seeding with a Hunter rotary strip seeder at the rate of 4 kg Huia or Menna seed/ha in early summer, following paraquat spraying at 0.4 kg active ingredient (ai)/ha. The clover should be allowed to become well established before grazing and that should be on a rotational basis so that the cover is not reduced below 4.5 cm height. Paraquat (1,1′-dimethyl-4,4′-bipyridinium ion) as the dichloride salt or dimethyl sulphate salt, is highly toxic to animals and has serious and irreversible delayed effects if absorbed, but as a herbicide it does not lead to food residues. One teaspoonful of the active ingredient is fatal up to 30 days after ingestion. The US EPA classified paraquat as a possible human carcinogen, but concluded that the risks to individual applicators are minimal, although paraquat is weakly genotoxic (EPA 1987). It is less toxic to birds than to mammals, and its toxicity to fish varies with the species and water hardness. Aquatic plants can concentrate high levels of paraquat, and tadpole mortality results when they feed on paraquat-contaminated plant material. Regulatory status: Austria, Finland and Sweden, banned; Hungary, severely restricted; Norway, voluntary cancellation; Germany, limited registration, field crop applications only once every four years; US, restricted-use pesticide (purchase and use only by certified applicators); Europe, under EC Registration Directive 91/414 the UK reviewed paraquat on behalf of Member States in 1997.

Fertilizers, soil fertility and pH

A soil pH of approximately 6.5–6.8 should be maintained, as below this range there will not be enough free Ca and much of the P will be fixed as ferric- or aluminium-phosphate. Above this range several minerals, including Fe, will be less available.

Phosphate fertilizer use (for pollution, see above)

Clover in particular benefits from P fertilizer application, with the adjustment of soil pH if the soil is definitely acid. Basic slag, as a slow-release alkaline P source, applied every four to five years, is now a rarity, owing to changes in the steel industry. Superphosphate is composed mainly of $Ca(H_2PO_4)_2 + CaSO_4.\ 2H_2O$ (18% P_2O_5, or 8% P) and is a readily available source of P not requiring to be as finely ground as the less-soluble slag and rock phosphate. It therefore has an immediate effect on P-deficient pastures and is valuable for application to seedbeds. Where superphosphate and lime are shown to stimulate clover growth the soil is likely to have been acid and deficient in available P, and where pastures are rich in legumes then in all probability the Ca and P contents of the soil are satisfactory.

A visual survey of the frequency of clovers and scarcity of fine-leaved grasses can be used as a ‘rule of thumb’ in predicting the well-being of the soil in these respects. Triple superphosphate (45% P_2O_5, or 20% P) in small amounts, i.e. 30 kg P/ha annually, or 120 kg/ha every 4 years (Table 10.6), with liming, should sustain clover presence on many mildly acid soils (pH 4.5–5.5). The amounts of pit chalk or limestone required on acid soils in order to raise the pH to within the desirable range and to ensure P availability will depend on the pH value and the texture of the soil, but amounts between 1.25 and 7.5 tonnes/ha can be used as a top dressing. In using limestone and rock phosphates it is critical that only finely ground material is purchased, as this characteristic will influence the availability of the Ca and P to the roots. Rock phosphates are not generally recommended for studs and are really suitable only on soils with a pH below 5. Limestone contains variable but useful amounts of Mg and Mn. Although limestone soils are rarely deficient in Mg, acid soils frequently are and dolomitic limestone provides a useful source of this element.

Assessing fertility and fertilizer requirement

When first embarking on grassland management for horses and at intervals of, say, every 10 years thereafter, it is desirable to carry out chemical determinations on the soil in order to assess, at the very least, its pH, P, K and Mg

status. Soil sampling must be carried out in a representative and sensible fashion, even within a field, so that distinctions can be drawn between clearly different soil types. Furthermore, the soil profile in old pastures can be such that the status is quite dissimilar in upper and lower layers reached by plant roots. Many soils show surface deficiencies of available P and Ca, accompanied by a lower pH, whereas lighter soils – especially where hay crops have been taken – are frequently K deficient in the upper layers. A full response to N fertilizers should not be anticipated if these primary deficiencies have not first been rectified.

Table 10.7 gives the indices used by the Agriculture Development and Advisory Service (ADAS) in the UK to classify grasslands in terms of their major nutrients. Where the value is 2 or over, no fertilizer treatment for that particular nutrient is required at the time of measurement. Recommended rates of P, K and N treatment of grassland are given in Table 10.8.

Where fields are set aside for hay, or silage, a generous use of fertilizers is worthwhile. K fertilizer may only be required when a hay crop is taken, and it can be in the form of sulphate or 'muriate of potash' (potassium chloride), or as part of a compound fertilizer. Soluble Mg fertilizers are rarely used, but can be applied to increase the mobile Mg content of pasture herbage. Where K is liberally used this generally lowers the mobile Mg content of herbage. As horse pastures are frequently depleted of N, with yellowing of the grasses, the rates can be fairly high. On the other hand, the excessive application of N may cause laminitis and the pollution of drainage water. Even so, in order to maintain the quality of the herbage and to get an economic output from the pasture, moderate applications of N fertilizers should always be considered desirable unless there is a reasonable proportion of well-distributed clover. A sward content of 25–35% of red or white clover yields the equivalent of 150–200 kg slowly released N/ha annually through N fixation. N application rates of 20–25 kg/ha, when required, three to six weeks before the pasture is grazed are recommended.

The composition of some straight fertilizers is given in Table 10.9. A 25-kg bag of compound fertilizer, N : P : K, 20 : 10 : 10 will contain 5 kg N and 2.5 kg each of P_2O_5 and K_2O. In all situations, granulated, rather than powdered, inorganic fertilizers should be used and at least a week should elapse after treatment before horses and ponies are allowed onto established pastures. This will give sufficient time for the granules to percolate down to soil level, avoiding the consumption of any significant amounts by grazing animals.

The principal organic fertilizer has been farmyard manure, preferably excluding horse manure, and its very approximate composition is given in Table 10.9. Its advantage over inorganic N sources is the slow release of N, but set against this is the expense of transport and distribution. It is probably more practical to distribute 50 tonnes/ha on a few paddocks than to use half as much on a much larger area, and there is some justification, particularly on light soils, for applications every 5–7 years. Fish-meal, now scarce, containing 10–11% N, is sometimes used in organic-based fertilizers. It has advantages, in terms of slow release, similar to those of farmyard manure.

Table 10.7 ADAS soil fertility scale (mg/L soil).

Index number	P	K	Mg
0	0–9	0–60	0–25
1	10–15	61–120	26–50
2	16–25	121–240	51–100
3	26–45	241–400	101–175
4	46–70	401–600	176–250
5	71–100	601–900	251–350

Table 10.8 Recommended rates of P, K and N treatment of grassland.

	For grazing (kg/ha) per year			For haymaking or silage – nutrients per cut (kg/ha)			
N, P or K index	P_2O_5	K_2O	N[1]	P_2O_5	K_2O	N High rainfall	Moderate[2] rainfall
0	60	60	20–50	60	80	60	80
1	30	30	20–50	40	60	60	80
2	20	20	20–50	30	40	60	80
>2	0	0	20–50	30	30	60	80

[1]The darker green the grass the less required. The larger quantities should be divided among three applications.
[2]45–65 cm (19–25 in) per year.

Where land has been ploughed up and new permanent pastures, or leys, are being sown, readily available nutrients should be provided for the seedlings; recommended rates of seedbed application are given in Table 10.10.

Table 10.9 Approximate composition (%) of straight fertilizers and farmyard manure.

Straight fertilizers		
Ammonium nitrate	N	34
Ammonium sulphate	N	20.6
Nitrochalk	N	15.5
Calcium cyanamide	N	20.6
Triple superphosphate	P_2O_5	45
Superphosphate	P_2O_5	18
Steamed bone flour	P_2O_5	29
Bone meal	P_2O_5	22
Guano	P_2O_5	13–27
Basic slag	P_2O_5	18–20 (45–50 lime)
'Muriate of potash' (KCl)	K_2O	60
Sulphate of potash with magnesia	K_2O	26 (5–6 Mg)
Kainit (Na, K, Mg)	K_2O	14
Kieserite ($MgSO_4$)	Mg	16
Calcined magnesite	Mg	60
Farmyard manure (kg/10 t)	N	15
	P_2O_5	20
	K_2O	40
	Mg	8

SWARD HEIGHT

A number of studies have investigated the effects of the intensity of grazing, or cutting, on the productivity of pastures. Horses and sheep graze more closely than cattle. Rye-grass and white-clover swards maintained at an average height of 3.6–3.8 cm with sheep have been shown to produce more DM annually than those maintained at 2.5 cm. With grazing cattle, similar unfertilized sward heights of 6 cm were shown at IGER to be more productive and to retain a greater presence of clover than swards maintained at 4.5 cm. The DM yields differed by 17% and 15% and clover presence differed by 4% and 23% in years 1 and2, respectively.

Reference was made to tillering (see 'White clover: its use and control and its relation to grazing intensity', above) and therefore to the spreading of grasses in a sward, as influenced by grazing. In contrast to the effect on clovers, grasses are, within limits, encouraged by grazing if adequate leaf remains, and consequently produce thousands more tillers per m^2 of sward (Table 10.11). Clovers also spread by producing thousands of stolons per m^2, but grazing causes a proportionately greater loss of leaf than in grasses.

It is clear that (1) repeated cutting for silage/hay leads to pastures that are less suitable for horse grazing, as there is a less firm mat of herbage for exercise, (2) close cutting in spring reduces subsequent grass seed production, and (3) repeated close grazing reduces the proportion of clover in the sward and the total annual yield of DM.

INTENSITY OF STOCKING WITH HORSES AND RUMINANTS

Horses should be removed from a pasture as soon as they have eaten the available herbage, if alternative land exists. They are more active than ruminants, and they can damage both the soil's structure in wet weather and the

Table 10.10 Recommended rates of nutrient application to seedbeds in establishing a pasture (kg/ha)

	N							
P or K index	Grass Spring sown	Autumn sown	Grass/clover (a)	Grass/clover (b)	Grass/lucerne	Lucerne	P_2O_5 (All mixtures)	K_2O (All mixtures)
0	125	50	40	75	50	25	100	125
1	100	50	0	50	25	0	75	75
2	75	0	0	0	0	0	50	30
>2	0	0	0	0	0	0	25	30

(a)If autumn sown or if it is intended to rely on clover as the main source of N for the sward.
(b)If little reliance is to be placed on clover as a source of N for the sward.

Table 10.11 Effect of maintained sward height on tillering and proportion of tillers that produce flowering heads mid-June in a Melle perennial rye-grass pasture in the UK (Treacher *et al.* 1986).

	Cutting date		Grazing height (cm)		
	Mid-April	Late summer	3	5	7
Tillers (1000 s/m^2)	24	11	35	29	24
Flowering tillers (%)			20	27	47

growing plants through prolonged trampling. In contrast to cattle and sheep, which spend periods ruminating, horses spend up to 60–70% of the day searching for the most delectable foliage (Shingu *et al.* 2000). DM intake/day per kg BW$^{0.75}$ and time spent grazing by horses on pasture are greater than they are for cattle (Arnold 1984; Menard *et al.* 2002). Overgrazing, poaching of the soil or stocking grassland during a heavy frost damages plants and depresses the rate of regrowth, encouraging the spread of prostrate and opportunist annual weeds seen particularly in an arc around gateways, drinkers and feed troughs. One of the few advantages of old, matted, sod-bound pastures is that they are less prone to damage in this way. However, the undergrazing of pastures tends to promote the nutritionally poorer grass species, partly through seeding, and parasite survival, as distinct from transmission, is extended.

The ideal stocking rate is no greater than that which will feed the horses in the growing season. It is preferable to stock few horses with the balance made up of cattle to clear the excess growth at the height of the season. Mixed stocking, either on a rotational basis or together with horses, initially decreases the number of horses that can be maintained, but these few will receive a better diet and will ingest fewer intestinal parasitic worm larvae. Moreover, the quality of the pasture can be maintained at a high level for many more years. By breaking up an area of land into paddocks, rotational grazing is facilitated and better parasite control is achieved, particularly where the grazing species are also rotated in each paddock. Occasionally, lactating mares or young stallions will bully cattle, so careful judgement should be exercised and rotation, rather than mixing of animal species, thus carries certain advantages.

Herbage yield and horse productivity

How does horse grazing affect pasture productivity and animal productivity? What degree of defoliation is optimum for horse productivity? Close cutting or grazing reduce yield, but as herbage leaves mature and die they obstruct light to the newly emerging, actively growing leaves. Moreover, the digestibility of the pasture crop changes radically with stage of growth, and differs between stem and leaf. A maximum rate of leaf growth and yield of digestible nutrients for horses over the year require that leaf is harvested at some intermediate stage and that dead herbage is removed.

Sound pasture management becomes increasingly important as the area available to each horse is reduced, when conditions become increasingly favourable to parasite transmission and bullying, and when excessive stocking depresses herbage regrowth (through removal of leaf and clover growing points; see 'White clover: its use and control and its relation to grazing intensity', this chapter). In the New Jersey summer, total grass density (number of tillers/m^2) was greatest with the highest stocking density, owing to the increase in density of tall fescue (*Festuca elatior*) at the highest stocking rate. The densities of all other species declined in summer compared with the spring (Singer *et al.* 2001, personal communication). Singer considered that their optimum stocking density was 0.6–0.8 ha per mature horse when Kentucky bluegrass (*Poa pratensis*) and annual bluegrass (*Poa annua*) represented 76% of the total grass density and common white clover (*Trifolium repens*) was the dominant legume.

There is a limit to the amount of trampling that grass can survive, particularly when soil is wet. The resulting bare ground becomes infested with docks, nettles and thistles. Thus, satisfactory husbandry is based upon:

- grazing paddocks just before the rate of grass-leaf growth declines, but well before senescence supervenes;
- allowing time for the grass to recover;
- replacing depleted nutrients;

Table 10.12 Productivity of Bermuda grass pastures over 56 days grazed by yearling Quarter Horses (Aiken *et al.* 1989).

Yearlings/ha	Herbage mass (kg DM/ha over 56 days)	Herbage allowance (kg DM/100 kg BW)	*In vitro* digestible DM (g/kg DM)	Weight gain per horse (kg/day)	Withers height increase (cm/56 days)
6.7	334	69	391	0.37	2.2
8.0	130	32	406	0.13	1.6
9.5	93	27	409	0.31	4.2
12.4	54	20	394	–0.31	2.0

- determining the optimum interval between grazings (which varies with season, rainfall, ambient temperature and soil fertility);
- applying an understanding of the life cycles of the critical pasture worm parasites of horses (see Chapter 11);
- understanding the temperament of each horse; and
- applying an understanding of each horse's environmental needs.

The horse digests forage less efficiently than do cattle. It therefore prefers, and obtains greater sustenance from, the younger leaves, but ample good pasture on its own can provide sufficient energy and protein for growth of horses over 5 to 6 months of age. Where the horse is forced by circumstances to graze less digestible parts of the herbage, some evidence indicates that it compensates by increasing its intake. This observation is not accepted generally. Experiments in the Netherlands (Smolders & Houbiers, personal communication) showed that grass cut at the haymaking stage, yielding 3700 kg DM/ha, was consumed daily by adult horses at the rate of 2.1 compared with 2.4 kg DM/100 kg BW of grass cut at the grazing stage, yielding 1900 kg DM/ha (these are equivalent to 100 and 113 g/kg metabolic weight, respectively). The younger grass had higher energy and protein contents and a lower CF content. An optimum was, however, found. The DM consumption rate of grass with a DM content below 14% was less than that of grass above 14% DM. Thus, apart from extremely young grass, there is a decrease in grass DM intake with increasing maturity of that grass. These Dutch horses obtained, as a percentage of their maintenance requirements, 170–200% energy and 460–500% digestible protein. Grace *et al.* (1998a) reported that TB filly yearlings gained 0.75 kg/day on a New Zealand pasture containing 201 g CP /kg DM and 11.4 MJ DE/kg DM. The DM intake was 6.5–7.5 kg/day (approximately 2 kg DM/100 kg BW) yielding an adequate daily DE intake of 81 MJ DE.

Any guidelines on pasture use must be very approximate as all pastures differ in productivity. The husbandry of growing horses has the objective of producing not only efficient weight gain, but also sound skeletal growth (see Chapter 8). These two objectives have a bearing on recommendations for stocking density. Aiken *et al.* (1989) measured the weight gain of Quarter Horse yearlings of 347 kg BW at stocking rates of 6.7–12.4/ha Bermudagrass (*Cynodon dactylon*) pasture. Weight gain per horse decreased in a complex way with increasing stocking rate. This probably related to the effect of grazing intensity on pasture yield of highly digestible herbage. Forage samples were vertically divided into top-, middle- and bottom-canopy layer thirds for analysis. The top third was that over 8 cm. Forage availability decreased in each of the three canopy layers as grazing pressure increased, but particularly in the least-dense, palatable top third. This third is the most photosynthetically active leaf tissue, the most productive, both for horse growth and, of course, for pasture growth. With increased stocking rates, grazing of the lower two thirds became unavoidable. As these layers contain stems and senescent plant tissue, animal performance was limited by the small proportion of leaf present. The data are summarized in Tables 10.12 and 10.13.

Stock-carrying capacity

Grazing pressures on pastures with continuous grazing should be set low enough to ensure sufficient regrowth of vegetation, but high enough to prevent accumulation of mature, poorly digested forage. As horses select the tender, top layers of forage, this situation argues for complementary use of ruminants which clear and utilize mature and senescent plant tissue.

When an allowance is made for loss of much of the edible foliage as a source of horse feed, but ignoring extremes of climatic fluctuations, the following carrying capacities have been estimated: 1 ha high-quality grassland should provide pasture and hay for 3–4 light horses of

Table 10.13 Productivity of Bermuda grass pastures in the top, middle and bottom layers of the canopy over 56 days when grazed by yearling Quarter Horses (Aiken *et al.* 1989).

	Forage availability (kg DM/100 kg BW)			Digestible DM* (g/kg DM)		
Yearlings/ha	Top	Middle	Bottom	Top	Middle	Bottom
6.7	10	21	38	421	403	383
8.0	4	11	17	442	425	404
9.5	2	11	14	413	405	392
12.4	1	8	11	414	400	391

*Measured *in vitro* by the method of Goering & Van Soest (1975).

about 400 kg, or 4–6 smaller ponies. Low-quality permanent pasture, however, might support only one horse per ha and, in the extreme, 25 ha dry range might be required to supply the needs of a single horse throughout the year. Average-quality grassland can produce sufficient growth for two horses or, with adequate moisture, when fertilized, for three horses per ha or, as summer pasture only, for double the number. On good temperate summer grassland in France, heavy-breed mares (700–800 kg BW) require 0.7–1.0 ha per mare and foal, or 1.5–2.0 ha when following cattle. With a rotational grazing system, the cutting of surplus herbage and N fertilization (80–150 kg N/ha), 2.0–2.5 growing horses can be grazed per ha. In contrast under harsh upland conditions in France only 0.5–0.7 growing horses per ha are possible (Micol & Martin-Rosset 1995). Many TB studs produce barely enough grass for one mare plus followers up to yearlings sales per 1–1.5 ha and then provide none of their own hay. The latter should not in any event be produced from paddocks that have been grazed by horses during the previous year at the least, if worm control is to be practiced assiduously.

Archer (1978a) found that only 10% of the area of long-established horse pastures was grazed. After ploughing and reseeding following an arable rotation, the grazing area was extended to 20–30%, which included the previously grazed areas. Horses will not graze near horse droppings and these areas are rejected if removal of the droppings is delayed for more than 25 h after they have been voided. This inborn habit reduces the transmission of parasitic worm larvae and leads to the establishment of both grazing and camping areas, with a consequent effect on productivity. Horse urine does not engender a similar instinctive reaction. Horses will, however, graze right up to cattle dung pats and graze evenly over areas after well-rotted cattle manure has been spread. Similarly, cattle will graze the longer grass around the horse dung pats. Ideally, therefore, horse droppings should be removed from pastures on a daily basis and not spread by harrowing. Although the latter practice will destroy more parasitic worms, it will enlarge the rejected area. The advantages of integrating ruminants with horse-pasture management are obvious. Steers are better than milking cows as they remove less Ca, P and N from the soil and both are probably better than sheep for which, nevertheless, there are some staunch advocates. Pastures heavily fertilized for dairy cows are generally unsuitable for horses.

Even with the best will and resolute adoption of mixed grazing, some areas of rank growth of low feeding value will remain, swamping any young basal growth. Such areas should be topped at least six times per season, with the toppings removed to avoid mounds of mouldy grass suppressing the underlying grass. The pursuance of this practice prevents weeds from seeding, promotes tillering and regrowth of young grass and will destroy some of the infective larvae.

All grazing animals thrive better if they have the companionship of other stock and this may be particularly true with highly strung hotblooded horses. Even goats, sheep, chickens, ducks or ponies can fulfil a useful role. The old adage ‘to get his goat’ implied that a favourite racehorse could be nobbled by stealing his mascot before a race. Gilbert White, in his *Natural History and Antiquities of Selborne* (1789), relates in a letter dated 15 August 1775 to Daines Barrington how a lone horse made an abiding companion of a solitary domestic hen, consoling and protecting it, in so far as it was possible, from the trials of avian life.

In addition to companionship, all horses should have access to shelter from the sun at high noon and from cold,

windy wet weather. This shelter is naturally formed by trees or by a simple three-sided covered structure.

Booth *et al.* (1998) assessed the effects of wetting on heat loss of mature Shetland pony stallions in winter coat. The ponies were wetted on the back with water at 5.26°C, housed at an ambient temperature of 2.0–9.5°C in an open-sided shed, and given meadow hay at maintenance levels. Heat production (HP) was measured by indirect calorimetry (see Chapter 6):

$$\text{HP (W/kg)} = \left\{\left(\dot{V}O_2 \times 270.5\right) + \left(\dot{V}CO_2 \times 82.7\right)\right\} / \text{live weight (kg)}$$

where W = watts, and $\dot{V}$ = volume at STP (Brouwer 1965). Skin temperature decreased, but not rectal temperature and HP was not increased over 3 h. The response would also depend on wind velocity and the nature of the shelter.

Critical temperatures

Chronic metabolic responses to temperatures of 1°C involve longer feeding periods, increased hair coat, decreased rectal temperature, decreased respiratory rate and slightly lower heart rates (Cymbaluk 1990; Cymbaluk & Christison 1993). Metabolic responses to acute heat exposure include increased rates of sweating and respiration, decreased feed intake and increased water intake (Morgan 1997; Marlin *et al.* 1999; Marlin *et al.* 2001).

The lower critical temperature for adult horses can be 5°C in mild climates or as low as −15°C in mature horses adapted to very cold outdoor temperatures (I am sure that, as I and many others have observed, acclimatized, well-fed, adult horses are quite happy in open-ended shelters with outside temperatures of <–30°C without any apparent distress).

Extending the grazing season and rejuvenation of pasture

A continuous supply of inorganic nutrients, including water, is required for the growth of pasture plants. In addition, roots require oxygen, so soil moisture must be present without waterlogging. For this reason, the structure, type, and humus content of soil are critical in ensuring a continuous supply of moisture at all levels through which the roots of pasture plants permeate. Cultivation to assist aeration and to extend the grazing season is brought about by relieving compaction and poaching of well-established pastures. Earthworms play an important role in soil aeration and cultivation is of most benefit in wet areas. Paraplowing, mole-ploughing or subsoiling may all improve water-table levels and increase early season yields.

It is not only economically important to extend the grazing season, but also ecologically desirable to reduce leaching of N from the soil, which occurs outside the normal season of growth. Mixed pasture species, including improved grass strains, assist in the achievement of this goal. Rye (*Secale cereale*), strip-seeded in autumn into permanent pasture, increases early-season growth and it is useful on light-textured free-draining soils in dry areas where N fertilizer use is restricted.

GRAZING BEHAVIOUR

There is a view that some environmental factors contribute to bad behaviour, even cribbing, among horses at pasture. What can be considered a poor-quality environment as seen through a horse's eyes, is learnt by experience and observation (Kanji Matsui *et al.* 2009). The proximity of other animals as company, ample palatable forage and some natural shelter from extreme conditions probably stand high in the estimation of most horses. A herd of horses quickly establishes a social order. Age and residency time have been identified as the main factors in the social rank of wild equids and of Icelandic horses, which is established by biting or kicking (Sigurjonsdottir *et al.* 2003; Pluhacek *et al.* 2006).

A crude comparison with the ruminant indicates that the horse has a small stomach and no rumen, necessitating short grazing sessions at relatively frequent intervals throughout the day. A study with mares allowed to graze during 12 h light and 12 h dark in North Carolina indicated that grazing took up 17.2 h daily, in which 89.7% of the daylight and 76.4% of the dark were occupied in the activity. Horses tend to graze at similar times, but generally without interference once a hierarchy has been established. Dominance hierarchy or patterns of agonistic and affiliative behaviour are apparent during feeding from a single source, when horses are in close proximity to each other.

The rate of intake of forage during grazing depends on the quality of herbage, its density and the appetite and size of the horse. Work by Cross *et al.* (1995) confirms that grazing endophyte-infected (*Acremonium coenophialum)* tall fescue (*Festuca elatior*) (see 'Poisonous plants', this chapter) depresses grass DM intake and body weight maintenance of horses. On the other hand, healthy tall fescue and creeping red fescue (*Festuca rubra*) are two of the most palatable grass species for horses.

Experiments in Kentucky (Cantillon & Jackson, personal communication) with Quarter Horse geldings showed that the consumption of Johnson tall fescue (*F. arundinacea*) or lucerne (*Medicago sativa*) over 7 h of

grazing averaged 5.5 kg organic DM, which was adequate for maintenance. There were 16–20 bites/min for grass species and 8 bites/min for alfalfa, and each bite took in 0.7 g organic DM grass, or 1.7 g organic DM alfalfa. However, Naujeck & Hill (2003) demonstrated that, at least with swards of Lolium perenne, at leaf heights of 3–19 cm, bite size was roughly proportional to sward height in which the horse removed 51–68% of the grass length at each bite. Bite rate was not reported, so whether or not consumption rate was proportional to height is not established. Horses generally prefer a sward height of 5 cm to one of 20 cm. However, Naujeck *et al.* (2005) reported that horses preferred perennial ryegrass swards of 15 cm height cf. <4.5 cm height. Some intermediate height is probably optimum – the governing factors seem to be stage of maturity and bite mass.

Soil ingestion

The ingestion of soil while grazing can occur in significant amounts, depending on the height of the herbage, the openness of the sward, the contamination of leaf by earth and on the species of animal. On rough terrain the soil intake by sheep is said to approach 20% of their daily intake of DM and measurements have shown that a 500 kg horse ingests as much as 1–2 kg soil daily while grazing. Apart from K, the mineral and trace-element contents of soil are generally higher than they are in herbage DM, although their availability, or digestibility, varies with the element and soil type. Most common mineral elements (possibly apart from I and Co) are required by growing plants. However, the proportions taken up by roots differ considerably from one element to another. Plants do not absorb heavy metal poisons such as Pb (cadmium is absorbed in greater quantities) in significant quantities, although the leaves can become contaminated by industrial fallout and the soil and subsoil can be polluted by industrial seepage. Soil consumption might then be a cause of, for example, Pb or F toxicity. In the author's experience, lead shotgun pellets admixed with grass silage destroyed a high-yielding dairy herd. However, high concentrations of toxic minerals in the soil may depend solely on their geological origin. The significance of seleniferous soils is discussed elsewhere in this chapter (see 'Trace elements', 'Toxic legumes and other species', Table 10.20; and Chapter 3).

SUPPLEMENTS ON PASTURE

The nutrient value of pasture varies particularly with:

- season
- climate
- plant species
- soil type
- fertilizers, and
- geological formation.

The prime effect of soil type is on the trace-element and mineral content of the plant. The Ca content can be influenced to some extent by liming, and that of trace elements by foliar sprays. Possibly the most economical way of overcoming pasture deficiencies of trace elements in particular, is by directly supplementing the horses. Supplementation carries with it the risk of harmful excesses of certain trace elements, unless horses are dosed individually. For many years, mineralized salt licks have been available. These licks are unsatisfactory as they can be washed away and some individuals 'hog' them, whilst others neglect them. Feed blocks are another form of supplementation.

Table 10.14 Declared composition of molassed feed block used by Murray (1993).

Ca (g/kg)	90
P (g/kg)	50
Mg (g/kg)	40
Na (g/kg)	80
Cu (g/kg)	5
Zn (g/kg)	4
I (g/kg)	0.1
Se (g/kg)	0.015
Mn (g/kg)	1.5
Co (g/kg)	0.1
Vitamin A (iu/kg)	333 000
Vitamin D_3 (iu/kg)	66 000
Vitamin E (iu/kg)	2 500

Feed blocks

Hardened and molassed mineral feed blocks containing all the important major and minor mineral elements on a cereal base have now been available for many years (Table 10.14). Blocks are hardened by pressure and binding agents, or by the use of chemical setting reactions. These reactions include the formation of calcium sucrosate (molasses combines with calcium oxide or hydroxide, but the product is said to restrict intake, owing to a bitter taste), or calcium sulphate (gypsum). The author's own experience with these blocks is that those that are well formed withstand quite wet weather, when they are held in appropriate containers, and that most horses at pasture feed from

them. Nevertheless, there is considerable individual variability in consumption with time and age of block. Feed blocks should be protected from contamination in a container, but that container should allow consumption by prehension as well as by licking – the horse should be able to bite the edges. Murray (1993) placed blocks in cut-down plastic barrels, stabilized in the lower half with concrete. The barrels measured approximately 0.7 m high by 0.4 m wide. The height had the advantage of allowing sheep to graze with the horses without the risk of Cu intoxication.

A detailed examination of the consumption of molassed feed blocks was undertaken by Murray (1993) in Ireland. The following were the main conclusions:

- Weather in Ireland did not influence intake.
- Weight of block remaining influenced intake. Below a residual weight of 4 kg, intake decreased markedly.
- Intakes of Ca, Mg and P from herbage and the block together were higher than that recommended by the NRC (1989), and the calculated intakes of the various trace elements were at all times below the maximum tolerance levels given by the NRC (1989).
- Plasma Ca and Mg concentrations did not reflect supplementation, whereas plasma and hair Cu concentrations increased with supplementation.
- Peak attendances at the block occurred early in the morning and at midday; the horse spending the longest there ate the most. Dominance hierarchy within a group influenced access to the block and consequently affected intake.
- Daily intake per horse ranged from 23–283 g, but averaged over a week the range was 32–215 g per day.

SAFETY OF GRAZING AREAS

Wooden fencing

The paddock principle of rotation allows a more complete recovery of the grass and prevents the excessive spread of flat weeds, such as plantain, daisy, buttercup and dandelion. All horse paddocks, structures and fences must be free from protruding nails, barbed wire, loose wire, tins, broken bottles, large stones, rabbit holes and anything that can ensnare horses, particularly where young stock are reared, as they tend to be more curious and flighty.

Creosote is now considered to be too risky for the treatment of fence posts, and good substitutes are available. These are effective when applied properly, normally allowing the posts to soak thoroughly. Any wood treatment is best carried out in the open, to avoid inhaling any fumes. The posts should be allowed to dry thoroughly before horses have access to them. Horses should have no access to containers of tar or other chemicals containing phenolic substances, as phenol is very poisonous to horses, and is rapidly absorbed through intact skin. Wooden railway sleepers cut lengthways make good posts. Wooden fencing is readily protected from chewing by running electric fencing wire along the top (Plate 10.1). The tops of fence posts should be flat, with no sharp edges and level with the horizontal poles nailed to their inner sides.

Electric fencing

In the past stud managers were reluctant to use electric fencing for controlling grazing. However, single-strand reflective tape is effective, as long as the current is maintained continuously. One study in North Carolina with TBs, Quarter Horses and yearlings showed that horses in groups of two or four, could be successfully rotated through 0.06 ha sections, back grazing the previous section. Occasional breakages of the fence occurred. The area was surrounded by three-wire electric fencing.

WATER SUPPLIES

A piped water supply should be clean and adequate (see Table 7.5) and, where natural watercourses are used, freedom from contamination for at least a few miles upstream should be confirmed (see Chapter 4 for effects of drought on the safety of natural water sources).

SILAGE AND HAYLAGE AND THEIR SAFETY

Silage quality is normally determined by the date or time of cutting. As grass grows during the spring and early summer, yield increases but digestibility falls. In terms of the ruminant D-value a decrease of 1 unit of D-value between 71 and 51 is equivalent to an increase in DM yield of perennial rye grass of 500 kg/ha, through delay in harvesting from the end of April to mid-June in the UK. The quality of forage legumes depends on the retention of leaf during harvesting. This is particularly true for lucerne, owing to the relatively high proportion of fibrous stem.

Silage should be made with safe raw materials free from weeds and contaminating pollutants. It should have undergone an adequate acetic acid fermentation and possess a high DM content without any evidence of moulding, which can occur in the spaces at the tops of silos where the conditions are hot and humid. Good-quality haylage is quite satisfactory and material compressed in plastic sacks with a mild fermentation and with 55–66% of DM is normally reasonably safe, but can be on the expensive side. (See Chapter 5 for haylage details.) To prepare haylage, stands of pure grass are cut at the bud stage and wilted for



Plate 10.1 Use of electric fencing installed on post-and-rail fencing to discourage wood chewing. However, the protruding posts and nails, especially on the top rail, present a hazard to horses. Evidence indicates that electric wire attached directly to the wood provides an adequate deterrent, as wood is a sufficient insulator.

18–24 h before baling tightly. Careful harvesting should minimize contamination of the herbage with earth. The baled product has a relatively high pH (about 5.5) and should not be fed to horses for at least a week after baling. Haylage from an unfamiliar source should be given in small quantities initially, even where the stock have previously received silage or haylage. Owing to its moisture content, its feeding value is about half that of high-quality nuts. However, its high content of soluble DM and ease of mastication facilitate a rapid intake of available energy; a few horses seem prone to gas colic if given inordinate amounts.

Badly made silage can be contaminated with pathogenic bacteria – *Listeria, Salmonella* spp. or *Clostridium botulinum* – any of which may cause problems. Some clostridial spores will inevitably be present on the ensiled crop. Horses and ponies, possibly through the absence of a rumen, are more susceptible than are ruminants to patho-

genic clostridial bacteria. The toxins of *Clostridium perfringens* invoke severe enteritis and enterotoxaemia; the neurotoxins of *C. botulinum* are particularly dangerous: they cause a descending paralysis commencing with tongue and pharyngeal paresis, with dysphagia, progressive paralysis, recumbency and eventual death. These botulinum bacteria multiply in the silage clamp or bale during abnormal fermentation. It is particularly important, therefore, that clostridial growth is entirely prevented in ensiled forage intended for horses. Wichert *et al.* (2008) examined the hygienic quality of roughage at 46 horse farms in Switzerland. The LPS contents were higher in straw than in hay samples, evidence supported by the macroscopic and microbial examinations. Eight straw samples and one hay sample were contaminated with deoxynivalenol. Silages had a high hygienic quality with a DM of 65.8% and a pH between 4.3 and 5.9.

Hanche-Olsen *et al.* (2008) examined horses with hindlimb digital bilateral extensor weakness in Norway. The common factor for affected horses seemed to be feeding big bale silage or, occasionally, hay of poor microbiologic quality. Clinical signs varied from intermittent knuckling of the hindlimbs to paraplegia. All horses euthanized had neuronal fibre degeneration involving sciatic nerves. The majority of horses with mild signs recovered after 5–6 months of rest.

The fermentation of sufficient water-soluble carbohydrate in silage brings about a fall in pH to 4.5, which will inhibit the growth of clostridia, where the DM content is >30%. Where the DM content is low a pH of 4.5 will not inhibit the proliferation of clostridia and secondary fermentation of the silage occurs, lactic acid is degraded to acetic and butyric acids, carbon dioxide is evolved with extensive deamination and decarboxylation of amino acids, yielding amines and ammonia. The resulting rise in pH activates the less acid-tolerant proteolytic clostridia. The ammonia they release brings about a further increase in pH. These changes are accompanied by a characteristic smell and loss of the attractive vinegary aroma.

The likelihood of clostridial proliferation is hampered by wilting the fresh material for 36–48 h before ensiling. In big-bale silage and haylage, DM contents of 50–66% are achieved. Satisfactory fermentation is facilitated in normal silage, with an adequate decline in pH, by harvesting forage containing not less than 2.4–2.5% of water-soluble carbohydrate in the fresh material. The National Institute of Agricultural Botany (NIAB) at Cambridge suggest 10% in the DM. In NIAB trials, where 100 kg N/ha was given in March, mean values in the first conservation cut at 67 D are given in Table 10.15.

Table 10.15 Soluble carbohydrate content of grass with a ruminant D-value of 67 (based on NIAB 1983–4a).

	Soluble carbohydrate (g/kg DM)
Italian ryegrass (*Lolium multiflorum*)	31
Hybrid ryegrass	26
Perennial ryegrass (intermediate and late) (*Lolium perenne*)	24
Perennial ryegrass (early)	23
Timothy (*Phleum pratense*)	14
Cocksfoot (*Dactylus glomerata*)	13

A proportion of rye grasses in any mixture obviously assists good fermentation and acid production during silage making. Less reliable material may have one or more of the following qualities:

- poor fermentation potential;
- moisture content is higher than desirable; or
- undesirable soil contamination.

If this is the case, or to avoid the growth of undesirable microorganisms and aerobic deterioration during 'feed-out', it may be necessary to add molasses to ensure the presence of adequate soluble carbohydrate. However, viscid liquids of this type are difficult to apply uniformly, so that additives are often used to achieve a pH of 4.2–4.6. Reliable additives are formic acid (2.3–3.0 L/1000 kg), which is corrosive, or cultures of *Lactobacillus plantarum* (4×10^6 cells/g fresh grass), which is more expensive. Both additives properly used can lead to a more rapid fall in pH, lower content of ammonia N and acetic acid, and higher content of water-soluble carbohydrate and lactic acid than untreated silage. Subsequent fermentation will reduce the pH further to a level that makes clostridial fermentation unlikely. If, however, a stable pH is not reached in silage with low DM content, then saccharolytic clostridia, which are present in the original crop as spores, will multiply and may initiate the secondary fermentation referred to above.

Big-bale silage/haylage, widely used in farming, and other plastic-wrapped silages have high DM content,

minimal fermentation, no additives and a pH of 5–6. They are subject to aerobic deterioration, owing to lower density than bunker silage and to the potential for punctures in plastic wrappings. They depend entirely on high osmotic pressure (low water activity) to inhibit clostridial proliferation and on the absence of tears in the plastic to prevent mould growth (see Chapter 5, Plate 5.2a–c). Deaths have occurred among horses consuming big-bale silage associated with the presence of clostridial organisms and/or toxins, without any contemporary effects on cattle. Great care should be exercised in selecting silage with the appropriate aroma, a high DM content and a pH of 4.5–5.5. These are essential prerequisites of silage, or haylage, used in horse feeding if health risks are to be minimized.

Dairy cattle given extensively fermented rye grass silage lost greater amounts of N in the urine compared with those given silage of restricted fermentation, or fresh grass. Extensive fermentation, cf. restricted fermentation, provided a higher proportion of propionate and a lower proportion of acetate in the VFA of the silage. Similar comparisons have not been undertaken with horses, although it is apparent that extensively fermented herbage, or that containing significant amounts of ammonia, or *n*-butyric acid, is not very palatable to horses. Practice and experience are necessary adjuncts to good recipes in the achievement of the optimum product. High DM (50–66% DM), fermented, plastic-wrapped herbage is gaining in popularity and is generally more palatable to horses than is silage with a lower DM. Adult horses tend to consume more DM from a high than a low DM silage/haylage.

Metallic contamination of silage

Acidity causes the solution of heavy metals that contaminate ensiled herbage. This is particularly important where pastures have been used for game or clay-pigeon shooting. Extremely high concentrations of lead, causing death, have been detected by the author in silage from fields over which clay-pigeon shooting has occurred. Dissolved lead is more toxic than metallic lead and horses may be seriously and permanently injured, or killed, by exposure of this kind. Other heavy metals might be similarly dissolved.

Maize silage (see also Chapter 8)

In various areas of France, and for many years, maize silage of 30–35% DM has been given to pregnant and lactating mares and to growing horses, as the main dietary constituent. Milk production is excellent and foal growth rate good. Supplemented with concentrates the silage is fairly well consumed by growing horses (Micol & Martin-Rosset 1995) and long experience has enabled breeders to use this material safely.

GRASSLAND IMPROVEMENT

Permanent pasture should be well drained and, where it is low lying, or the subsoil is heavy, consideration should be given to piped drainage and, in the appropriate areas, pumped low-level ditches. Improvement in drainage reduces poaching and allows the stocking density to be increased. By lowering the water table, plant roots are encouraged to permeate lower horizons of the soil so that they gain access to a greater variety of minerals and to moisture for longer rather than shorter periods during a drought. This extends the grazing season and encourages rye grass, cocksfoot, timothy and clover, discouraging rushes, tufted hair-grass or tussock grass (*Deschampsia caepitosa*), *Agrostis*, couch, Yorkshire fog and water-tolerant meadow grasses. Fertilizers, therefore, can be more effectively used, and the increased potential growth of the resulting sward decreases the concentration of parasitic worms. It also decreases the risk of liver fluke infection. Although horses have a pronounced resistance to liver fluke, they can become infested and their economic worth is thus prejudiced. Where mixed grazing is practiced, the sheep and cattle can, of course, be treated before they are introduced. The snail burden of the land is reduced by copper sulphate treatment or, for example, by grazing with ducks.

If piped drainage is impractical, mole drainage, or the subsoiling of impervious heavy land will improve its structure and, for a shorter period, bring about the advantages of more permanent drainage. Sometimes such marginal land is infested with quite inedible species, including cottongrass (*Eriophorum* spp.), rushes and, in better-drained acid areas, by thick mats of *Nardus* grass. Here, initial improvement can frequently be effected through burning and hard grazing with steers. Most poor-quality permanent pastures can be improved by occasionally discing or heavy harrowing to break up a surface mat. This allows aeration, encouragement of better grasses, and some control of moss (*Lycopodium* spp.), mouse-ear chickweed and buttercup. In dry areas, however, little disc penetration will occur unless there has been recent rain.

Chemical control of weeds

Although the cutting of pernicious weeds such as docks (*Rumex* spp.) and thistles before they seed is helpful, it must be done on a regular basis, five or six times a season, and even then control of creeping thistle is doubtful. The use of selective weedkillers based on (4-chloro-2-

methylphenoxy)acetic acid (methazone) and/or 2,4-dichlorophenoxyacetic acid (2,4-D) to control broad-leaved weeds without damage to mature grass has been recommended. MCPA is of low toxicity and 2,4-D of moderate toxicity to animals, and pastures properly treated with them have not caused fatalities, regardless of how soon they have been grazed. It should be borne in mind, however, that both are moderate health hazards to humans: methazone has an acute oral LD_{50} of 1046 mg/kg in rats; acute dermal LD_{50} > 2000 mg/kg in rabbits; acute inhalation LC_{50} > 2.64 mg/L in rats (4-h nose-only exposure), while 2,4-D has high acute neurotoxicity on oral and dermal exposure.

Creeping thistles, docks and, in some upland studs, bracken, encroach progressively on grazing areas. The Long Ashton Research Station at Bristol, UK, has achieved effective control of these species with low-dose sulphonyl-urea herbicides, although these are not entirely clover-safe.

Where small prostrate weeds, such as members of the buttercup family (Ranunculaceae), are widespread, spraying by tractor is most convenient, but where there are patches of creeping thistles, docks and nettles, a knapsack sprayer can be used to advantage: this causes less damage to clovers and other valuable broad-leaved herbs. Spraying should always be carried out according to the manufacturer's directions:

(1) when there is little wind;
(2) at a time of rapid weed growth, but before flowering;
(3) refraining in periods of drought but avoiding rain for a few hours after spraying; and
(4) not before newly sown pasture grasses have at least three leaves, but preferably not for 2 years.

MCPA is effective against common nettle, daisy, dandelion, spear thistle (*Cirsium vulgare*), annual scentless mayweed (*Tripleurospermum inodorum*), black bindweed (*Polygonum convolvulus*), redshank (*P. persicaria*), cleavers (*Galium aparine*), common chickweed (*Stellaria media*), charlock (*Sinapis arvenis*), fumitory (*Fumaria officinalis*), poppy (*Papaver rhoeas*), fat hen (*Chenopodium album*) and small nettle (*Urtica urens*). It will check broadleaved dock (*Rumex obtusifolius*), bulbous buttercup (*Ranunculus bulbosus*), creeping buttercup (*R. repens*), coltsfoot (*Tussilago farfara*), creeping thistle (*Carduus arvensis*), curled dock (*Rumex crispus*), field horsetail (*Equisetum arvense*), perennial sowthistle (*Sonchus arvensis*) and soft rush (*Juncus effusus*). The most satisfactory control is achieved by spraying bulbous buttercup and chickweed in autumn and ragwort in early May. Bracken fern is very resistant to spraying, but the most effective time is August. Pastures should be left for 15 days before they are grazed in order to allow adequate time for the penetration of the herbicide, and the dead residues of poisonous weeds should be removed from the pasture.

Poisonous plants

Weeds found in Europe that are poisonous to horses in both the growing and dried states are listed in Table 10.16 and those reported worldwide to be toxic to horses are listed in Table 10.17. After cutting and drying, or after being destroyed by sprays, weeds that retain their toxicity are more palatable than before and so should be removed and burned. Horses and ponies do not normally graze growing poisonous weeds, but young stock or other stock freshly introduced to a pasture, following a period in stables, might eat a variety of such plants. Furthermore, during times of drought, some that are deep-rooted or near watercourses and remain green entice the unwary horse. I was confronted with just such a situation in the Middle East, leading to hepatotoxicity, where the only significant amount of green herbage over a wide area was *Heliotropium europaeum* (Table 10.17).

Reseeding

If there is little pressure on available grazing land and the appropriate equipment is available, it can make economic sense to reseed worn-out pastures after ploughing, as long as the land is potentially fertile. No permanent improvement would, however, result unless drainage, ditching, liming, fencing and hedging are first put in order. If there are large areas of pernicious perennial weeds in the old turf, they should first be destroyed with herbicides and two to three weeks should elapse before ploughing begins. For spring sowing, autumn ploughing of the old turf is desirable. Sometimes it is necessary to use heavy discs or cultivators to break up the turf. Also, there is some justification for killing all the old turf by spraying with glyphosate, delaying ploughing for at least two weeks to ensure root kill. On very light soils it is occasionally possible to plough during the late winter and sow in March or April in the northern hemisphere, but it is essential to achieve consolidation of the ground and a fine tilth after ploughing.

A pasture-seed mixture should be sown as soon as a good seedbed can be effected, before the surface soil is subjected to periods of desiccation. In the UK, sowing

Table 10.16 European plants poisonous to horses.

Species	Common name	Location	Toxic parts and effects reported
Aconitum napellus	Monkshood	Shady stream sides	Whole plant, especially root, very potent
Agrostemma githago	Corncockle	Cultivated fields	Especially seeds, saponins
Anagallis spp.	Pimpernel	Sandy coastal areas, cultivated land	Whole plant
Aquilegia vulgaris	Columbine	Damp places, lime-rich woodlands, grassland	Probably whole plant, mild hydrogen cyanide
Arenaria spp.	Sandwort	Dry places	Whole plant, mild
Atropa bella-donna	Deadly nightshade	Lowland, hedge banks, edges of woods	Whole plant, especially berries
Bryonia dioica	White bryony	Lowland hedges	Red berries, vegetative parts, diarrhoea
Cannabis sativa	Hemp	Wasteland	Flowers, fruits, low toxicity, but horses at greater risk
Chaerophyllum temulum	Rough chervil	Road sides	Seeds and shoots
Chelidonium majus	Greater celandine	Shade, banks, walls	Stems
Cicuta virosa	Cowbane, water hemlock	Damp places	Whole plant, especially roots, very poisonous, including hay
Conium maculatum	Hemlock (smooth spotted stems)	Damp places	Whole plant, mature hay unlikely to be harmful
Cyclamen hederifolium, C. europaeum	Sowbread	Woods, banks, especially southern Europe	Whole plant
Daphne mezereum	Mezereon	Woodlands	Berries and bark
Datura stramonium	Thornapple	Waste lowland	Whole plant
Delphinium spp.	Larkspur	Cultivated land	Whole plant, potent
Digitalis purpurea	Foxglove	Hedgerows, wood margins	Whole plant
Equisetum spp.	Horsetail	Stream banks, damp woods	Whole plant, including hay, especially horses
Euonymus europaeus	Common spindletree	Hedgerows	Whole plant
Frangula alnus	Alder buckthorn	Woods, hedges	Leaves, bark, fruit, diarrhoea
Helleborus spp.	Hellebore	Lime-rich woodlands, scrub	Whole plant, potent

Table 10.16 *Continued*

Species	Common name	Location	Toxic parts and effects reported
Hyoscyamus niger	Henbane	Clearings, edges of woods	Whole plant
Hypericum spp.	St John's wort	Grasslands	Whole plant, photosensitivity
Iridaceae	Iris, narcissus, daffodil	Gardens	Bulb
Laburnum anagyroides	Laburnum	Gardens, self-sown	Whole tree
Lactuca virosa	Acrid lettuce	Road sides	Whole plant
Liliaceae *(Allium oleraceum, Endymion non-scriptus, Fritillaria meleagris, Convallaria majalis, Colchicum autumnale)*	Field garlic, bluebell, fritillary, lily of the valley, meadow saffron	Woods, meadows	Bulb
Linum catharticum	Purging (fairy) flax	Pastures, meadows, uplands	Seeds
Linum usitatissimum	Common flax, linseed	Escaped from cultivation, common	Seeds
Lolium temulentum	Darnel	Waste	Possibly fungal infection
Lonicera xylosteum	Honeysuckle	Woodlands	Berries and leaves
Lupinus spp.	Lupin	Light, dry soils	Whole plant, including hay, especially seeds
Mercurialis annua	Annual mercury	Clearings, edges of woods	Whole plant
Mercurialis perennis	Dog's mercury	Poor land	Whole plant
Oenanthe crocata	Hemlock, waterdropwort	Damp places	Whole plant, especially roots
Papaver spp.	Poppy	Cultivated land, wasteland	Whole plant
Paris quadrifolia (Liliaceae)	Herb Paris	Damp alkaline woodland	Low toxicity
Pteridium aquilinum	Bracken fern	Heaths, wasteland, woods	Whole plant, including rhizome and hay
Ranunculus spp., *R. sceleratus*	Buttercup, celery-leaved buttercup	Damp places, pastures	Whole plant, irritant, colic, inflammation
Rhamnus catharticus	Common (purging) buckthorn	Chalky thickets	Fruit, diarrhoea
Rhododendron spp., *Azalea*, *Kalmia*	Rhododendron, azalea, kalmia	Woodland	Whole plant

Table 10.16 *Continued*

Species	Common name	Location	Toxic parts and effects reported
Saponaria officinalis	Soapwort	Hedges, banks, damp places	Whole plant, mild, saponins as for *Stellaria*
Senecio spp.	Especially ragwort	Old pasture	Whole plant
Solanum dulcamara	Woody nightshade, bittersweet	Road sides	Whole plant
Solanum nigrum	Black nightshade	Cultivated lowland	All parts, especially berries
Solanum tuberosum	Potato	Cultivated land	All parts including tubers when damaged
Stellaria spp.	Chickweeds	Ubiquitous annual	Whole plant, mild
Tamus communis	Black bryony	Woods, hedgerows	Possibly berries, mild toxicity
Taxus baccata	Yew	Woods, gardens	Whole tree, very potent

Table 10.17 Plants reported in the scientific literature to have caused poisoning in horses and ponies (Hails & Crane 1982).

Species	Common name	Effects reported
Agrostemma githago	Corncockle	Hypersalivation, abnormal thirst, accelerated respiration
Allium validum	Wild onion	Haemolytic anaemia
Amsinckia intermedia	Tarweed	Walking disease, haemolytic anaemia, liver necrosis, possibly nitrate toxicity
Aristolochia clematitis	Birthwort	Tachycardia, weak pulse, decreased appetite, constipation, polyuria
Arum maculatum	Lords-and-ladies, Cuckoopint	Purgative, irritation of GI mucosa and kidneys, fatty liver
Astragalus spp.	Locoweed or milk vetch	Eye lesions, locoism, Se accumulation in several species
A. lentiginosus, *A. mollissimus*	Locoweed	Depression, anorexia, ataxia, hyperexcitability and especially violent responses to stimuli; effects irreversible
Atalaya hemiglauca	Whitewood	Anorexia, dullness, irritability
Centaurea repens	Russian knapweed	Nigropallidal encephalomalacia, severe damage to nerve cells, muscular hypertonia

Table 10.17 *Continued*

Species	Common name	Effects reported
Centaurea solstitialis	Yellow star thistle	Nigropallidal encephalomalacia (chewing disease), hypertonia of muscles and muzzle
Cestrum diurnum		Calcinosis hypercalcaemia (other *Cestrum* spp. cause severe gastritis and liver degeneration, but not reported in horse)
Coriandrum sativum	Coriander	(No signs given in Hails & Crane 1982)
Crotalaria spp.		Hepatotoxicity, jaundice, dyspnoea, weak pulse, collapse
C. crispata		Kimberley horse disease, anorexia, dullness, staggering gait
C. dissitiflora var. *rugosa*		(No signs given in Hails & Crane 1982)
C. retusa		Kimberley horse disease, liver and central nervous system lesions, anorexia, dullness, staggering gait
Cuscuta spp.	Dodder	Enteritis, anorexia, nervous symptoms
C. campestris, C. breviflora	Peppery cuscus, dodder	Not described, but staggering, salivation, increased pulse and respiratory rates reported in cattle
Echium lycopsis (or) *E. plantagineum*)	Purple viper's bugloss	Hepatotoxicity, blindness
Equisetum spp.	Horsetail, marestail	Thiamin deficiency – the horse is highly susceptible
E. fluviatile	Water horsetail	Akin to bracken poisoning owing to presence of thiaminase
E. hyemale	Rough horsetail or Dutch rush	Loss of condition, some diarrhoea, usually caused by eating hay containing horsetails – administer thiamin or dried yeast
E. palustre	Marsh horsetail	
E. variegatum	Variegated horsetail	
Eupatorium spp. (incl. *E. rugosum*)	Including white snakeroot and hemp agrimony	Trembling, lethargy
Eupatorium adenophorum	Croftonweed	Pulmonary oedema, possible nitrate toxicity
Galeopsis spp.	Hempnettle	Pulmonary oedema, enteritis, anorexia
Glyceria maxima	Reed sweet-grass	Cyanide poisoning
Helichrysum cylindricum	Everlasting flower	Blindness, encephalopathy
Heliotropium europaeum	European heliotrope	Hepatotoxicity
Hypericum crispum		Photosensitivity
Indigofera dominii, I. enneaphylla	Birdsville indigo	Birdsville disease, drowsiness, immobility, discharges from eyes and nose, dragging of hind feet, liver lesions
Juglans nigra	Black walnut	Acute laminitis
Jussieia (Ludwigia) peruviana		
Lathyrus nissolia	Grass vetchling	Incoordination and collapse during exercise

Table 10.17 *Continued*

Species	Common name	Effects reported
Lupinus spp.	Lupin	(1) Chronic: lupinosis – congenital defects, liver damage (due to mould growing on lupins); (2) acute: respiratory paralysis due to lupin alkaloids when large amounts of the plant eaten
Medicago sativa	Lucerne	Photosensitization, possible oestrogen toxicity; certain strains cause anaemia and hepatotoxicity
Morinda reticulata		Se toxicity
Nerium oleander	Oleander	Highly toxic – convulsions, diarrhoea, colic, petechial haemorrhages
Oxytropis campestris, O. sericea	Locoweed	Nervous signs, anorexia, ataxia; damage permanent
Papaver nudicaule	Iceland poppy	Ataxia, convulsions
Perilla frutescens		Lung disease
Persea americana	Avocado	(No signs given in Hails & Crane 1982)
Phaseolus vulgaris	Kidney, haricot or navy bean	Central nervous system lesions, GI disorder
Pimelea decora	Rice flower	Colic, diarrhoea, collapse, ulceration of mouth, tongue, oesophagus, gastritis (also St George disease in cattle)
Polygonum aviculare	Knotgrass, wireweed	Nitrite poisoning, GI irritation
Prunus laurocerasus	Cherry laurel	Cyanide poisoning (also caused by other *Prunus* spp. e.g. *P. serotina*– wild black cherry)
Pteridium aquilinum	Bracken	Thiamin deficiency
Quercus spp. (incl. *Q. rubra* var. *borealis*)	Oak	Acorn poisoning: hepatotoxicity; oak-leaf poisoning: dullness, diarrhoea, constipation, dark urine
Ricinus communis	Castor-oil plant	Castor-bean poisoning: dullness, incoordination, sweating, tetanic spasms, watery diarrhoea
Robinia pseudo-acacia	False acacia	Acacia bark poisoning: colic, diarrhoea, irregular pulse, hyperexcitability, paralysis
Senecio spp. *S. jacobaea*	Groundsel, ragwort Common ragwort	Hepatotoxicity Hepatotoxicity
Setaria sphacelata	Bristle grass	Oxalate poisoning, osteodystrophia fibrosa
Sinapis arvensis	Charlock	Cyanide poisoning, colic, gastroenteritis, diarrhoea
Solanum malacoxylon		Calcinosis – wasting, stiffness, hypercalcaemia, calcification of arteries
Sorghum almum	Sorghum	Cystitis, ataxia (growing grass)
Sorghum bicolor	Sudan grass	Cystitis, ataxia (growing grass)
Sorghum halepense	Johnson grass	Cyanide poisoning
Sorghum sudanense	Sudan grass	Possible lathyrism, equine cystitis and ataxia, ankylosis (growing grass)

Table 10.17 *Continued*

Species	Common name	Effects reported
Sphenosciadum capitellatum	Whitehead	(No signs given in Hails & Crane 1982)
Stipa viridula	Sleepy grass	Hypnosis
Swainsona spp.	Darling pea	Depression, emaciation, ataxia, blindness
Tanaecium exitiosium		Weakness, staggering, collapse, frequent micturition, inflammation of stomach and heart
Taxus baccata	Yew	Heart failure – trembling, dyspnoea, collapse
Taxus cuspidata	Japanese yew	Heart failure – trembling, dyspnoea, collapse
Trichodesma incanum		Severe liver damage in horses and other farm stock
Viscum album	Mistletoe	Incoordination, dilated pupils, salivation, hypersensitivity

should be accomplished by March or April, otherwise a delay until July or August may be inevitable, without good prospects of rain. It is also imperative that plant foods be provided: Table 10.10 gives suggested rates of application. In areas of low rainfall, seeds should be sown 2.5 cm (1 in) deep; in areas of higher rainfall the sowing depth should be about 1.2 cm (0.5 in). Drilling deeper than 2.5 cm, which would be advisable in the height of summer, will result in seedlings of small seeds, such as poas and clovers, not reaching the surface, therefore for reasons of economy, they should be omitted from the seed mixture. A fine firm tilth is produced by harrowing and rolling in order to avoid a puffy, rapidly drying surface soil. Two seed mixtures are suggested in Table 10.18. These are composed of persistent strains that should not be given a cover crop and which suffer more than quicker-growing strains from drought in the seedling stage if they have been sown in late spring in poorly formed seedbeds.

In formulating one's own seed mixtures, the smaller the seed the lower the weight required per ha. Thus, a lower weight of rough meadow grass, timothy and wild white clover would be required than of perennial rye grass or meadow fescue and less is required of persistent leafy tillering strains of grasses for permanent pastures than would be used of aggressive strains in short-term leys. The better late-flowering strains of rye grass and timothy should not be mixed with aggressive early-flowering strains in the establishment of a permanent pasture. On light soils, smooth meadow grass (Kentucky bluegrass, *Poa pratensis*) is a helpful constituent of the mixture by virtue of its underground stems, which knit the surface soil

Table 10.18 Suggested seed mixtures and minimum seeding rates (kg/ha) for permanent pastures when a good seedbed has been established (under adverse conditions the quantities might need to be increased up to double these amounts).

	kg/ha
Seed mixture 1	
Perennial rye grass (Melle[1] or Contender)	10.00
Perennial rye grass (Talbot, Parcour or Morenne)	3.50
Smooth meadow-grass[2] (Arina, Dasas)	0.75
Creeping red fescue (Echo)	2.00
Rough meadow-grass (VNS)	0.75
White clover (Blancho or NZ Huia)	0.75
Total	17.75
Seed mixture 2	
Perennial rye grass (Trani or Springfield)	10.00
Perennial rye grass (Melle)	5.00
Timothy (S48, S352, S4F)	2.00
White clover (Milkanova)	2.00
Total	19.00

[1]A mixture of strains similar to S23 and S24 could be used to give a range of heading dates.

[2]Kentucky bluegrass or smooth meadow-grass (*Poa pratensis*).

together. On heavier soils this species might be replaced with tall fescue.

For some time a new sward will be more susceptible to destruction by treading than would an established turf and so ideally it should not be grazed in the first year, but should nevertheless be topped to prevent the grasses and any annual weeds from flowering. If the latter are present in excessive quantities, a selective weedkiller could be used, although this would check the establishment of clover, and in any event should not be used before the grasses have developed two or three leaves. Another option might be grazing by sheep late in the first year, but not when the surface is wet. A hay crop should not be taken from a newly established permanent pasture during the first two years at the very least.

Where ploughing and reseeding are impractical, or it is essential to establish a new pasture quickly, the author has had considerable success in horse paddocks by spraying the old turf with glyphosate once or twice during a period of rapid growth and leaving it for at least 14 days. Following this, there are two alternative procedures for reseeding:

(1) The surface is thoroughly disced (Plate 10.2), harrowed and rolled to effect a good tilth and seedbed before drilling the seed. This is delayed until the surface trash has decayed and if there are large amounts it can be burnt or raked off. The essential point is that seedlings should be surrounded by mineral soil in a firm seedbed. Burning will destroy seeds of exotic plants that have remained dormant in permanent pastures for many years and that can yield a fascinating, but mildly bothersome, crop in the young sward. Moreover, excessive vegetative matter, which contains some herbicide residues, can suppress the growth of the seedlings, but the cultivations allow soil microoganisms to contact and rapidly destroy residual herbicide. In areas of low rainfall the cultivations will dry out the surface soil during the summer, which also suppresses seed germination.
(2) After the vegetation has died, surface moisture is retained and time is saved by direct drilling the seed immediately into the soil with a special drill that has heavy disc coulters for cutting through the dead turf. The same principles of top dressing and rolling at the appropriate times should be applied as for the more traditional husbandry described.

Another alternative is to graze a worn-out paddock intensively with steers, then disc and harrow heavily at a time when the surface soil is sufficiently moist to allow penetration of the implements. After a reasonable seedbed has been attained, sow, harrow, roll and fertilize as before. Heavy regrowth of the old turf can be controlled by regular topping until the new seedlings are well established. This procedure is the least expensive but unlikely to give complete satisfaction, especially where the previous sward was dense or its regrowth too rapid during the seedling stage.

Plant disease control

Grass seedlings are sometimes killed soon after germination by soil-borne *Fusarium culmorum*. There is increasing reluctance for the licensing of pesticides that may have harmful environmental characteristics. Success has been achieved in the control of this fungus by using another fungus that is antagonistic to the *Fusarium,* but harmless to the grass seedlings. Indeed, a number of endophyte species (living entirely within the plant tissue and producing no spores) have been discovered that are synergic with grasses, i.e., they even promote the health and growth of the grass species. Endophytes occur in both rye-grass and fescue pastures (see also ‘Equine dysautonomia’, and ‘Fescue toxicosis’, this chapter). Endophytes are transmitted in infected seed and the resulting plants are more resistant to a range of pests, grow more vigorously, producing a higher DM yield, and are even more drought-tolerant and winter-hardy. Nevertheless, considerable care is now exercised by plant breeders in the use of these fungi as there is confirmed evidence that certain endophytes are a cause of disease in grazing stock. Rye grass is frequently infected by *Acremonium lolii* which is likely to be the cause of sporadic disease in grazing animals (see ‘Rye-grass staggers’, this chapter), associated with unthriftiness and loss of coordination, observed in the UK, New Zealand and in several other countries, while *A. coenophialum* is the cause of fescue toxicity. Interestingly, it is thought that the tremorigenic toxin causing the animal disease is different from that afflicting insect pests, so useful progress should be possible in selecting endophyte strains for inoculating seeds that are benign to four-footed stock. Only time will tell.

TROPICAL GRASSLAND AND FORAGES

A characteristic of most tropical forages is a high yield of DM, which is relatively rich in fibre but impoverished of CP and P (Table 10.19) and from which the rewards of animal production are relatively meagre. There are, nevertheless, large differences between the products of wet and dry seasons. In one study (Kozak & Bickel 1981) the CP content of grasses decreased from a range of 6–10%

(a)



(b)



(c)



Plate 10.2 Preparation of soil for resowing permanent pasture at Upend Stud, Newmarket, Suffolk, UK. Discing began eight days after spraying with glyphosate, a satisfactory seedbed was achieved 21 days after spraying and seed was drilled after 24 days, followed by rolling. Pictures (a) and (b) show stages in seedbed preparation and picture (c) shows pasture one year after sowing. Previously this paddock was rife with dock and thistles.

Table 10.19 Dry-matter composition of four tropical grasses fed to horses compared with a temperate (Newmarket, UK) grass sample (D. Frape unpublished observations).

	Young Napier grass	Napier hay	Mature Napier hay	Signal grass	Signal grass hay	Paragrass hay	Guatemala grass	Newmarket grass
Crude protein (%)	5.7–10	5–6	2.7–2.9	11–11.5	5–6	5.4–9.8	7.4–7.5	12.7
MAD fibre (%)	43–51	54–55	54–55	41–42	51–52	42–44	46–47	27.5
Ash (%)	3.2–4.5	3.6–3.8	2.1–2.3	7.3–7.8	3–3.2	3.4–8	6–7	9.95
Ca (%)	0.046–0.16	0.08–0.1	0.1–0.12	0.09–0.13	0.09–0.1	0.11–0.2	0.05–0.06	0.93
P (%)	0.06–0.16	0.11–0.13	0.1–0.12	0.17–0.18	0.14–0.15	0.15–0.23	0.17–0.175	0.31
Mg (%)	0.21–0.32	0.20–0.22	0.22–0.25	0.26–0.27	0.11–0.12	0.18–0.36	0.087–0.088	0.16
K (%)	0.9–1.3	0.8–0.9	0.38–0.4	2.8–3.3	0.95–1.05	0.66–2.6	2.1–2.2	1.9
Na (mg/kg)	170–690	200–300	170–190	510–650	170–180	1280–11300	150–170	1800
Zn (mg/kg)	21–38	34–36	20–22	27–29	—	35–43	—	38
Se (mg/kg)	0.055	—	—	—	0.03	0.03	0.04	0.1
F (mg/kg)	8.4	—	—	—	—	—	—	—

MAD, modified acid-detergent.

in the rainy season to 4–5% in the dry season in Tanzania without any change in the CF content, yet apparent digestibilities of the CP were 34–58% and 16–25%, respectively. Kozak & Bickel also noted that the digestibility of the DM of pasture forage decreased from 47–63% at heading to 30–53% at flowering.

It has been asserted that the yield of digestible nutrients per ha tropical grassland is at best only half what can be achieved from temperate grassland. The low digestibility of tropical grass is apparently due to a higher lignin content than in temperate grass. Analyses reveal that, as the environmental temperature rises, there is a fall in the cellulose content of tropical grass but a proportionate rise in the hemicellulose and lignin contents. Malaysian experience has shown that horses fed as much cut tropical grass as they can eat, plus oats or other concentrates, lose weight. Their general health and performance can be improved by limiting their access to grassland, or its products, and confining them for greater periods of time to the stable, which affords shade, cool ventilation and a more satisfactory control of insect-borne disease. Furthermore, where the land is light, significant amounts of sand may be consumed by grazing stock and this interferes with bowel function.

Plate 10.3a shows TB paddocks at 1350 m in Zimbabwe at the end of October, during the foaling season (surprisingly the hottest part of the summer, before the rains come). The grass is dry and bleached and the only green material appears to be *Indigofera* spp., Leguminosae, probably *I. setiflora* (Plate 10.3b). Species of this genus contain a hepatotoxic amino acid, indospicine, which competes with arginine, causing liver and kidney damage (Birdsville disease). Horses are protected from Birdsville disease in Australia by supplementing their feed with arginine-rich substances such as peanut meal or gelatin. Signs of toxicity include loss of appetite, apathy, discharges from the eyes and nose, loss of flesh, laboured breathing and severe incoordination, with a dragging of

(a)



(b)



Plate 10.3 TB stud in Zimbabwe, 1350 m, during October. This is the foaling season and the hottest and driest month, when pastures can be of poor feeding value: (a) a general view of the paddock; (b) the only green herbage in the paddock, *Indigofera* spp., a legume said to contain substances toxic to ruminants.

the hind feet. The horse can fall backwards during a canter. Many other potentially harmful plant species were identified in and around these TB paddocks (Table 10.20).

TB studs in Zimbabwe give mares 9 kg (barren mares) to 12 kg (lactating mares) relatively high protein concentrates daily. This also reduces the waste heat of fermentation which would be generated if large amounts of roughage were given. Other reasons for the large concentrate allowance are not only the low digestibility of the herbage, but also a recognition of adverse reactions if large amounts of herbage containing hazardous plants are consumed.

Oxalate poisoning

(See also 'Grass toxins', this chapter.) In the author's experience, working horses introduced to the tropics

Table 10.20 Hazardous plant species identified in and around paddocks in a subtropical TB stud.

Plant species	Comments
Eleusine indica, Panicum novemnerve	Thyrotropic cyanogenic glycosides
Setaria pumila, Portulaca spp., *Oxalis* spp.	Oxalates reducing bone calcification
Verbena bonariensis	Members of the Verbena family contain hepatotoxic icterogenin and other triterpenoids, e.g. rehmannic acid, identical to that causing *Lantana* poisoning: 'Beach disease' (see below)
Nicandra physalodes	A poisonous member of Solanaceae
Senecio venosus	Members of the genus *Senecio* contain hepatotoxic alkaloids
Sideranthus spp.	Se accumulators*
Amaranthus hybridus	Nitrates and the alkaloid lycorine, salivation, diarrhoea, paralysis
Richardia scabra	(False ipecacuanha) Very poisonous
Tagetes minuta	Very poisonous, roots poisonous to nematodes
Spermacoce senensis	Very poisonous
Lantana camara	Very poisonous, rehmannic acid, a hepatotoxin, 'Beach disease': cholestasis, anorexia, haemorrhagic gastroenteritis, ataxia, photosensitization
Convolvulus sagittatus var. *aschevsonii*	Very poisonous, purgative, nervous signs, incoordination, tremors, pulmonary oedema, polyuria, blindness; contains lysergic acid alkaloids
Cynodon dactylon	(Bermuda grass) Hepatotoxins causing secondary photosensitization
Pennisetum clandestinum	(Kikuyu grass) Salivation, thirst, distension and inflammation of GI tract, incoordination etc.
Polygala albida	Members of the genus *Polygala* cause tremors, gastroenteritis, incoordination, cerebral congestion
Leucas martinicensis	Tetracyclic polyol andromedotoxin causes hypotension, respiratory depression, attempts to vomit. The amino acid mimosine, a depilatory, causes haemorrhagic gastritis in horses
Indigofera spp.	Hepatotoxic amino acid, indospicine, causing liver and kidney damage: 'Birdsville disease'

*Over 2 mg Se/kg was detected in representative pasture herbage samples.

and required to subsist on indigenous forages and cereal grains frequently exhibit decreased performance, lameness (changing from one leg to another and involving creaking hip and shoulder joints), arching of the back, swelling of the facial bones and muscular wasting of croup and rump. Similar observations and the poisoning of grazing cattle have been recorded in various parts of south-east Asia, the Philippines, Brunei and north Australia (Seawright *et al.* 1970; Blaney *et al.* 1981a,b).

The main forage crops grown for horses in Thailand are called locally 'Yakon' and 'Mauritius Grass'. Some Purple Guinea, Rhodes and Star Grass are also grown, but almost no legumes. These grasses contain excessive oxalates and low levels of available Ca and apparently of Cu. There

are frequent cases of OCD and wobbler syndrome, and a trace-element supplementation is often helpful (Wood 2001, personal communication). The poor nutritional quality of tropical grass has frequently been overcome by the introduction of high-quality clovers, trefoils and *Medicago* spp., including lucerne, selecting those varieties low in tannins, as increased tannin concentrations are prevalent when legumes are grown on tropical soils deficient in P.

The signs described above are those of osteodystrophia fibrosa (big-head), found by the author (unpublished observations) and others (Blaney *et al.* 1981a) to be caused by large amounts of oxalate and small amounts of Ca and P in many tropical grasses. Most published reports have concerned species of the genus *Setaria* (Blaney *et al.* 1981b; Seawright *et al.* 1970), which can contain 30–70 g oxalate/kg DM. A simple method for the quantitative estimation of oxalate in tropical grasses has been proposed by Roughan & Slack (1973). Lesser but harmful quantitites of oxalates have been found in the widely used Napier (*Pennisetum purpureum*) and Signal (*Brachiaria* spp.) grasses and Paragrasses. For several reasons Napier is an inferior grass for horses. Some chemical characteristics of each of these tropical grasses, in comparison with temperate grasses, are given in Table 10.19. The samples were shown to be deficient also in Se. Ponies given a diet containing 10 g oxalic acid/kg plus 4.5 g Ca/kg exhibit decreased urinary Mg and are in negative Ca balance through reduced Ca absorption (Swartzman *et al.* 1978). It has been concluded that problems in horses arise where the dietary DM contains more than 5 g total oxalates/kg with a Ca : oxalate ratio of less than 0.5 (Blaney *et al.* 1981a). Horses presenting typical signs, and in negative balances of both Ca and P, supplemented once per week with limestone, rock phosphate or dicalcium phosphate and 50–60% molasses, consumed up to 200 g Ca and 50 g P per hour and changed to positive Ca and P balances (Gartner *et al.* 1981). Success has also been achieved by supplementing horses with 125 g limestone per day (providing 45 g Ca) until the initial abnormality has subsided and then reducing the amount to 100 g daily. If rock phosphate is introduced to the diet, the F intake should not be allowed to exceed 50 mg/kg of the total diet.

POISONOUS PLANTS

Poisonous plants are generally unpalatable. They are consumed when there is feed scarcity, when the horse has not previously encountered them, or when present dried in hay. Poisonous-weed seeds/moulds, as contaminants of grains, include *Lolium temulentum, Ricinus communis* and *Claviceps purpurea*. Table 10.16 lists the plant species more likely to be a cause of horse illness in Europe.

Plant toxicosis caused by associated microorganisms in grasses

There are several microbial species growing in plants that are a cause of intoxication in horses. The subject of microbial toxins has received much attention in recent years, probably as a result of the development of laboratory methods for their determination.

Fescue toxicosis

Perhaps one of the most pressing syndromes, at least in the US, has been equine fescue toxicosis. Approximately 688 000 horses in the US are kept on tall fescue (*Festuca arundinacea*) pastures, and for many years there have been reports of reproductive problems in mares grazing this species (Cross *et al.* 1995). Clinical manifestation in mares of the effects of ingesting ergot alkaloids of endophyte-infected Fescue grass include prolonged gestation, early embryonic mortality, abortions, dystocia (abnormal labour at foaling), tough, thickened (edematous) or retained placental membranes, agalactia, increased rates of newborn and mare mortality, weak and dysmature foals, increased sweating during warm weather, reduced serum prolactin and progesterone and increased serum oestradiol-17β concentrations. Apparently, endophyte-infected tall fescue pastures suppress serum progesterone in late gestation, but fail to suppress it, or T_3 or T_4 concentrations in early- or mid-gestation (Hill *et al.* 2001). Unlike the effects in many other species, horses consuming infected tall fescue do not exhibit increased body temperature, but show increased susceptibility to high environmental temperatures and light intolerance (Porter & Thompson 1992); recent hot summers in Europe may have increased the frequency there of this disease in horses, cattle and sheep.

In a study of six breeding stallions given feed containing ergot alkaloid-infected fescue seed, Fayrer-Hosken *et al.* (2008) were unable to determine any detrimental effects on sperm motility and sperm morphology when compared with controls. Webb *et al.* (2008), using orchard grass (*Dactylis glomerata*) determined that ergot alkaloids of endophyte-infested tall fescue (*Festuca arundinacea*) hay (average 1995 mg ergot alkaloids/kg) did not affect post-exercise recovery of respiration and heart rate when horses were subjected to a light workload during the summer months.

The abnormalities in gravid mares are caused by vasoconstrictive ergot peptide alkaloids (pyrrolizidine alkaloids have also been isolated) produced by endophytic

Table 10.21 Ergot profiles in endophytic fungi isolated from grasses growing in central Georgia, USA (Bacon 1995).

Ergot alkaloids	*Balansia* spp.		*Acremonium coenophialum*
	B. epichloe	*B. henningsiana*	
Chanoclavine I	+	+	−
Isochanoclavine I	+	+	−
Agroclavine	+	−	+
Elymoclavine	+	−	+
Penniclavine	+	−	+
Ergoclavine	+	−	−
Ergonovine	+	−	±
Ergonovinine	+	−	−
6,7-Secoagroclavine	+	−	+
Dihydroelymoclavine	−	+	−
Festuclavine	−	−	+
Ergovaline	−	−	+
Ergovalinine			+

fungi, principally *Acremonium coenophialum*, but also by *Balansia epichloe* and *B. henningsiana* identified in tall fescue (Table 10.21); but ergovaline, the primary ergopeptine isolated from *A. coenophialum*, at dietary concentrations up to 308 µg/kg apparently causes no adverse effect on nutritional or reproductive performance of mares. *Acremonium* grass endophytes are taxonomically aligned with the family Clavicipitaceae that live their entire life within the aerial parts of their grass host, producing no spores. These fungi infect other warm season weeds and grasses (including *Agrostis, Andropogon, Eragrostis, Paspalum, Sporobolu* and *Stipa*), in none of which, however, are they pathological to the plant.

Endophyte-infected tall fescue hay is less digestible than endophyte-free hay, and young horses consuming infected pasture grow more slowly than do those on endophyte-free pasture. The alkaloids are apparently serving as D2 dopamine receptor agonists, explaining their prolactin-lowering effect. Cross *et al.* (1995) advise that domperidone, a dopamine receptor antagonist, is effective in preventing the signs of tall fescue toxicosis in horses without neuroleptic side-effects. The minimum effective dose in gravid mares is 1.1 mg/kg BW/day given orally for 30 days before foaling, or 0.44 mg/kg BW given subcutaneously for 10 days before foaling.

Rye-grass staggers

Acremonium lolii is the endophyte of perennial rye grass (*Lolium perenne*), which produces the indole-isoprenoid alkaloids paxilline and lolitrem (Porter 1995), causing rye-grass staggers, a neurological condition characterized by incoordination, staggering, head shaking and collapse in horses. Lolitrem is in highest concentration in the leaf sheaths and lowest in the leaf blades. Thus, staggers is most frequently observed in closely grazed pastures and it has been reported in horses eating rye-grass straw. Reports from New Zealand and Oregon, US, are the most frequent. The ergopeptine alkaloids produced by this endophyte could be a cause of poor growth and poor reproduction on rye-grass pastures. However, pony mares given pelleted rations comprised of 45–60% perennial ryegrass contaminated with *Penicillium cyclopium* continued to eat, despite the development of neurological signs of ryegrass staggers (Blythe & Holtan 1983). *P. cyclopium* produces xanthomegnin and viomellein, but whether it also produces paxilline, or lolitrem is unclear to me, author). The subject of the effect of natural toxins on reproduction in livestock has been reviewed by James *et al.* (1992) and Cheeke (1995).

Pasture treatment against Acremonium *spp.*

Apart from domperidone treatment of mares, at present there seems to be no effective prophylactic treatment for the pastures in which tall fescue or rye grass is infected with *Acremonium* spp. The pasture infection improves the vigour and persistency of these grass species. In New Zealand, prevention is afforded by protecting the grass against weevil damage, and plant breeding is likely to be

the route by which the problem is overcome. At present, by virtue of the increased vigour endowed, endophyte-infected cultivars of both rye grass and tall fescue make up an increasing share of the total seed production.

Stringhalt

Stringhalt is a pathological high stepping gait in horses with hyperflexion of the hind limb, first reported in Australia and subsequently in New Zealand, Chile, California, Virginia and Washington state. It is associated with grazing pastures containing *Hypochoeris radicata* (Compistae, Common Cat's-ear found widely in the Europe). *Lathyrus spp.* have also been implicated. Lesions include axonal degeneration, especially of the larger myelinated fibres, segmental demyelination and neurogenic muscular atrophy. No toxin has been identified. Improvement occurs on removal from the pasture and treatment for 1 week with thiamin and phenytoin (15 mg/kg BW *per os*, once a day) (Gardner *et al.* 2005). Plant material can differ in toxicity depending on location, as samples of *H. radicata* from plants on a neighbouring farm, where no such disease had occurred, brought about recovery of a colt, only to exhibit clinical signs again when the original material was fed (Araújo *et al.* 2008).

Sleepygrass toxicosis

Sleepygrass (*Stipa robusta*) is a perennial bunch grass found on rangelands of the southwestern US. Consumption of the grass causes a profound stuporous condition in horses, which lasts for several days. *Acremonium* endophytes, containing the ergot alkaloid lysergic acid amide, are probably the causative agents (see 'Fescue toxicosis', above).

Zearalenone

It has been proposed that a foetal-loss syndrome facing Kentucky's pregnant mares and causing poor foal growth was caused by the *Fusarium* toxin zearalenone in the pasture and hay at concentrations of 0.1–0.3 mg/kg. A feed-additive mycotoxin-binding agent that prevents absorption of zearalenone in the GI tract is now available commercially in Kentucky.

Vomitoxin

Forage samples in southwestern Ontario were examined for the *Fusarium* toxins: vomitoxin, zearalenone and T_2 toxin. Vomitoxin, at concentrations exceeding 4 mg/kg DM, is associated with loss of appetite, vomiting, lesions of the intestinal tract, immunosuppression, lethargy, and ataxia. Raymond *et al.* (2001) found that 22% of forage samples contained 2–4 mg/kg DM. Wright *et al.* (2003) detected low concentrations of ergot alkaloid and fusarium mycotoxin contamination of hay and straw to which mares were exposed in Ontario, but found no relationship with several reproductive parameters. The level of contamination in most 'presentable' hay samples appears to be without ill-effect.

Annual rye-grass toxicosis

Annual rye-grass toxicosis occurs particularly in South Africa and Australia and is caused by a group of highly toxic glycolipids called corynetoxins. The signs are neurological disturbances, presented as high stepping gait, ataxia and convulsions. It is a lethal condition causing damage to the cerebellum. The aetiology is complex. Seedlings become infected by a soil nematode, *Anguina agrostis*, that leads to a gall in the flower, where the worm lays eggs. The nematode is nontoxic, but if it, in turn, is infected by a bacterium, *Clavibacter toxicus,* corynetoxins are produced and the galls become toxic. There is some evidence that the toxin is produced only if the bacteria are themselves infected with a bacteriophage. Control of the toxicosis requires that nematode infection of the grass is prevented. Crop rotation, field burning, clipping immature seed heads, fallowing and avoidance of transfer of infected material to other fields are control methods.

Equine dysautonomia (EGS, grass sickness)

Grass sickness is now recognized in England, France, Scandinavia, Switzerland, the Falkland Islands and Patagonia, but rarely in North America, Australia or Ireland. At autopsy in France of 547 Norman horses with neurologic disorders, 6.3% had dysautonomia (Laugier *et al.* 2009). Post-mortem findings in the coeliacomesenteric ganglia of horses grazing Chilean pastures at near freezing temperatures, indicated they had been suffering from dysautonomia, described locally as *mal seco* (Araya *et al.* 2002). *Clostridium botulinum* was first implicated as a causative agent in EGS when it was isolated from the GI tract of a horse with the disease in 1919. As early as 1923, Tocher suggested that EGS was a toxicoinfection and he applied a *C. botulinum* toxin/antitoxin mixture to vaccinate horses. This was shown to give protection and to reduce mortality. Clostridial bacteria can multiply in soil under anaerobic conditions and so a clostridial cause is consistent with grazing by adult horses. This is especially true when the grazing venue is changed, possibly causing an upset to the gastrointestinal milieu and its protective microflora. It is speculated that the reason other horses in contact with EGS cases seem to be at lower risk is that

they ingest a non-infectious dose of the organism and develop immunity. Recently, horses with EGS have been found to have lower levels of systemic IgG to both surface antigens of the type C neurotoxin, but horses previously in contact with EGS, or which had grazed land where it had occurred, revealed higher levels of antibodies to these antigens (Hunter & Poxton 2001). This indicates a potential for vaccination.

Classical botulism is associated with dysfunction of the neuromuscular junction and death is usually associated with respiratory failure. There are no consistent histopathological lesions in any tissue. In contrast, EGS is characterized by profound neuronal destruction in the autonomic, and particularly the enteric, nervous system. Wijnberg *et al.* (2006) employed quantitative electromyography and post-mortem examination of EGS cases to demonstrate degenerative changes, not only in the autonomic ganglia, but also minor degenerative changes in the spinal lower motor neurons. Clinical signs include muscular tremors, patchy sweating, difficulty in swallowing, salivation, dilation of the bowels and stomach and chronic loss of weight. It is recognized that *C. botulinum* causes two distinct clinical syndromes in man: that resulting from the ingestion of the toxin only, and infant botulism, in which the organism replicates in the gut, releasing toxin that is absorbed through the gut wall. This is known as a toxicoinfection and it has been proposed as a cause of EGS. There are eight antigenically distinct types of toxin, but most equine botulism is caused by *C. botulinum* types B and C. Type C, found in EGS, uniquely produces three neurotoxins that cause neurotoxicity and neurodegeneration as well as interference with neuromuscular transmission. Hunter *et al.* (1999) adduced evidence that *C. botulinum* type C and the neurotoxin (BoNT/C) were present in the ileum of nearly half the cases of EGS they investigated, but in only 7% of controls. *C. botulinum* type C is prevalent in Israel, and although outbreaks of infection are rare in their horses, anti-BoNT/C antibodies were detected in a third of the horses surveyed there by Steinman *et al.* (2007). However, no cases of EGS have yet been reported in Israel.

Other environmental agents may augment the risk. Highly cyanogenic clover is abundant in EGS fields and B.C. McGorum (personal communication) proposed that the ingestion of this clover, containing the cyanogenic glycosides, linamarin and lotaustralin, contributes to the neurotoxicity of EGS. Moreover, the detoxification of these substances depletes plasma sulphur amino acids, found to be below normal in EGS cases (McGorum & Kirk 2001). Before EGS horses become anorexic, McGorum concluded, they probably have high concentrations of blood cyanide and of plasma and urinary thiocyanate. Elevated plasma dihydroxyphenylalanine levels in EGS horses (McGorum *et al.* 2003), probably reflect a disturbance in catecholamine metabolism. Clearly, much is yet to be determined about EGS and its control, and conclusions should not be drawn prematurely about the cause(s).

Atypical myopathy

Reports exist (Vercauteren *et al.* 2007; Votion *et al.* 2007) of a dysautonomia and atypical myopathy characterized by acute postural and respiratory muscle rhabdomyolysis in young grazing horses. This results in weakness, stiffness, recumbancy, trembling, myoglobinuria and tachypnoea with expiratory dyspnoea, caused by acute muscular degeneration, without any evidence of autonomic nervous system involvement. The associated environment is generally cool with ground frosts, the pasture is frequently either bare with soil contamination of the herbage, or it has been subjected to the mechanical spreading of manure. Fat animals seem to be less predisposed to the condition.

Chronic lupinosis

Chronic lupinosis, reported in the USA, South Africa and Australia, is caused in sheep and, occasionally, in horses by the presence in lupin species of the fungus *Phomopsis leptostromiformis*, producing phomopsins, causing liver damage and icterus. Acute lupin poisoning also occurs occasionally (see 'Toxic legumes and other species', this chapter).

Plant toxins

In the plant world there is a vast array of substances that are toxic and hazardous to horses when consumed in disproportionate amounts. Many of these are known as alkaloids, which are organic, basic compounds, although the chemical diversity of all hazardous compounds is considerable, and their range of toxicity to the horse is wide. Numerous broad-leaved plant species on, or around, pastures are toxic to a greater or lesser degree as a consequence of their production of endogenous toxins. Access by horses to shrubs, trees and hedgerow plants is a typical cause of intoxication, although minor intoxication can occur, for example, from widely abundant members of the Ranunculaceae. In the author's experience, heavy infestation of pastures by some members of this family causes buccal irritation in horses. Members of this family contain an irritant yellow volatile oil, protoanemonin, but in differing amounts. This substance can cause irritation to the mouth of grazing animals, but hay containing Ranunculaceae

is safe, at least as far as this chemical is concerned, as the curing process causes precipitation of protoanemonin in a harmless form.

More severe toxicosis is caused by contamination of upland hay by bracken which causes progressive ataxia. The whole plant is toxic and the principles include a cyanogenetic glycoside in relatively low concentrations, a thiaminase (causing the ataxia), an aplastic anaemia factor, a factor causing haematuria and a carcinogen, although these last three could be identical. Water meadows are another source of hazardous plants and dry parched grassland, on which drought-resistant harmful shrubs survive, is yet another. In many cases conclusive diagnoses are unlikely.

Photosensitization

Photosensitization refers to the production of skin lesions caused by the interaction of sunlight with exogenous substances that are capable of activation by solar radiation to form free radicals. Primary photosensitization is caused by sunlight reacting directly with dietary substances in the skin after absorption. Examples include hypericin contained in St John's worts (*Hypericum* spp. especially *H. perforatum*), fagopyrin in buckwheat (*Fagopyrum esculentum*), and toxins in bog asphodel (*Narthecium ossifragum*), in species of *Vicia* and sometimes in lucerne (*Medicago sativa*), alsike (*Trifolium hybridum*) and red clovers. On other occasions, therapeutic drugs might be incriminated. Lucerne photosensitization is caused by pheophorbide-α contained in the leaves of dried lucerne. The pheophorbide-α is formed by breakdown of chlorophyll under the influence of chlorophyllase, during processing. There is a higher activity of this enzyme in legume forages than in grass.

Many absorbed toxins are detoxified in the liver and excreted in bile. This process is obviously less efficient where there is a measure of liver dysfunction. Secondary photosensitization occurs when a damaged liver is unable to clear, for example, phylloerythrin, a photodynamic, chlorophyll metabolite, in the bile. Skin reactions are most likely to be observed following the consumption of large amounts of green forage by horses that have received some hepatotoxic agent. Numerous warm-season grass species cause secondary photosensitization, characterized by photophobia and severe dermatitis. These species include *Panicum* and *Brachiaria* which contain steroidal saponins, causing liver damage, possibly by interaction with mycotoxins.

Facial eczema in sheep is secondary photosensitization caused by the mycotoxin sporidesmin, contained in spores produced by the fungus *Pithomyces chartarum*. The toxin causes free-radical liver damage. As Cu strongly catalyses the oxidation, protection is afforded by Zn supplementation which reduces Cu absorption and blood Cu level. Zn also binds with a sporidesmin metabolite, preventing its autoxidation. It is of concern in New Zealand, where sheep and cattle develop severe dermatitis on light-skinned areas of the body.

Toxic legumes and other species

Yew (*Taxus baccata*) is the most toxic plant (non-legume) in the UK, and little more than 100 g of it will kill a horse by cardiac arrest. The second is laburnum (*Laburnum*), a legume, all parts of which contain the alkaloid, cytisine, which acts similarly to nicotine, a nicotinic receptor agonist, causing incoordination (convulsive movements) and coma. There are many other leguminous species known to cause damage to horses, among them broom (*Cytisus scoparius*) and lupins (*Lupinus*). The toxicity of lupins is principally confined to the seeds, and the various strains differ in their potency. Sweet lupins (*L. lutens*) have a low alkaloid content and are grown on poor land as a source of fodder. If horses eat them death is rare and is caused by respiratory paralysis, not by liver damage. An accumulative poisoning associated with progressive liver damage (chronic lupinosis) occurs in sheep and horses in Australia. Here the causative agent is a fungus growing on the lupins.

Sweet clover or melilot (*Melilotus*) contains coumarin which is broken down to dicoumarol in hay made under bad harvesting conditions, or during moulding. Dicoumarol prolongs blood clotting time. Both white and red clovers contain toxic factors. Both contain appreciable amounts of oestrogens which are also found (in much higher concentrations) in subterranean clover (*Trifolium subterraneum*) grown on light soils. Some reports indicate the presence of oestrogenic activity in moulds infecting the clover leaves. These hormone-like substances have been associated with infertility and increased teat length in sheep and it is an open question as to whether any comparable problem occurs in grazing mares.

Alsike clover (*Trifolium hybridum*, derived from Alsike, a village near Uppsala, Sweden, although the clover is not a hybrid) is a tall-growing plant with pinkish white flowers, which may cause poisoning in horses. Wright & Ireland (2003) observed inappetence, lethargy, dehydration, pyrexia, oedema of the lower limbs, hepatitis, icterus, diarrhoea and petechial haemorrhages and ulcers of the oral cavity when adult TBs received a diet of sweetfeed, soaked sugar-beet pulp and hay containing alsike clover in amounts exceeding 20% in some bales. Two genera of

vetches in the US, milk vetches, collectively known as locoweeds, (*Astragalus*) and the related *Oxytropis* (see Table 10.17), are implicated in several abnormal conditions of horses. One caused by locoweeds is recognized as eliciting irreversible nervous signs.

Plants of the genera of locoweeds, including *Swainsona, Oxytropis* (yellow milk-vetch, mountain milk-vetch), *Astragalus* (wild lentil, yellow alpine milk-vetch, wild liquorice) and *Ipomoea,* cause α-mannosidosis, a lysosomal storage disease of herbivores, as a consequence of the presence of Swainsonine. This is a toxic indolizidine alkaloid which inhibits the lysosomal enzyme α-mannosidase, mimicking genetic mannosidosis. Nollet *et al.* (2008) recorded the first European case in a horse presenting with trembling and mild cerebellar ataxia. Swainsonine was detected in the serum, but a full recovery was achieved with fluid therapy, potassium supplementation, a dopamine drip and administration of diazepam. Loretti *et al.* (2003), in southern Brazil, reported clinical signs of intoxication in ponies, more particularly a change in behaviour, characterized by many cycles of excitement/depression, stiff gait, muscle tremor, abdominal pain and death, following introduction to pastures heavily infested with *Sida carpinifolia*. Multiple cytoplasmic vacuoles in swollen neurones of the cerebellum, spinal cord and autonomic ganglia were noted *post mortem*. There was also marked vacuolation of the renal proximal convoluted tubular cells. Lectin histochemistry of neuronal vacuoles revealed staining that coincided with that of inherited mannosidosis.

Spotted locoweed (*Astragalus lentiginosus* var. *diphysus*) which contains swainsonine, is found in pinyon–juniper woodlands on the Colorado Plateau. It is readily eaten by horses, and causes anorexia and behavioural instability (Pfister *et al.* 2003). Another locoweed (*Oxytropis sericea*) causes a serious problem on rangelands in the western USA. Pfister *et al.* (2002) demonstrated that lithium chloride, administered by stomach tube at a rate of 190 mg/kg BW, conditioned a strong and persistent aversion to this weed, although some horses required more than one pairing of the aversion agent with the taste of the weed for the effect to persist. Pfister *et al.* (2007) demonstrated that horses previously intoxicated by locoweed (*Astragalus* and *Oxytropis* spp.) can form strong and persistent aversions to it, although the strongest aversion was found in horses that had never been intoxicated.

Although none of the poisonous members of these genera is found in the UK, certain pasture species of Vicia and Lathyrus found there are mildly toxic. In parts of the US, locoweeds begin their growth in late summer and remain green through the winter. They must be grazed for a period before poisoning is obvious. Some species are Se accumulators, containing up to 300 mg/kg, and are poisonous for this reason.

Reports of calcinosis in horses from the consumption of *Cestrum diurum* (in Florida) and *Tricetum flavescens* (in Austria) exist. The plants increase the release of $1.25(OH)_2D_3$, so increasing Ca absorption resulting in hypercalcaemia (Mello 2003) (see Table 10.17).

Grass toxins

Toxins are rarely produced by grass tissues, although several species are known to do so.

Kikuyu grass poisoning

In addition to its soluble oxalates, Kikuyu grass (*Pennisetum clandestinum*) causes poisoning of horses that is probably related to saponins which accumulate during periods of rapid growth of the grass. Signs include false simulated drinking, anorexia, depression, pilo-erection, drooling, colic, grinding of teeth, cessation of intestinal movement and of defecation.

Reed canary grass poisoning

Reed canary grass (*Phalaris arundinacea*) is a forage grown on wet, poorly drained soils. It contains at least eight different alkaloids that have caused poisoning in farm livestock; but low alkaloid reed canary grass is less susceptible to the cereal rust mite (*Abacarus hysterix*) which infects large areas of timothy grown in the Mid-Atlantic region of the US. Reed canary grass is relatively unpalatable and there have been no reported cases of intoxication in horses. Ordakowski-Burk *et al.* (2006) found that reed canary grass was well accepted by horses and contained more protein, but less Ca than timothy. Nevertheless, the voluntary intake and digestibility of DM, ADF, NDF were all greater for timothy.

Oxalate poisoning

Various tropical grass species, including buffel grass (*Cenchrus ciliaris*), pangola grass (*Digitaria decumbens*), setaria (*Setaria sphacelata*) and Kikuyu grass (*Pennisetum clandestinum*) contain soluble oxalates that react with Ca to form insoluble Ca oxalate, reducing Ca absorption. This causes mobilization of bone mineral and secondary hyperparathyroidism, or osteodystrophy fibrosa in horses. Cattle and sheep are less affected, but not unaffected, owing to degradation of oxalates in the rumen. Concentrations of 5 g or more soluble oxalates/kg DM in forage grasses induce the condition in horses. Oxalate content of these

grasses is highest under conditions of rapid growth. Oxalate toxicity is also discussed under 'Tropical grassland and forages', this chapter.

Poisonous trees

Many tree species contain substances that are toxic to a greater or lesser degree (Table 10.17). The toxins are present in the leaves, bark and/or fruits. A high proportion of horses bedded on black walnut (*Juglans nigra*) wood shavings develop laminitis. An aqueous extract of the heartwood given by stomach tube was shown to cause limb oedema, mild sedation and Obel grade 3 or 4 laminitis (see Chapter 11) within 12 h. The signs are inconsistent with those caused by carbohydrate overload.

Cyanogenic species

Hydrogen cyanide toxicity

Cyanide toxicosis is caused by the inhibition of cytochrome oxidase (EC 1.9.3.1), a terminal respiratory enzyme in all cells, depriving the cell of ATP. Signs of acute intoxication include laboured breathing, excitement, gasping, staggering, convulsions, paralysis and death. In tropical and subtropical countries, enzootic equine cystitis and ataxia occur in horses grazing fresh summer annual forages of the genus *Sorghum* (Johnson grass, Sudan grass, *S. sudanense* and common sorghum). However, acute toxicosis is more likely with the consumption of sorghum hay, especially with ground and pelleted sorghum hay, owing to the rapid rate of intake and of cyanide release. Sorghums contain a cyanogenic glycoside, dhurrin, from which free cyanide can be released by enzymatic action. The glycoside and enzyme are contained in different plant cells, but damage to the plant from wilting, trampling, frost and drought result in the breakdown of cell walls, with the mixing of the juices and release of free cyanide. Arrow grass (*Triglochin*), wild black cherry (*Prunus serotina*), choke cherry (*P. virginiana*), pincherry (*P. pennsylvanica*) and flax (*Linum*) also contain cyanogenic glycosides which are readily hydrolysed to HCN. They are most toxic during rapid growth immediately after a freeze. Heavy N fertilization, wilting, trampling and plant diseases increase the hazard. Silage or haylage produced from the grasses is risky. Cystitis, or inflammation of the urinary tract, and incontinence are more common in mares than in stallions or geldings, but posterior ataxia, from which they seldom recover, is manifested in all horses.

Thiocyanate

Cyanide is readily detoxified in animal tissues during low rates of intake, by reacting with thiosulphate to form thiocyanate, the blood and urinary concentrations of which increase during chronic exposure:

$$S_2O_3^{2-} + CN^- \rightarrow SO_3^{2-} + SCN^-$$

However, the chronic production of thiocyanate (SCN^-) can induce ataxia, degenerative lesions of the central nervous system, goitre and a deficiency of S, owing to urinary loss of S as thiocyanate.

Hepatotoxins

Malnutrition can increase the risks of many toxins, or one toxin may compound the effects of another. Although definite conclusions are usually impossible, there are several well-known interactions in the field. *Heliotropium europaeum* (Boraginaceæ) is a plant that produces the pyrrolizidine alkaloid hepatotoxins heliotrine and lasiocarpine, which cause lesions associated with Cu accumulation and a subsequent haemolytic crisis of chronic Cu poisoning. *H. europaeum* would not normally be grazed if there were other more appetizing vegetation. A straightforward case was presented to the author once, in which severe liver damage and death occurred among sheep. These animals browsed in an environment where the pink flowers and green foliage of *H. europaeum* were about the only species not desiccated and these plants were set against the background of a blue streak of Cu salts in the bed of a dried-up wadi.

Seneciosis

In the UK and many temperate lands the most common source of pyrrolizidine alkaloids is ragwort (*Senecio jacobaea*), which is frequently consumed in hay. Seneciosis is a disease caused by the ingestion of certain plants of the genus *Senecio* (Compositae) which induce liver enlargement, degeneration, necrosis, cirrhosis and ascites. A constant feature has been the occlusion of the centrilobular vein (veno-occlusive disease), briefly reviewed by Hill (1959). Although ragwort is a hazardous member of this group, the inconspicuous groundsel (*S. vulgaris*) is also a source, albeit a lesser one, of the toxins. There have been many reports of seneciosis from the US, Europe and other continents. The worldwide distribution of the disease is attested by the variety of names used to describe it: 'walking disease' of horses and cattle in Nebraska, 'walking about' disease of horses and cattle in Australia, 'Pictou' disease in Nova Scotia, 'Zdar' disease in former Czechoslovakia, 'Schweinberger' disease in Germany, 'Dunziekte' of horses and cattle in South Africa, and 'Winton' disease in New Zealand. It is also recognized

in goats, chickens, quail, doves and pigs. *Crotalaria* (Leguminosæ) and *Heliotropium* have similar hepatotoxic properties so their effects are included under the umbrella term seneciosis.

Dry summers leave many paddocks and pastures in a poor state, with typical grass species dying back and only deep-rooted and drought-resistant weeds surviving. In this weather, ragwort and its seeds spread and an increase in the frequency of liver damage among horses and other herbivores can be expected to occur. The variation in resistance amongst grazing animals to this toxin reflects a variation in the efficiency of its urinary elimination (Holton *et al.* 1983). Elevated plasma concentrations of GGT (EC 2.3.2.2) are a useful early indicator of the hepatic damage caused by ragwort toxins. Horses with ragwort-damaged livers should be given a well-balanced diet containing good-quality protein supplemented with B vitamins and trace elements.

The common comfrey (*Symphytum officinale*) contains at least nine potentially hepatotoxic pyrrolizidine alkaloids in its leaves and roots, which are less toxic than those in ragwort. Comfrey has been recommended for inclusion in horse feeds, but this recommendation cannot be supported.

Aflatoxicosis

Liver damage through aflatoxicosis, derived from *Aspergillus flavus* intoxication, to which the horse is very susceptible, is less likely now within the EU as a result of legislation. This toxin was first described in groundnuts, or peanuts (*Arachis hypogaea*), but has subsequently been detected at lower concentrations in other plant species, including some cereal grains.

HOMEOPATHY

The principle of homeopathy is to give a potentially toxic chemical, which in large doses causes the signs of a specific disease, in small doses, so curing the disease. Unfortunately, much of the evidence supporting the claims is anecdotal and there is a need for them to be subjected to acceptable experimental methods of examination. The remedies used in homeopathy are extracted from materials in the animal and plant kingdoms and from natural minerals (Evans 1995), and include:

- bryonia (wild hops);
- belladonna (deadly nightshade);
- silica (pure flint);
- sulphur;
- sepia (Cuttlefish ink); and
- apis (honey bee).

The development of some treatment procedures can be hazardous, e.g. ‘nosodes’. Several of the treatments involve use of irritant substances extracted from plants, e.g. pulsatilla from *Pulsatilla nigricans* (Ranunculaceae), arnica from flowers of *Arnica montana* and related Compositae, with well-known pharmacological properties. The key question is: while high doses can be harmful, can the very low doses typically used have any measurable effect?.

STUDY QUESTIONS

(1) What would you propose should be done about worn-out, worm-infested pastures on (a) heavy clay and (b) light alkaline soils?
(2) Where (a) sheep or (b) cattle can be purchased, how would you propose a mixed pasture management system should be organized for a 50-ha stud with 30 mares and followers?
(3) What would be the sequence of decisions in planning to make haylage for a stud of 20 mares?

FURTHER READING

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11
Pests and Ailments Related to Grazing Area, Diet and Housing

For a surfeited horse. Take a handful of pennyroyal, half a handful of hyssop, an handful of sage, an handful of elder leaves or buds, an handful of nettle tops, fix large sprigs of rue, and handful of celendine, cut small and boiled in three pints of stale beer, which must be boiled to a quart.

Sir Paulet St John 1780

ARTHROPOD PARASITES

Lice

There are two species of horse lice: *Haematopinus asini* is a blood sucker and *Damalinia equi* lives on skin scales. The females lay eggs on the hair and scratching or rubbing is frequently observed, being a greater problem in the winter than in the summer as many are lost when the winter coat is shed. Control is achieved by dipping, spraying or dusting with insecticides, but a second treatment should be undertaken to kill those that will hatch from eggs already laid.

Ticks

Grazing horses, particularly those sharing ground with wild grazing and browsing animals, are prone to infestation by ticks, which can transmit diseases. However, the cattle and sheep tick found in the UK does not usually cause signs in horses. In the US, the soft tick (*Otobius megini*) lives deep in the ears, but the adults, which do not feed, live in cracks in stables, fences and under troughs where they also lay eggs. Larvae of hard ticks (*Dermacentor andersoni; Amblyomma americanum*) are found in various places on the horse, and the adults, after mating, fall to the ground, where eggs are laid in secluded locations. The tropical horse tick (*Dermacentor nitens*), whose primary host is the horse, transmits equine piroplasmosis, a protozoan blood disease. Insecticide should be applied to all parts of the skin where the ticks might be attached, including the ears. As the parasites spend long periods off the host, the grass and other areas around the stable should also be treated. With slight infestations, the ticks can be detached by application of chloroform to release their mouthparts.

Lyme disease was first described in 1977, following an outbreak in man of arthritis in Lyme, Connecticut. The disease is caused by *Borrelia burgdorferi* and antibodies to this spirochaete bacterium have been detected in the sera and synovial fluid of horses in the UK (there is, unfortunately, a lack of specificity owing to cross-reacting antibodies), the majority of which did not display clinical manifestations of Lyme disease. These signs include arthritis, myositis, weight loss, fever, laminitis and possibly meningoencephalitis. Diagnosis requires histological demonstration of silver-stained spirochaetes in skin biopsy specimens, or the (difficult) culture of the organism from blood or cerebrospinal fluid. The bacterium is transmitted by several species of the ixodid tick (*Ixodides ricinus* and *I. persulcatus* in Europe) in the northern hemisphere, which feed on many species of animal. The susceptibility of these animals to infection with *B. burgdorferi* is largely unknown, although they undoubtedly affect the prevalence of the infection. Acute infection responds to appropriate antibiotic treatment, but the chronic arthritis is often unresponsive.

Mites

Mites (*Psoroptes equi, P. cuniculi, Chorioptes bovis*) cause itch or scabs and are controlled by dipping or spraying with insecticide. As a general rule, high-pressure sprays frighten horses so that low-pressure hand-pumped sprayers are preferable and the horse should be confined to a chute during treatment.

Biting midges

An intensely pruritic dermatitis (sweet itch), which occurs during the summer months, is caused by species of *Culicoides* (Baker & Quinn 1978), a blood-sucking midge whose saliva induces an immediate-type hypersensitivity. Plasma zinc has a potent immunomodulatory capacity, influencing T-helper-cell organization and cytokine secretion. Stark *et al.* (2001) demonstrated a negative association between plasma zinc concentration and the severity of *Culicoides* hypersensitivity. Whether this relationship is an evoked response, or part of the cause needs investigation. Stock should not be grazed over wet areas where the midges are found and they should be stabled before dusk. In the Netherlands it is prevalent in habitats that have clay soils with heather and woody vegetation. The prevalence is lower in coastal habitats and environments with high rainfall and many cold days per year (van Grevenhof *et al.* 2007). Some control is achieved with antihistamines.

Flies

Several species of fly are more of a nuisance than a direct cause of trouble. The warble fly (*Hypoderma lineatum*) can cause some damage, mainly in young horses when the larva penetrates the skin of the legs and wanders under the skin to the back. When it is nearly ready to emerge it should be poulticed. The screw-worm fly (*Callitroga hominivorax*) does not occur in Western Europe and has probably been eliminated from the US. It causes wounds in the skin in which it lays eggs from which the larvae hatch. Direct treatment of the wounds with insecticide is appropriate. Several species of botfly (*Gastrophilus*) are widespread (see also, Other worm parasites, below). Some control can be achieved by sponging areas of the skin on which eggs are attached to hair, using water at a minimum of 49°C (120°F).

General stable hygiene is a major factor in the control of all flies, including the immediate removal of dung, contaminated feed and bedding. Routine grooming assists not only by removing potential trouble but by ensuring that there is a regular scrutiny of the horse's coat.

Blister beetles

Blister beetles (*Epicauta* spp. and *Macrobasis* spp.) are lethal when ingested by livestock. Various species are distributed throughout Canada and the US. They range from 0.8 to 2.7 cm in length and are black, black with grey hairs, black with red or yellow contrasting stripes, yellow with black stripes, metallic green or purple. They travel in swarms and feed on flowering plants such as lucerne or clover. When lucerne hay is harvested, the insects can be crushed and incorporated in the bale. Upon ingestion, cantharidin, an extremely stable toxin, for which there is no known remedy, is released. It is claimed that 6 g of the beetle are sufficient to kill a horse. Cantharidin causes severe inflammation of the oesophagus, stomach and intestines and during urinary excretion causes severe irritation to the urinary tract. The horse develops colic and dies within 48 h.

Hay baled in July and August is more likely to be infested than that cut earlier. If the insects are detected, they will be present in considerable numbers as a consequence of their swarming nature. By knocking biscuits of hay before feeding at least one or two fall out from infested material. The existence of the risk is, however, not a justification for the exclusion of lucerne hay from the diet.

WORM INFESTATIONS

In temperate countries, helminth infection (helminthiasis) in horses is limited to GI nematodes, including lung worm, and to liver fluke (trematodes). In tropical countries, however, horses suffer spirurid and filarial infections as well. Foals are heavily infected with migrating large strongyle nematode larvae with a pre-patent period of 6–12 months and therefore injurious infections can be present for many months before eggs are detected. Adult horses become severely parasitized by migrating larvae, even if wormed, when sharing pastures with horses that are not wormed.

The determination of the severity of worm infestation is no simple matter. Faecal egg counts simply reflect the presence of egg-laying worms. The only reliable means of establishing the degree of parasitism by GI nematodes is to analyse serum proteins. Alpha and beta globulins peak in concentration six months after infection and thereafter the latter of these proteins declines. There is a coincidental depression in serum albumin and eventually in haemoglobin. Worm egg counts are, however, of use in assessing anthelmintic efficacy in control schemes. Raised eosinophil counts reflect only migrating larvae so that these counts might not differ between treated and untreated animals. The counts tend to be highest in July and August in the northern hemisphere and so are not diagnostic. Both small and large infections with strongyles cause an elevation in the immunoglobulin IgG (T) concomitantly with depressed serum albumin before a patent infection occurs.

GI parasitic nematodes

The eggs of parasitic nematodes such as *strongylus* spp. pass out in faeces from adults in the gut and hatch into the

first and second larval stages while contained in droppings on pasture. These stages are not infectious (some species, such as the ascarid *Parascaris equorum*, undergo maturation through all three stages within the egg, and so are more resistant to deleterious environments, and can survive for years). Larvae of the third larval stage of *Strongylus* move out onto the surrounding blades of grass and are infectious. These developments require moisture. Once eaten, larvae of the third stage pass through two more stages before becoming adult, mating and beginning to lay eggs. The complete cycle takes about eight weeks in the summer, the rate depending on the ambient temperature. As autumn approaches, an increasing number of the parasites stop development and hibernate in the gut wall, to emerge again in the spring. The larvae of some nematode species migrate within the body, passing along blood vessels through the liver and lungs, causing damage. Larvae in the pasture can survive winter to infect horses the following spring in northern temperate latitudes. Free-living stages of equine strongyles are highly dependent on climatic influences. The proportion of a parasite population that is not exposed to anthelmintic treatment is described as being 'in refugia'. The season in which the pasture refugia population is small differs between climatic regions, and anthelmintic treatment at these times should be avoided as it would place significant selection pressure for resistance on worm populations (Nielsen *et al.* 2007). Most parasites are 'host specific', that is, parasites of sheep and cattle will not generally infect horses, but those of donkeys will.

Strongyloides westeri (threadworm)

Threadworms are small worms that live in the small intestines of foals. Foals are infected shortly after birth, either by ingestion of colostrum and milk containing the larvae, or by the larvae penetrating the skin. Heavy infestations cause sufficient damage to the intestinal lining to precipitate diarrhoea, loss of appetite and dullness. Foals are susceptible up to six months of age, after which they will usually have developed immunity.

Parascaris equorum (large roundworm)

Large roundworms can reach a length of 50 cm and their lifecycle is 10–12 weeks. Eggs containing infective larvae are picked up from the pasture, or from contaminated stable bedding. The larvae migrate through the vascular system to the liver and lungs, before returning to the small intestine, where they develop into adults and lay eggs. Adult worms arrest growth and harm the appearance of foals. Heavy infestations can block the gut and migrating stages in the lungs cause 'summer colds' with fever, coughing and loss of appetite.

Cyathostome spp. (small redworm)

Small strongyloid redworms are major parasites. They are very small and live in the large intestine. The life cycle is 5–18 weeks and infective larvae are eaten with the pasture during the summer. The larvae of these worms are predominant in pastures in the UK during the summer. The seasonal emergence of huge numbers of larvae from the gut mucosa during the late winter and early spring can cause debilitating acute diarrhoea, weight loss, colic and even death. Horses infected with larval cyathostomiasis, or *Strongylus* spp., frequently show elevated serum β-1-globulin concentrations. Inflammation caused by cyathostomes can also elevate α-2-globulin; and leakage from the gut causes depressed serum albumin levels (protein-losing enteropathy), useful in diagnosis if the horse is not passing larvae. Most anthelmintic treatment will not affect larvae already encysted in the mucosa. A five-day treatment with fenbendazole is efficaceous, but benzimidazole- (fenbendazole) resistant cyathostomes have been detected in half the horses sampled in central Scotland by Chandler & Love (2002). Oral moxidectin, however, effectively suppressed faecal cyathostome egg output over a sustained period and Schumacher *et al.* (2009) found no evidence of resistance in draught horses routinely treated with moxidectin for 5 years, as measured by faecal egg counts. Ivermectin treatment of mature horses infected with cyathostomins resistant to fenbendazole and pyrantel was effective (Little *et al.* 2003). One dose of pour-on ivermectin given at 1 mg/kg BW eliminated strongyle egg production between 3 and 10 weeks after treatment. It increased red cell and reduced leucocyte counts in mares (Francisco *et al.* 2009). Apart from this, improved biological control should be considered. The nematode-trapping microfungus, *Duddingtonia flagrans*, is an aggressive trapper of free-living stages of nematode parasites. Fernández *et al.* (1999) demonstrated that this nematophagous fungus, included at the rate of 1×10^6 spores/kg BW in a feed supplement, led to >90% reduction in faecal pat contamination with horse strongyle third stage larvae, without any adverse effect on the supplement. Waller (1999) considers that the biological approach should be used in conjunction with anthelmintics, pasture hygiene and grazing management. Bello & Allen (2009) found the centrifugal flotation technique was consistently superior to the McMaster technique for recovery of cyathostomin eggs, determined by larval culture, from faeces, especially with low faecal egg numbers.

Strongylus spp. (large redworm)

Large redworms are reddish-brown worms, 2–5 cm long and have a life cycle of 6–11 months. The larvae have been detected in smaller numbers on pastures in the UK in recent years. Picked up from the pasture, the larvae of *Strongylus vulgaris* penetrate the gut wall and migrate to the main coeliac artery, where they are responsible for severe damage and blood clots. These thrombi can become dislodged and then block smaller branches of the artery. This interruption to blood supply typically causes colic.

Cyathostomes and large redworms are an important cause of debility in young grazing horses. In Denmark, Thamsborg *et al.* (1998) subjected foals of three to six months of age to pastures with either low, or high, larval contamination by large strongyles and cyathostomes for four weeks in September. The high level of contamination caused debility, inappetence and intermittent diarrhoea without colic. A transient neutrophilia and eosinophilia occurred two to eight weeks after the start of exposure, followed later by anaemia and decreased serum albumin. A marked hyper-β-globulinaemia was detected at 16–20 weeks after the start.

Slocombe & Lake (2007) in Canada administered 2.64 mg pyrantel tartrate/kg BW daily in the ration of 2–3-year-old ponies with a mean strongyle egg count of >2200/g of faeces and compared their mean egg count with those of a control group. The egg count for the untreated group remained high, whereas the treated group decreased to 0.2–16.8/g faeces. Strongyle larval counts for the pasture with the untreated ponies rose to 26 790 larvae/kg dry herbage in August, cf. 610 larvae/kg for the treated group. At the end of the season the number of strongyles in untreated and treated ponies was 69 288 and 8452.5, respectively. In the untreated ponies, 21 species of strongyles were found; approximately 84% of them were from eight species of cyathostomes cf. the treated ponies, 14 species of strongyles with 77% from one species, *Cylicostephanus minutus* (52.0%), and immature cythostomes (25%). No adverse reactions occurred in the treated ponies.

Two anthelmintics are approved for inclusion in horse feeds in the US (AAFCO; NRC 2007). These are pyrantel tartrate, which can be fed continuously at 1.2 mg/kg BW, and febendazole, which is permitted for feeding at either 5 mg/kg BW for one treatment with repeat dosing at 6–8-week intervals. Both of these are given for the control of large and small strongyles, pinworms and ascarids. However, febendazole is given at the rate of 10 mg/kg BW for ascarid control and neither compound is permitted for feeding to horses destined to be slaughtered for meat.

Oxyuris equi (pinworm)

Pinworm females are up to 10 cm in length with a life cycle of 4–5 months. The adults migrate to, and lay eggs on, the skin surrounding the anus, causing irritation. Rubbing of the anal region causes the opening of wounds, the hair to be removed and the eggs to drop off to the stabling and pasture, from which they are picked up.

Dictyocaulus arnfieldi (lungworm)

The life cycle of lungworm is 2–4 months. Infective larvae can remain in a state of retarded development and are picked up from the pasture. The larvae are swallowed and migrate through the bloodstream to the lungs, where they develop into adults. Eggs are laid there, coughed up, swallowed and passed out in the faeces. Although donkeys frequently act as carriers, they and foals rarely show signs. The examination of faeces for larvae is useful in detecting carrier animals responsible for spreading the infection. Most horses possess some resistance and do not develop patent infections. However, where they do, the pre-patent period is three months before larvae are normally found in the faeces. The parasite can elicit persistent coughing for periods exceeding one year in adult hosts, during which time veterinary lavage of the trachea for the detection of larvae in the washings is a rational diagnostic technique, even though the larvae are not readily demonstrated. Although lungworm evoke eosinophilia there is no detectable change in serum proteins. Management of lungworm infection requires identification of the carrier, which can be a donkey, or unhealthy mare shedding faecal larvae without showing signs. This animal should then be removed and treated with effective anthelmintics. Fluke and lungworm are not as economically significant among horses in the UK as GI nematodes.

Trichostrongylus axei (stomach hairworm)

Stomach hairworms have a life cycle of three weeks, they live in the stomach causing damage and irritation and are able to infect cattle and sheep. Larvae of this parasite have been found to assume major importance on pastures in the UK from August until October.

Habronema muscae (large-mouthed stomach worm)

The adults of the large-mouthed stomach worm live in the stomach. Eggs are passed out in the dung, where they hatch and are picked up by fly maggots feeding in the dung. The larvae are carried in the mouthparts of the fly. As the fly feeds it passes the larvae to the feeding horse,

which swallows the larvae. Larvae deposited on sores and wounds of the horse's skin do not complete their life cycle, but cause intense irritation and 'summer sores'.

Onchocerca spp. (neck threadworm)

Adults of the neck threadworm live in tendons and ligaments. The larvae (microfilariae) live under the skin and in eye tissue, and are taken up by feeding midges. Microfilariae in the eyes cause problems.

Other worm parasites

Gastrophilus (bot fly)

The bot fly lays eggs on the legs and face of the horse. These hatch and enter the mouth, where the larvae live in the tissues of the lining and the tongue for several weeks before entering the stomach. The larvae attach to the stomach wall, where they remain until the following spring (the stomach shown in Plate 1.1 contained bot fly larvae). These larvae can cause ulceration and perforation of the stomach wall during this time. They then pass out in the faeces. The larvae mine underground and pupate. The adult flies emerge during the summer to lay eggs. Oral ivermectin or other insecticidal anthelmintic can be given. The lifecycle is one year.

Anoplocephala perfoliata (tapeworm)

Horses with obstructions of the ileum and caecum are frequently found to harbour the tapeworm, and horses with concurrent infestation run an increased risk of ileo–caecal colic (Proudman & Edwards 1993). Proudman *et al.* (1998) concluded from a survey that many of their spasmodic colic cases, and most ileal impaction cases, were associated with tapeworm (*A. perfoliata*) infection. Tapeworm eggs are passed out, contained in proglottids (segments), in the dung. The eggs are consumed by free-living oribatid mites, which are, in turn, eaten with the grass by the horse. The adult worms attach to the wall of the intestines at the junction of the small and large gut. The prevalence of *A. perfoliata* in equine populations is 20–80% Much of the apparent variation can be attributed to the difficulty of diagnosis, as detection of the eggs in faeces is notoriously difficult and immunological memory causes a lack of sensitivity of the tapeworm enzyme-linked immunosorbent assay (ELISA). The sensitivity of both faecal egg count and ELISA serum antibody levels increases greatly with infection intensity (number of tapeworms) (Kjær *et al.* 2007). More sensitive coproantigen and molecular methods are being developed (Abbott & Barrett 2008).

Control of GI worm parasites

Control requires an effective worming programme and good pasture management. The essence of control is to reduce the number of infective larvae on pastures grazed by susceptible stock, particularly those under three years old. There is some evidence that tolerance to both strongyle and ascarid nematodes is developed, so that stock should not be kept entirely isolated from infective sources. Faecal egg counts reflect only the activity of adult worms in the intestines.

Horses should be treated orally with anthelmintics on arrival at a stable or as directed by the veterinary surgeon; however, an initial loading dose, much larger than normal, is prudent in cases of severe infection with strongyles, as such doses of thiabendazole or fenbendazole can be larvicidal. Veterinary guidance is essential. Moderate but closely defined doses of oxfendazole or ivermectin (Dunsmore 1985) have been shown to possess efficacy against adult ascarids and small and large strongyles at all stages from egg to adults, including migrating larvae. Anthelmintic efficacy against mature and migrating larvae of various parasitic species was found to be enhanced by the co-administration of oxfendazole and piperonyl butoxide as a metabolic inhibitor. By this means the effective lifespan of benzimidazole anthelmintics can be extended (Sánchez Bruni *et al.* 2005).

Young stock should always have access to the cleanest pasture until they have developed some tolerance to worms. Mares must therefore be properly treated so that they do not pass on any severe infection to their offspring; droppings can be removed expeditiously by the use of vacuum cleaner attachments to tractors. Stabled horses should also be treated routinely, particularly where they have been given access to pasture, even for short periods, in the summer. Table 11.1 gives a simple routine of treatment. The life histories of two species are described in Figure 11.1.

Strongyles (redworm)

Strongyle eggs develop into infective larvae only in the period between March and October, especially in warm weather. Infective larvae can survive the winter in the UK, but in the spring there is a rapid disappearance of these larvae from pasture with increasing ambient temperature. Overwintered larvae die out by June. Nevertheless, in the early grazing season, this source augments that from eggs passed by other horses during early grazing. The high level of infectivity accumulating during the summer on pasture can be contained by regular anthelmintic dosing at 4–6-week intervals, which complements the management

Table 11.1 Treatment programme for GI parasites in northern latitudes.

	Treatment	Purpose	Additional activity
January			
February	C for 5 days	Encysted small redworm	
March	B double dose	Tapeworm	Faecal examination of all stock
April	A, B or C every 6–10 weeks	Grazing season	
May			
June			Faecal examination of early foals
July			
August			Faecal examination of late foals
September	B double dose		
October	A or C	Mid-October to end of December migrating large redworm	Faecal examination of all stock
November	C	Late October to end of December encysted small redworm	
December	A	Bot	

A, avermectins; B, pyrimidines, e.g. pyrantel tartrate; C, benzimidazoles, e.g. oxfendazole (the effective life span of these anthelmintics can be extended by combining their use with piperonyl butoxide, a syngergist, cytochrome P450 non-specific esterase inhibitor to decrease the endogenous detoxification of the benzimidazole).

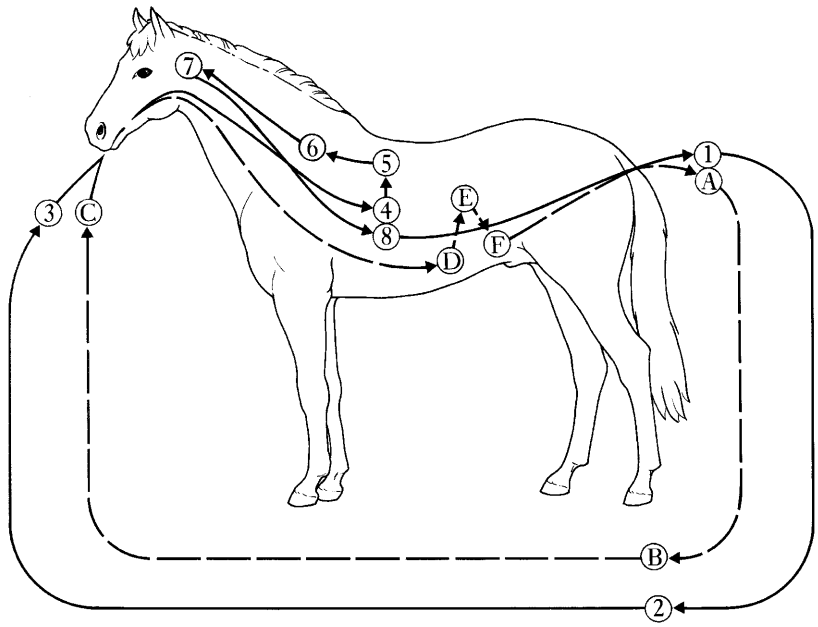
procedures given in Chapter 10. For strongyle control there is little point in dosing foals less than two months old as the pre-patent period of small strongyles is 8–10 weeks and the developmental stages are not susceptible to most anthelmintics in the normal dose range. Badly infested pastures could need ploughing and reseeding, or at least should be rested till June, by which time overwintered larvae will have largely gone. However, young stock should not be given access to them until July or August before which time grazing should be restricted to cattle or sheep.

Ascarids (large roundworms)

Ascarid infection is common in horses under three years old, by which time a considerable measure of resistance will have developed. Foals are especially susceptible and it is thought that nearly all become infected without necessarily developing signs. The migrating larvae damage successively the liver and lungs within 14 days of infection. Eggs occur in the faeces from 80 days of age. Eggs acquired by the young foal through coprophagia of the dam's droppings are normally immature and pass passively through the foal's intestines.

Clinical signs of severe infection include pyrexia, coughing, nasal discharge, nervousness, colic and unthriftiness. To preclude this, foals should be treated at four-week intervals from one month of age for control of the intestinal stages. Eggs can remain viable on pasture over winter and in suitable conditions some persist in the environment for many years. The pasture management advised for strongyles is also applicable to the control of ascarids. For a more detailed discussion of parasitic worm control in the stud see Rossdale & Ricketts (1980).

Various wormers have a different spectrum of species against which they are effective, differences in activity against adults and larvae and differences in the number and frequency of dosing. The worldwide escalation of parasite resistance is a major cause of concern for the horse industry (Brady & Nichols 2009). Inadequate dosing can lead to the development of resistance to wormers, and small redworms are now widely resistant to the benzimidazoles. Three families of wormers are used:



Ascarids

Parascaris equorum
(in young stock)

(1) Immature eggs appear in droppings from 12–15 weeks after infection
(2) Eggs may become infective in pasture or stable within 2 weeks, or remain dormant for up to several years
(3) Mature eggs ingested by susceptible foal or yearling
(4) Second stage larva hatches from egg and penetrates intestinal wall
(5) Larva reaches liver from 7 days after ingestion of infective egg
(6) Larva reaches lungs from 7–14 days after injestion
(7) Larva coughed up and swallowed
(8) From fourth to approximately thirteenth week larva grows and matures in small intestine and begins shedding eggs 12–15 weeks after infection

Strongyles

Strongylus vulgaris
(in young and adult stock)

(A) Non-infective eggs shed on pasture
(B) Larva matures within egg case on the pasture during the grazing season
(C) Third-stage infective larva ingested
(D) Larva penetrates submucosa of intestine within a few days
(E) By 14 days after ingestion larva has reached the anterior mesenteric artery where it develops over a 4-month period
(F) Young adult returns to intestine (especially caecum or colon) where it becomes sexually mature by 6 months after ingestion when egg shedding commences

(*Strongylus edentatus* is similar but larvae migrate only as far as the colonic wall before returning to the lumen)

Figure 11.1 Life history of a roundworm (*Parascaris equorum*) and large redworm (*Strongylus vulgaris*).

(1) avermectins
(2) pyrimidines
(3) benzimidazoles.

There are several principles to worming:

- Know the weight of each horse so that correct dosages are given.
- Treat any horse on arrival at a new establishment and keep it stabled separately for at least two days.
- Treat all horses grazing together at the same time and with the same product. If they are at pasture during the winter, continue treatment during that period.
- Keep a diary of the date and product used for each horse.
- Do not overstock paddocks.
- Worm horses two days before moving them to clean pastures.
- Collect dung from the paddocks two to three times per week. This is probably the most important point in parasitic worm control. Moreover, strict adherence to this procedure can increase the grazing area by 50%, by eliminating the characteristic separation of horse pasture into roughs and lawns.
- To rest a pasture, do not graze it with horses from autumn until the following mid-summer. Where possible use cattle or sheep to clear infected pastures of parasites.
- Rotate wormers used in the grazing season on an annual basis, not every time the horses are wormed. The rotation should be based on changing from one to another of the three chemical groups listed above.

PROTOZOAN PARASITES

Ingestion of faeces-contaminated food or water can lead to transmission of the protozoon *Giardia duodenalis* and *Cryptosporidium parvum*, in locations where these organisms exist. In the Sierra Nevada Range of California, Atwill *et al.* (2000) detected *G. duodenalis* in the faeces of 4.6% of packstock horses and mules and estimated a prevalence for *C. parvum* of <2.4%. Suspect water sources should be fenced off if possible.

Reference to the infectivity of liver flukes (*Fasciola hepatica*) in horses was made in Chapter 10. Their presence is detected by faecal egg counts and their influence by liver-function tests, indicating liver damage. Untreated cattle and sheep encourage their spread, and snails are an obligatory intermediate host.

AILMENTS RELATED TO DIET

Microbial spoilage of feeds

Bacteria grow in feeds where the moisture content is over about 16%. This results from poor drying or from secondary water uptake in humid atmospheres and condensation on the surface of feeds. Cereal grains that have been ground or crushed, or feeds such as bran, with a large surface area, are more susceptible to bacterial and mould growth. (See also 'Feed storage', Chapter 5.)

Endotoxaemia and laminitis

The clinical signs of laminitis seem to be caused by several different mechanisms, each of which might lead to different lesions, but with similar end results. Genetic predisposition is involved in at least one of these mechanisms. Evidence shows that endotoxaemia and laminitis are related in the horse. Endotoxemia is an important risk factor for development of acute laminitis in horses during hospitalization (Parsons *et al.*2007). The dietary causes of acute laminitis and endotoxaemia are, to a considerable extent, limited to the consequences of an excessive consumption of readily fermentable carbohydrate by horses that have been inadequately adapted to the diet.

The endogenous release of corticosteroids in response to stress, previous exposure of tissues to endotoxin, followed by stress, are factors that increase the probability of laminitis occurring following grain overload, or an overload of lush young grass. Platelet-activating factor (PAF) is a mediator of endotoxaemia, but whether PAF receptor antagonists play an important role in equine laminitis control is uncertain. Endotoxaemia and lactic acidosis are also implicated in obstructive bowel disease and equine colic precipitated by abrupt increases in the intake of starch.

Endotoxaemia

The horse is particularly sensitive to endotoxins and endotoxic shock can be fatal. Endotoxins are LPSs which are a structural component of the outer bacterial cell wall of the Gram-negative, nonsporing rods of the Enterobacteriaceae, including *Escherichia coli*, inhabiting the intestines. LPSs are habitually present in the intestinal contents (as much as 80 µg/mL). In fact, repeated administration of sublethal doses of LPSs results in attenuation of the host response. Moreover, both early- and late-phase endotoxin tolerance have been recognized (Allen *et al.* 1996). This tolerance not only provides protection in some individuals, but points to ways of providing prophylaxis. However, large doses of LPSs are toxic. Clinical signs of endotoxaemia are mediated by prostaglandins which seem

to inhibit gastric-acid secretion. Doherty *et al.* (2003) reported that intragastric infusion of LPS caused decreases in gastric acid and [K^+] output and an increase in [Na^+] output, partly through the mediation of prostaglandins. Endotoxaemia results in a systemic inflammatory response characterised by increased production and release of inflammatory mediators (TNF, ILs, eicosanoids), alterations in tissue perfusion, and initiation of intravascular coagulation. The pathways involve the interaction of LPS with an acute-phase protein called LPS-binding protein. The i.v. infusion of a protein-free emulsion of phospholipid (100 mg/kg over 30 min, mean 430 mL) appears to interfere with the interaction of LPS with its receptor proteins and so markedly reduces the inflammatory effects of LPS (Moore *et al.* 2007).

During grain (soluble carbohydrate) overload there is a rapid increase in numbers of lactic-acid-producing bacteria (species of anaerobic lactobacilli and streptococci) concomitant with a decline in intracaecal pH, which can fall from 7 to between 4 and 5 within 12–24 h. Starch-fermenting organisms grow much more rapidly than those that ferment cellulose, and the starch fermenters proliferate at their expense. Organisms that use lactic acid as a source of energy are not present in sufficient numbers to cope with the surge and their numbers decline, as some are unable to withstand the very low pH values attained. The ciliate protozoa, which are much larger and slower growing than bacteria, normally engulf starch, fermenting it at a relatively slow rate. They then act as a starch reservoir, preventing an excessive rate of bacterial starch fermentation, but they are also killed by the acid environment and so no longer function as buffers. As these normal homeostatic mechanisms are destroyed, acid production proceeds at an accelerating rate. The author's own evidence (Frape *et al.* 1982a) showed that numbers of protozoa increased with increasing starch intake up to a threshold, beyond which there was a precipitous decline in numbers.

De Fombelle *et al.* (1999) abruptly introduced barley as half of an otherwise forage diet in adult ponies. The barley was given before the forage in each of two meals daily at the maintenance level. The introduction led to an increase in the molar % of lactate and propionate and to a decrease in that of acetate in colonic contents 30 h later, without a change in the total VFA content. Fibre utilization was depressed, but no adverse effect on health occurred. After 14 days, the colonic pH had decreased from 6.7 to 6.3. Dawson *et al.* (1999) reported similar changes in ponies given barley as 50% of dietary DE. Again, lactic-acid production increased with increases in the population of lactic-acid-producing enterococci, but there was also a rapid increase in the population of lactic-acid-utilizing bacteria (these probably included *Veillonella* and *Propionibacter* spp., author), so that lactic-acid concentration was lower than with no, or 30% barley. Hence, the response to the introduction of starchy food can be without untoward effects in many horses.

Where adverse effects occur, many of the Enterobacteriaceae die, releasing large amounts of LPS into the gut lumen, causing loss of the integrity of the colonic mucosa. This is likely to be caused by intestinal ischaemia, perhaps abetted by the lactic acidosis, or by previous parasitism. Concentrate overload has led to concentrations of endotoxin of 1–30 µg/mL in the small intestine, but in the large intestine the concentrations can be up to 160 µg/mL. Consequently there is a considerable transmural movement of LPSs, leading to their presence in portal and systemic blood.

In health, endotoxin is cleared rapidly by the Kupffer's cells of the mononuclear phagocytic system within the liver and so plasma levels are normally <0.1 ng/L. The overloading of this system, leading to endotoxaemia (plasma levels of 2.5–82 ng/L with two peak concentrations 32 and 48 h after carbohydrate overload), is associated with early systemic vasoconstriction, tachycardia, arterial hypoxaemia, hyperpneoa, respiratory alkalosis, pulmonary hypertension and fever, followed by an increase in vascular permeability, haemoconcentration, systemic hypotension, an alteration in mucous membrane colour, a prolongation of the capillary refill time, capillary thrombosis, thrombocytopenia and neutropenia with neutrophil sequestration in blood vessels. The decrease in capillary blood flow causes decreased blood perfusion of vital organs, but there is an increased perfusion of the GI tract. The decreased capillary flow and slow refilling of the capillary bed is associated with cold extremities. Incomplete perfusion of the lungs through capillary shunting causes incomplete oxygenation and lowered oxygen tension of the blood (hypoxaemia). This response, added to the restricted blood flow through other tissues and organs, including restricted extraction of lactate by the kidneys, aggravates the situation and contributes to anaerobic glycolysis and further production of lactic acid. A restricted hepatic blood flow reduces the removal and metabolism of lactic acid, and diarrhoea causes dehydration, contributing to haemoconcentration. Hypovolaemia is common in critically ill horses. Horses can lose large volumes of fluid into the GI tract and this can be exacerbated by endotoxaemia-associated vasodilatation, often accompanying gastrointestinal disease. In response the

horse secretes aldosterone (causing retention of sodium), vasopressin (causing vasoconstriction, retention of water and sodium) and atrial natriuretic peptide (ANP), causing diurisis, natriuresis and vasodilatation) that interact to allow fluid and electrolyte homeostasis.

Endotoxaemia is said to occur in at least 25% of horses with colic admitted to clinics (Moore *et al*. 1981a). The effects of endotoxin on the colon are very different from their effects on the small intestine. The decreased perfusion of tissues, with a shift to anaerobic metabolism, causes blood lactic acid concentrations of >700 mg/L, which are associated with death in LPS shock. Experiments in which these toxins have been administered i.v., or intraperitoneally, at rates of 2–30 µg/kg BW, have led to an increase in inflammatory circulating eicosanoids such as thromboxane (TXB_2), prostacyclin, 6-keto-prostaglandin $F_{1\alpha}$, and prostaglandin E_2 (PGE_2) (Ward *et al.* 1987; King & Gerring 1991). The eicosanoids are derived from arachidonic acid, mobilized during damage to the endothelium of blood vessels by endotoxin (see 'Polyunsaturated fatty acids', Chapter 5). Horses with intestinal ischaemia have a plasma endotoxin concentration in the range 30–100 ng/L. Plasma endotoxin of 0.1 µg/L (100 ng/L) leads to an increase in body temperature of approximately 1°C. Tóth *et al*. (2009, 2010) administered i.v. endotoxin (7.5 ng/kg BW per h for 8 hours), or oligofructose (5 g/kg BW) by nasogastric infusion, or both to mature healthy horses. All three treatments reduced insulin sensitivity. In the second study prolonged insulin infusion for 48 h caused laminitis in all horses. The synthesis and action of inflammatory mediators are central to the aetiology of endotoxaemia, and potent cyclo-oxygenase inhibitors (which inhibit conversion of arachidonic acid to eicosanoids) are of use if administered promptly. The feeding of fish oil (probably 500 g/day plus vitamin E) as a prophylactic might help.

Where grain overloading has occurred, and endotoxaemia is likely, early therapy is most rewarding. Treatment should also be directed towards preventing laminitis:

- Previous therapeutic approaches have concentrated on cyclo-oxygenase-2 (COX-2) inhibitors, NSAIDS and glucocorticosteroids, ultimately inhibitors of prostaglandin synthesis. However, convincing evidence for the efficacy of these agents in the horse is lacking and therapeutic intervention for LPS action has switched from the post-transcriptional level to pre-transcriptional signal transduction, which is thought to reduce organ damage caused by overreaction of the immune system to pathogens and allergens (Werners *et al.* 2005).
- fluid replacement (this is important in treatment, including glucose, and correction of any bicarbonate deficit, after measuring acid–base status). Oral fluid therapy is adequate in mild cases, but i.v. administration of physiological solutions will be necessary in severely affected horses;
- colloid solutions (increased vascular permeability and loss of blood colloid is a characteristic of endotoxaemia);
- evacuation of the starch overload from the GI tract. Mineral oil, administered by nasogastric tube, assists by decreasing bacterial fermentation, and facilitating evacuation of colonic contents. Lipoid pneumonia has resulted from administration of mineral oil using a nasogastric tube. Tachypnoea and tachycardia are common presenting characteristics and incorrect placement of the tube must be avoided (Scarratt *et al.* 1998; Bos *et al.* 2002);
- virginiamycin treatment (see 'Lactic acid production and measurement', this chapter). This treatment is not permitted under EC rules.

Laminitis

The first scientific investigation of laminitis was conducted by Obel in 1948, whose name remains linked to the scale describing the severity of lameness. Obel's four grades of laminitis are:

- Grade I: feet lifted incessantly and alternately; no lameness at a walk, but at a trot horse moves with a short stilted gait;
- Grade II: horse is willing to move, but gait is characteristic for laminitis. A forefoot can be lifted without difficulty;
- Grade III: horse moves reluctantly and vigorously resists attempts to lift a forefoot; and
- Grade IV: horse moves only when forced.

Laminitis of Obel Grades I to IV occurs with unerring frequency following endotoxaemia.

Laminitis is the local manifestation of a serious metabolic disturbance. The front feet are frequently the most severely affected, and the animal adopts a straddled stance with the front feet pushed forwards. Often a strong digital pulse can be detected at the fetlock; sometimes petechial haemorrhages are observed in the buccal cavity and oedema seen along the underline of the belly. Abdominal pain (colic) accompanies ischaemia with a loss of intestinal borborygmi. Chronic inflammation of the hoof initiates a more rapid growth of the hoof wall than would otherwise occur so that the toe of the hoof extends and curls up, heavy rings developing on the wall in response to inflam-

mation of the coronary band. Infections can focus on cracks developing between the hoof wall and the sole. Where the angle between the hoof wall and the surface of the third phalanx (pedal bone), measured on X-ray photographs, is excessive, the prognosis is doubtful. The malformation can be partly overcome by rasping and corrective shoeing and any overweight condition should be rectified.

The primary lesion in laminitis is the opening of arteriovenous anastomoses, causing inadequate perfusion of the dermal laminae. The endotoxin-induced formation of microthrombi and venous thrombosis contribute to this failure in circulation. Platelets, critical for thrombus formation, play an important role in the pathophysiology of ischaemic vascular diseases, and heparin has been shown to decrease the prevalence of carbohydrate-induced laminitis. The incubation of equine platelets in the presence of LPS and leucocytes caused the platelets to release 5-hydroxytryptamine (5-HT, serotonin), a potent vasoconstrictor of equine digital blood vessels (Bailey *et al.* 2000). The leucocytes provide PAF which is necessary for this release.

NRC (2007) considers that there are three theories on the aetiology of laminitis: (1) vascular haemodynamic, (2) toxic, metabolic, or enzymatic, and (3) traumatic or mechanical overload (Bailey *et al.* 2004). The first indicates there is a change in digital vascular function, with impaired blood supply to the laminae, resulting in failure of the dermal–epidermal junction, initiated possibly by vasoactive monoamines released from the gut (but see insulin and laminitis, below). The second states that a trigger factor delivered by the vascular system activates metallic matrix metallo-proteinases, leading to a breakdown of the basement membrane and separation of the dermal–epidermal junction; and a third cause could be trauma/mechanical overload. Inflammation is a common denominator in mechanisms initiating laminitis, indicating the importance of anti-inflammatory therapy (Orsini *et al.* 2009). Amines play a vasoactive role and contribute to the peripheral circulatory disturbances of laminitis. Bailey *et al.* (2002) determined that a caecal overload of either starch or inulin caused a large increase in the amine content of caecal fluid, a response arrested by virginiamycin. There is increased blood flow to the foot, recognized by a bounding digital pulse, with a shunting of blood away from the laminae. The ischaemia, and lack of nutrition, of the laminae eventually cause a degeneration of the bond between the hoof and the pedal bone (third phalanx), which sinks and rotates owing to the horse's weight, initiating a chronic phase of the disease. In the acute phase it is therefore essential to correct the loss of blood to the laminae in order that chronic damage is arrested. As in humans, feeding horses large meals episodically leads to a transient postprandial plasma hypovolaemia, associated with a loss of sodium and water from the circulation into the GI tract. This could stimulate a peripheral vasoconstriction to maintain systemic blood pressure, but apparently it does not occur in equine digital arteries (Hoffman *et al.* 2001d).

Lactic acid production and laminitis

The predisposing health problems for laminitis are: colic 23%, grain overload 23% and grass founder 8% (Slater *et al.* 1995). Garner *et al.* (1977) reported that, following carbohydrate overload, the greatest increase in blood lactate normally precipitates circulatory collapse and death, with or without signs of laminitis. Lesser increases in blood lactate are more frequently associated with laminitis and the lowest increases with neither effect (circulatory collapse or laminitis). The development of laminitis is the most frequent outcome of lactic acidosis. In their study, 70% of cases developed this condition, whereas only 15% suffered fatal cardiovascular collapse. The rate of increase in blood lactate, as determined by blood measurements at 8 and 16 hours, gives a fair indication of whether the horse will die or will develop laminitis, and so provides the basis for appropriate therapy (Garner *et al.* 1977). Where arterial plasma lactate exceeds 8 mmol/L, death is inevitable, whereas survival is probable with maximum values below 3 mmol/L (Coffman 1979c).

Measurement of lactic acid

The shock of endotoxaemia results in increased anaerobic glycolysis in muscles. The product of this is L-lactic acid, associated with metabolic acidosis. Reliable measurements of this acid require careful, problem-free sample collection and immediate mixing with cold perchloric acid or sodium fluoride. To measure blood D-lactic acid (see below) the samples should be treated with perchloric acid and analysed using D-lactate dehydrogenase. Alternatively, plasma anion gap: $[Na^+] + [K^+] - (\text{chloride} + \text{bicarbonate})$ measurement is free from these problems and is a slightly better prognostic indicator than lactic acid measurement. It is a good measure of the accumulation of acid anions (e.g. lactate, ketoacids, phosphate) generally (Gossett *et al.* 1987), and is suitable for routine application.

Lactic acid is, of course, normally produced in the muscles during anaerobic work, as described in Chapter 9. Why then is the lactic acid produced in the intestine potentially more lethal? First, anaerobic work can be sustained for only a few minutes, after which aerobic conditions lead to the complete metabolism of the lactic acid. In contrast,

Table 11.2 Daily ration to be divided into at least three feeds for horses and ponies with laminitis.

	Mature weight (kg)	Concentrate	Concentrate per day (kg)	Grass hay per day (kg)
Pony	200		—	3.50
	300		—	4.25
Horse	500			*Ad libitum*
		Micronized oats	1.5[1]	
		Oatfeed or molassed chaff	1–2	
		Soya/micronized soya	0.5	
		Soya hulls	1.5	
		Wheatfeed	1.5	
		Molasses (beet or cane)[2]	0.5–0.6	
		Limestone flour	0.05	
		Potassium chloride	0.03	
		Sodium bentonite[3]	0.05	
		Stabilized fish oil[3] + vitamin E	0.35	

[1]Micronized and crushed oats are well digested precaecally.

[2]If molassed chaff is given, provide only 0.5 kg molasses as well.

[3]These should assist in the prophylaxis of laminitis. If fish oil is unavailable replace by raw flaxseed (linseed) oil or less well by rapeseed oil. However, neither fish oil nor soya oil added to a diet, with equal starch intakes, reduces the glucose or insulin responses (Vervuert *et al.* 2009b) and oils rich in n-6 fatty acids might increase the inflammatory response, associated with laminitis.

lactic-acid fermentation persists for 24–36 h. Second, at least 10 species of *Lactobacillus* and *Streptococcus bovis* produce lactic acid, either as a racemic mixture of the D(−) and L(+) forms, or in the D(−) form alone (only a few produce the L(+) form), whereas that produced in the muscles is solely of the L(+) type. This latter type is dehydrogenated by lactic dehydrogenase, with the formation of pyruvic acid. However, lactic dehydrogenase in the muscle is unable to catalyse the dehydrogenation of the laevorotatory D(−) isomer, which therefore accumulates in tissues following absorption and so can exacerbate the effects of endotoxin, as its protracted existence leads to greater tissue damage. Horses need a fully functional liver to catabolize large amounts of lactic acid and those that have suffered liver damage through disease and infestation, or from bad feed management, are less able to cope with rapid increases in the dietary energy allowance. Lactic acid accumulating in the hind gut can also induce diarrhoea. A granular form of virginiamycin (4 or 8 g/kg feed, with 8 kg feed/day), (Rowe *et al.* 1994; Johnson *et al.* 1998), which retains activity in the hind-gut of horses, has been shown to suppress D(−) lactic acid production. J.B. Rowe (personal communication) prevented acidosis, an elevation of blood D(−) lactic acid concentration and laminitis by oral supplementation with virginiamycin at the rate of 0.48 g/100 kg BW per day. This treatment is not permitted under EC regulations. A dietary supplement of virginiamycin reduces explorative behaviour, possibly owing to a reduction in feeding motivation (Freire *et al.* 2008).

Dietary prevention of grain overload

It is impossible to deduce what the threshold level is for starch-induced laminitis, as there is a wide gulf between the starch doses used to induce laminitis (>1000 g starch/100 kg BW) and doses (as little as 24 g starch/100 kg BW) that can cause a decrease in caecal pH, e.g. 6.26 *vs.* 6.48, and lower acetate and higher propionate concentrations (McLean *et al.* 1998b). Johnson *et al.* (1998) gave horses meals providing approximately 1500 g starch (300 g/100 kg BW) and recorded a decrease in faecal pH from 6.7 to 5.9. A decrease in the pH below approximately 6.4 was associated with abnormal behaviour, including wood chewing and bedding consumption (also see 'Gastric ulcers', this chapter).

The underlying processes in grain overload indicate that. by slowly increasing the concentrate ration, the bacteria that ferment lactic acid and the protozoa that engulf starch are encouraged to multiply (Table 11.2). These increased numbers of organisms act as a substantial buffer

against a decline in pH of the large intestine. The concentrate portion of the ration should never be increased rapidly. Forty days should be allowed when raising the concentrate portion of the diet from 1 to 8 kg. A low intake of almost any source of starch leads, primarily, to its digestion in the small intestine, but with larger meals, starch spills over into the large intestine. In order to prevent digestive dysfunction resulting from starch overload of the small intestine, starch intake in horses given 2–3 meals/day, should be limited to approximately 0.4% of BW per feeding (Potter *et al.* 1992a). More recent evidence (see 'Laminitis control', Chapter 2) indicates that the NSC components of feed should be limited to 0.25% of BW per meal. These components include both starches and fructans, as Bailey *et al.* (2002) showed that dietary inulin and corn starch lowered caecal pH to a similar extent.

Much of the earlier work suffers from a neglect of differences between species of grain in their starch digestibility, which affects the proportion entering the large intestine. Processing has a major impact. Micronization of cereal was shown to be preferable to either extruded or rolled barley, as it achieved greater precaecal starch digestion (McLean *et al.* 1998b) at moderate and high rates of intake.

Pasture, fructans and laminitis

Equine laminitis is the second highest cause of death in horses after colic, and grazing is a major source of agents. An association between rapidly fermentable pasture carbohydrates, e.g. fructans**,** and laminitis exists; but a direct relationship between this carbohydrate and the disease had not been demonstrated (NRC 2007). Fructan content of pastures is highest in spring, lowest in mid-summer and intermediate in the autumn. During the day photosynthetic activity and carbohydrate use by the plant lead to a maximum concentration of water-soluble carbohydrates in the afternoon, when grazing can lead to an intake 2–4 times the water-soluble carbohydrate intake of the remainder of the 24 h. The daily range of fructan intake during grazing can range from 2.5–10.5 g/kg BW (Longland *et al.*1999). The upper end of this range falls within the range of oral fructan dosage (7.5–12.5 g/kg BW) reported to induce laminitis. Lesions have been detected 24 h after FOS administration by nasogastric tube of 10 g/kg BW (French & Pollitt 2004) and 5.0 g/kg BW, which also altered glucose dynamics, but not those of insulin (Kalck *et al.* 2009), establishing a link between field cases and pasture fructan content (see below).

Undigested soluble carbohydrate (fructans) in the hind gut causes a rapid rise in the population of Gram-positive bacteria (streptococci and lactobacilli) and peak D-lactic acid acidaemia occurs 18 h after dosing (van Eps & Pollitt 2006; Nourian *et al.* 2007). Cool season grass-legume mix pasture has been reported to contain only 18 ± 8 to 57 ± 17 g fructans/kg DM (Vervuert *et al.* 2005d), a range lower than that which is associated with laminitis. Nevertheless, Berg *et al.* (2005) found that 24 g/day of fructooligosaccharides given to quarter horses decreased faecal pH and increased acetate, propionate, butyrate and lactate concentrations of faeces. Chicory root inulin given to ponies at the rate of 3 g/kg BW, decreased faecal pH, and increased the faecal concentrations of tryptamine (2.5-fold increase) and tyramine (2-fold increase). No changes were noted in plasma amine concentrations or plasma D- or L-lactate, indicating that there is a threshold of hindgut pH change before mucosal damage can result in the release of these factors into the circulation (Crawford *et al.* 2005, 2007).

A low dosage rate of FOS, as a prebiotic, can be protective against sudden dietary changes. The effects of an abrupt introduction of barley grain, providing starch at the rate of 0.28 %BW, replacing starch at the rate of 0.08% BW in one of the daily meals, with or without a continuous dietary supplement of 30 g FOS (chain length 3 to 5) was assessed (Respondek *et al.* 2008). The colonic *Lactobacillus* and *Streptococcus* populations and D-lactate concentrations increased during the first 29 h following the barley meal in the control group, whereas in the FOS group the populations remained stable at concentrations similar to those reached in the controls, both before and after the barley meal, but D-lactate concentrations did not accumulate. Hussein *et al.* (2004) showed that horses could be supplemented with cereal grains in amounts not exceeding 0.2% of BW, as total non-structural carbohydrate, without adversely affecting health; but with a decrease in faecal pH from 7.04 to 6.74. Swyers *et al.* (2008) determined that the probiotic *Lactobacillus acidophilus* at a dose of 10^8 cfu/50 kg BW per day, marginally limited the decline in faecal pH, so minimizing the risk of acidosis caused by an abrupt increase in starch intake from 1.2 g/kg BW per meal to 2.4 g/kg BW per meal (i.e. 0.24% BW). These data support the conclusion that cereal grain allowance should build up to no more than 0.25% BW per meal.

Black walnut (Juglans nigra) and laminitis

Laminitis resulting from poisoning with black walnut (the buds, nut hulls, stems, roots and to a lesser extent, leaves and stems contain 5-hydroxy-1,4-naphthoquinone) is associated with a high susceptibility of digital laminae to damage by the superoxide anion, produced by emigrant inflammatory leucocytes (Loftus *et al.* 2006). Black walnut

juice also induces eosinophilic colitis, so indicating its association with colic in horses (Merritt 2005).

Laminitis and insulin resistance

Insulin resistance (IR) is a state in which target cells fail to respond to normal circulating concentrations of insulin. The pathological processes implicated in the clinical development of laminitis comprise inflammation, vasoconstriction, thrombosis, ischaemia, endothelial dysfunction, free radical damage and enzymatic degradation. IR is associated with all these laminogenic processes, any one of which could explain the putative mechanism by which IR predisposes horses to laminitis (Durham *et al.* 2008). Insulin resistance can result from elevated blood levels of pro-inflammatory cytokines, so is it likely to be a direct cause of laminitis? **Resistin** is secreted from adipocytes, and high circulating levels have been associated with obesity and insulin resistance. It (1) increases transcriptional events leading to an increased expression of several pro-inflammatory cytokines and (2) decreases insulin responsiveness to the glucose transport system. These data suggest a role for resistin as an autocrine/paracrine factor affecting inflammation and insulin sensitivity in adipose tissue.

Whereas to date models have focused on carbohydrate perturbations of the gastrointestinal tract, or those of inflammation (Black Walnut), McGowan (2008b) proposed a new model of laminitis. Conditions associated with endocrinopathic laminitis include equine Cushing's syndrome, also called pituitary *pars intermedia* dysfunction, insulin resistance, and iatrogenic corticosteroid administration. The marked overactivity of the *pars intermedia* in Cushing's syndrome results in excess ACTH production, leading to excess cortisol secretion that has an opposing action on glucose metabolism to that of insulin, such that hyperinsulinaemia and insulin resistance are consequences. Horses with abnormally high blood levels of insulin (>188 μIU/mL) are much more likely to develop laminitis and usually survive for less than 2 years (McGowan 2008b)**.** A programme of dietary control and exercise are important in reducing plasma insulin concentration and the risk of Obel grade laminitis **(**Walsh *et al.* 2009).

Bailey *et al.* (2007) determined whether pasture, and specifically the addition of fructan carbohydrate to the diet, induces exaggerated changes in serum insulin concentration in insulin-resistant, non-obese ponies predisposed to laminitis. Compared with healthy controls, blood insulin concentration in these ponies was greater, it decreased in response to feeding hay, it increased (5.5-fold) following the feeding of inulin with hay and the increase in serum insulin concentration 19 h after dexamethasone administration was greater. Ponies presenting with a peripheral cushingoid syndrome: laminitis, hypothyroidism and obesity, had twice the normal blood concentration of insulin when grazing winter pasture, but on lush spring pasture they developed laminitis with an even greater basal plasma insulin concentration (Treiber *et al.* 2005b, 2006). Bailey *et al.* (2008) determined that ponies predisposed to laminitis and control ponies reacted similarly at pasture during winter, whereas, in June, laminitis-prone ponies, cf. controls, were insulin resistant, with higher blood pressures and increased serum insulin, plasma TAG and uric acid concentrations. Summer pastures appear to induce metabolic responses in some ponies, leading to expression of the pre-laminitic phenotype, which includes hypertension as well as insulin resistance. Signs of this metabolic syndrome might not be apparent in affected ponies during grazing of winter pasture. Its underlying cause is likely to be enhanced metabolic efficiency ('easy keepers', Frank 2009) and its control is through diet and exercise (Schmidt & Hickey 2009).

Interestingly, evidence produced in Australia has shown that glucose uptake by the hoof lamellae, the cells of which have no insulin receptor, is insulin independent (McGowan 2008b). However, pro-inflammatory changes and effects on the microvasculature of insulin resistance may precipitate laminitis in horses by affecting blood flow. Slowly developing endocrinopathic laminitis was induced in ponies exposed to supraphysiologic circulating insulin (1000–1100 mU/L) concentrations for 2–3 days (Geor 2008) and prolonged these concentrations (mean 1036 mU/L), while maintaining euglycaemia (5.2 mmol/L) (Asplin *et al.* 2007), effects confirmed by Tóth *et al*. (2009, 2010). The ponies were healthy, 6.5 years old, and non-obese, with no history of laminitis and no evidence of endocrine or other abnormalities. Laminitis occurred slowly in all four limbs, with ponies developing mild signs (Obel grade 1) by 32.6 h and signs of lameness (Obel grade 2) by 55 h. There is a predisposition to laminitis of an insulin-resistant phenotype characterised by obesity, IR, hyperinsulinemia, and hyperleptinemia. Euglycaemic hyperinsulinaemia in ponies causes lamellar proliferation, probably through a mitogenic pathway characteristic of acute and chronic laminitis that might weaken the distal phalanx attachment (Nourian *et al.* 2009). This differs from the carbohydrate overload or oligofructose (and black walnut) effects where there is separation at the lamellar dermal/epidermal interface, so the hyperinsulinic lesions might be reversible. Carter *et al.* (2009) found

high diagnostic accuracy in the prediction of pasture-associated laminitis amongst healthy adult Welsh and Dartmoor ponies examined on pasture in late winter (March) during the morning, using several measures of obesity (plasma insulin >32 mU/L and leptin >7.3 ng/mL). McGowan (2008b) concludes that pro-inflammatory, or vascular, effects are involved in triggering endocrinopathic laminitis associated with insulin resistance. Treatment of insulin-resistant horses should include attention to diet and exercise, as laminitis caused by hyperinsulinaemia might be reversible.

Obesity is becoming a major health concern in horses because of its associations with insulin resistance, oxidative stress/inflammation, and laminitis. An interrelationship exists of obesity in mares with inflammatory cytokines, TNFα, IL1, IL6 and insulin sensitivity (Vick *et al.* 2007). The USDA National Animal Health Monitoring System 1998 Equine Study estimated that approximately 1.4% of the US horse population are overweight or obese. Thatcher *et al.* (2008) determined the prevalence of overweight and obesity in a sub-population of 300 mature, Light Breed horses in Virginia. Based on the BCS, six horses (2%) were under condition, 141 horses (47%) were in optimal condition, 96 horses (32%) were over condition, and 57 (19%) were obese.

Pre-emptive avoidance measures are recommended in horses at risk with a focus on (1) improving insulin sensitivity (eg, caloric restriction, increased exercise, judicious use of pharmacologic agents such as levothyroxine sodium, and metformin) and (2) minimizing exposure to environmental factors that increase the risk of laminitis (elimination of grains and sweet feeds from the diet and restricting access to pasture during high-risk periods such as spring). Nevertheless, measures of insulin sensitivity have low repeatability within horses, so conclusions should not be based on single determinattions (Pratt *et al.* 2009).

Fat supplementation of Shetland ponies results in glucose intolerance (Schmidt *et al.* 2001). However, the supplementary use of oils rich in the lower *n*-3 fatty acids, such as linseed or rapeseed, is beneficial, as the higher *n*-3 fatty acids EPA and DHA have an inhibitory effect on inflammatory mediators, improve insulin resistance and, as rich energy sources, can replace soluble carbohydrate in the diet (Neelley & Herthel 1997; Hall *et al.* 2004).

Laminitis and trauma

Laminitis is caused by overwork and concussion of the feet; the trauma causes inflammation and altered blood flow through the foot leading to separation of the dermal–epidermal junction.

Possible dietary pathways by which laminitis is caused

(N.B. Lesions can differ between causes)

(1) starch or fructan (NSC) overload → rapid hind gut fermentation → excessive D- or DL-lactic acid → excessive endotoxins and amines → systemic inflammatory response and/or microthrombosis and/or vasoconstriciton → further lactic acidosis.
(2) NSC overload → rapid hind gut fermentation, leading, with genetic predisposition to pro-inflammatory cytokine production and obesity → elevated circulating insulin/ insulin resistance.
(3) excessive NSC and genetic predisposition to hyperglycaemia/obesity → abnormal endothelial function.
(4) black walnut → 5-hydroxy-1,4-naphthoquinone → superoxide anion → damage to laminae.
(5) excessive NSC and stress of exercise → excessive corticosteroid production (Cushingoid) in overweight animals → hyperinsulinaemia

Other associated situations potentially leading to laminitis:

(1) excessive weight bearing on a sound leg and post-exhaustion myopathy;
(2) inadequate regular exercise;
(3) hoof trauma on hard surfaces
(4) excessive tube feeding of sick, aphagic horses with a high carbohydrate–protein diet.

Treatment

In cases of grain overload, mineral oil administered by nasogastric tube slows the absorption of endotoxin. Evidence of pedal-bone displacement and rotation should be sought radiographically and the animal should be placed on sand or mud, or have frog supports bandaged to the sole. Marked displacement is an indicator of a likely fatal outcome to laminitis. Shoeing, sole support and trimming were commonly part of the treatment protocol for chronic cases. The rapidly fermentable carbohydrate content of the diet should be reduced abruptly and replaced by good-quality hay and/or feed approved for laminitis control (see also Chapter 2, General requirements of analytical method). Feed should not be withdrawn completely, as this could cause hyperlipaemia, especially in ponies. Fat ponies and horses with a history of laminitis should be given grass hay and should not be turned out onto lush pasture (Table 11.2). Where the dietary Ca : P

ratio is low, the addition of limestone to the diet can have both therapeutic and prophylactic effects. Carter *et al.* (2009) propose treatment with botulinum toxin, type A. This results in a decrease in torsional forces on the distal phalanx.

Severe laminitis accompanying endotoxaemia and intracellular lactic acid accumulation is associated with loss of K from muscle cells into the plasma, decreasing the intra- to extra-cellular K concentration ratio. This causes membrane depolarization, but urinary K excretion does not necessarily increase (nevertheless, assessment of urinary K : creatinine clearance ratios is useful). K depletion leads to vasoconstriction of muscle capillaries, causing local ischaemia and hypoxia, anaerobic glycolysis and metabolic acidosis. Plasma K is normal, raised or lowered. The maintenance of normal cellular K concentrations depends on the integrity of energy-yielding systems. Intracellular K leaks from red blood cells into plasma in the absence of adequate glucose and analysis of whole blood should be conducted within 2 h of collection. In acute cases of K loss, i.v. dosing with limited amounts of K is appropriate, but must be carried out slowly while cardiac action is continuously monitored. Abnormal electrocardiographic changes can occur at plasma K concentrations of 6.2 mmol/L and severe cardiotoxic effects have been reported at 8–10 mmol/L. Molasses (cane or sugar beet) and KCl are recommended to be given with the hay in cases of K depletion, as the vasodilatory effects of K could be helpful.

Nitric oxide Fundamentally, acute laminitis is a vascular disease associated with areas of ischaemia or haemostasis within the hoof (Moore *et al*. 1981b). A key to this is a failure of the arginine–nitric oxide (NO) system. NO is produced by the action of NO synthase on its substrate, the amino acid L-arginine. NO relaxes vascular smooth muscle to cause vasodilatation. L-Arginine, administered i.v. at the rate of 0.42 g/kg BW, as a 10% saline solution, given at a rate of 1 mg/kg BW per min, caused immediate reperfusion of laminal tissue in an acutely laminitic pony (Hinckley *et al.* 1996). The involvement of NO was further implied when glyceryl trinitrate paste applied topically to the pasterns of an acutely laminitic pony reduced the 'bounding pulse' in the treated limbs, reduced lameness and lowered systemic blood pressure. However, it is too early to suggest that a dietary supplement of L-arginine for animals at risk is a preventive approach. L-Arginine is likely to be a semi-dietary essential amino acid in the horse. The dietary requirement is probably in the region of 25 mg/kg BW per day, so that daily supplements of the order of 5–10 g for a 500 kg horse might be considered. This is only 3% of the dose given to a laminitic pony and might not have a preventive effect.

Thyroxine (T_4) Abnormal blood T_3 and T_4 concentrations have been observed in horses affected with laminitis. The values can be depressed during the two days prior to the onset of lameness and horses with chronic laminitis have elevated serum T_3 levels. In cases of equine goitre, associated with depressed plasma T_4 levels, the recommended approach is to replace existing feedstuffs with feeds of known quality and to treat affected animals with thyroxine. The reason for this is that hypothyroidism can have resulted from excessive, or inadequate, dietary iodine. If either of these is a cause it should be established by dietary analysis before dietary changes are invoked.

White line disease

The hoof capsule consists of the hoof wall, sole, frog, bar and white line. The white line is located between the *stratum medium* of the hoof wall and the sole. It is softer than the hoof wall or the sole, and so is able to help dissipate the stresses of the hoof in motion that act to separate the wall from the sole. Disease of the white line, thought to be contributed to by nutritional problems, is visible as dark, discoloured regions that remain unnoticed under the horseshoe. If left untreated, hoof-wall separation and infection eventually occur. Kuwano *et al.* (1999) discovered 11.5% of TB racehorses in Japan with the disease. It was more frequent in the fore- than in the hind-hooves and the incidence increased with age.

Some other causes of lameness

Lameness has a multitude of different causes. Broster *et al.* (2009) examined 227 horses from India and Pakistan and found all were lame, and 98 % showed gait abnormalities in all 4 limbs caused by multiple pathological abnormalities. Lameness during training is not clearly related to diet, although poor Ca status can increase the risk of stress damage to long bones. Hardness and other features of the surface on which the horse is trained seem to be critical. A good, well-formed turf is protective, probably through a greater compliance compared with dirt. Moyer *et al.* (1991) reported that the incidence of dorsometacarpal disease (buck shins, sore shins, shin splints and stress fractures) was less in horses training on wood fibre than in those training on dirt. The wood fibre was a more compliant surface. There was a decrease of approximately 10% in stress and strain on the metacarpus (force per unit area) during fast work in horses on the wood fibre.

Osteochondrosis

Osteochondrosis (OC) is discussed in Chapter 8. The effect of OC in a joint on susceptibility to lameness depends greatly on the joint affected. Some reports on horses in training indicate that OC of the tarsocrural joint causes a degree of movement disturbance, whereas others have reported no relationship. OC of the stifle joint, on the other hand, is more frequently associated with clinical signs of lameness.

Colic (abdominal pain) and related disorders

Characteristics of colic

Tinker *et al.* (1997a,b) reported an incidence density rate of 10.6 colic cases/100 horse-years and a mortality of 0.7 colic deaths/100 horse-years. The highest incidence was amongst horses of 2–10 years of age, with TBs having the highest and Arabians the lowest breed-specific incidence rates. More than one change in hay feeding, changes in concentrate feeding and feeding high levels of concentrates yielded the highest risks. The provision of whole grain reduced this risk, supporting the well-held view that excessive intake of readily available carbohydrates is a major cause. Sutton *et al.* (2009) examined the records of 208 colic cases in Israel. Overall mortality rate was 25%. Mucous membrane colour and, especially, capillary refill time were prognostic indicators of mortality. Kaya *et al.* (2009) reported that the factors precipitating colic in Austria were decreased water consumption, high amount of concentrate intake ($P = 0.037$), low hygienic quality of hay ($P = 0.027$) and high ambient temperature on the arrival date ($P = 0.003$).

Many colics involve the presence in the stomach, or intestines, of a thick, sticky mass of fermenting feed or a compacted mass of roughage. Colic waxes and wanes in concert with intestinal smooth muscle contractions and the pain is present in several abnormal conditions. As this implies no diagnosis it is apposite to discuss the various types and causes of colic and the management favouring a healthy prognosis. Probably all equine animals experience colic several times in their life, so that in various degrees of severity it is very common,but in its most severe forms it is associated with disorders which are the most common causes of death. Records show that 80% of cases recover spontaneously in under 4 h, but in the remaining 20% a disturbance that is initially mild can become fatal, unless immediate action is taken. Colic usually accompanies a rise in blood lactate, and the severity and outcome are closely correlated with this increased value. Lactate concentrations in the peritoneal fluid are also typically higher than in the blood, except for cases of impaction.

Most colics are characterized by some of the following postures and reactions in various forms and intensities: tail twitching; pawing the ground and restlessness, with the horse getting up and down frequently; playing with its food and water; submersing the nostrils and blowing bubbles; and generally losing appetite. The head is frequently turned towards the flanks and, in the extreme, the horse rolls and thrashes about, risking further damage. However, one might enter the box to find the horse cast, with no intestinal sounds, no droppings, or a very few small ones, and a much-distended abdomen. Frequent staling (urination) is attempted in an endeavour to relieve pressure on the bladder. The rapidity of heart beat and respiration rate and the extent of sweating and fever will depend on the severity of the disorder. Normal heart rate is generally 38–40 bpm but the rate rises to 68–92 bpm in moderate colic and to over 100 bpm in severe pain. Similarly, respiration rate, normally 12–24/min, can exceed 72/min and the normal body temperature of 37.7 ± 0.3°C (100 ± 0.6°F) will be elevated. Other signs can include diarrhoea with undigested cereals in the faeces, a foul-smelling breath, ingesta in the nostrils, frequent stretching, and, occasionally, skin changes in the form of a nettle rash. Capillary perfusion time is increased, as measured by thumb pressure on the gum, colour being regained over more than the normal 1–2 s. Dehydration is also expressed as a delay in the return of the skin to its normal posture after being pinched (see Chapter 4, Water Requirements). Proudman *et al.* (2005) argue that elevated pre-operative total plasma protein is not only a measure of hydration status in small intestinal colic cases, but an important determinant of post-operative survival.

Clostridia

Excessive consumption of concentrates favours the rapid multiplication of clostridia. Equine intestinal clostridiosis (enterotoxaemia), resulting from the rapid growth of *Clostridium perfringens* type A or D in adults and *C. perfringens* type C in foals, has been described. *C. perfringens*, which secretes an enterotoxin, is a normal inhabitant of the gut. Several groups (Båverud *et al.* 1997, 1998; Weese *et al.* 2001) have observed that, in approximately a quarter of adult horses and foals presenting with intense abdominal pain or diarrhoea and enterocolitis, *C. perfringens*, *C. difficile*, or their toxins, can be demonstrated; these pathogens were only present in very few healthy controls. It is possible that, in small amounts, *C. perfringens* toxin is harmless, but when the population

of the bacterium is large, excessive gas is produced and the toxin causes damage to the intestinal mucosa precipitating diarrhoea. The toxin is neutralized by antibody, but immediate therapy involves the replacement of depleted tissue water and electrolytes and the relief of GI tympany (ballooning).

Båverud *et al.* (2003) confirmed that *C. difficile* was not only associated with acute colitis in mature horses, following antibiotic treatment, but it was also present in many healthy neonatal foals. They confirmed that it survived for at least four years in equine faeces.

Gastric ulcers

More than half of TBs in training suffer from gastric ulceration. Lesions lie in the nonglandular *pars proventricularis*, particularly the region adjacent to the *margo plicatus*. Lesions were present in the gastric mucosa of nearly all TB horses in training examined by Murray *et al.* (1996) and those in the glandular mucosa were much less severe than those in the squamous mucosa, which became particularly severe as time in training and racing progressed. In Sweden the prevalence is highest amongst Standardbreds and TBs (Sandin *et al.* 2000) and Swedish Standardbreds that had raced during the previous month, or were in preparatory training, were affected to a greater extent than fit horses that had not raced (Jonsson & Egenvall 2006). Intense exercise appears to play a major role in the development of squamous gastric ulcers, both in race horses and in endurance horses (Nieto *et al.* 2004), but the prevalence is low in horses given limited controlled exercise and kept at pasture (Andrews *et al.* 2005). Endurance horses are often given hypertonic electrolyte solutions that seem to increase the number and severity of squamous lesions (Holbrook *et al.* 2005). Exercise causes a marked, and sudden, decrease in gastric volume (Lorenzo-Figueras & Merritt 2002) that results in exposure of the squamous mucosa to the acidic contents (pH 1–2). It has been suggested that exercise decreases the rate of gastric emptying of this acidic fluid (Lester 2004) and that exercise training results in an increase in serum gastrin response (Furr *et al.* 1994) that could enhance acid secretion. Confinement with lack of socialisation and intermittent (meal) feeding could contribute to an increased risk of gastric ulceration (Coenen 1992b; Lester 2004). Intermittent (12 h) feed deprivation decreased diurnal proximal gastric pH relative to that in fed horses, whereas nocturnal pH decreased similarly in fasted and fed horses (Husted *et al.* 2009). (See also 'Gastric lesions in foals', Chapter 7.)

There is good evidence that the risk of gastric ulceration increases with the length of time horses are in race training (Orsini *et al.* 2009). Stress-associated catecholamine secretion results in sufficiently frequent vasoconstriction, hypoxia and inanition of the mucosa to precipitate the lesion. Amongst TBs, gastric ulceration is not restricted to those in training. Amongst 62 TB broodmares at one breeding farm 70.9% had gastric ulcers with no differences observed between pregnant and non-pregnant mares (le Jeune *et al.* 2009), and 53% of 201 Danish pleasure horses were shown to have gastric ulceration (Luthersson *et al.* 2009a). The variables associated with ulceration amongst Danish pleasure horses were: straw bedding the only forage available, >2 g starch/kg BW per day, or >1 g starch/kg BW per meal, unavailability of water in the turnout paddock and the interval between forage feeding was >6 h (Luthersson *et al.* 2009b).

Signs generally include periprandial colic, bruxism, ructus and reflux. Dukti *et al.* (2006) determined the causes in 100 horses with abdominal pain. Gastric ulceration was only moderately associated with the outcome and Rabuffo *et al.* (2009) found no association between colic and gastric ulceration. It is considered that gastric-acid hypersecretion, gastric emptying disorders and disturbances in gastric mucosal blood flow are potentially involved in the initiation of gastric ulceration. The combination of bile salts and gastric acid is more damaging to the squamous mucosa than the acid alone, so that fasting for as little as 12–24 h presents a risk, increasing as it does, the gastric bile-salt concentration (Berschneider *et al.* 1999).

An alternative explanation, as proposed in the 3rd edition of this book, relies on the fact that when concentrates enter the stomach, the pH remains higher for longer than with hay, so that considerable fermentation occurs. During hay feeding, fermentation is rapidly arrested by the low pH. Ulceration of the non-glandular stomach could be caused by exposure to organic acids in the absence of a thick mucous layer and bicarbonate. The grain-rich diet given to performance horses is subject to fermentation by gastric bacteria producing VFA and lactic acid. A starchy diet and fasting play major roles in causation of gastric ulceration in donkeys, as Burden *et al.* (2009) reported that 41% of 426 aged donkeys at a Sanctuary had gastric ulceration, commonly at the *margo plicatus*. The associated factors for this were: kidney disease, hyperlipaemia and a high soluble carbohydrate diet.

Both HCl and VFA cause a disruption in the bioelectric properties and barrier function of the non-glandular mucosa. At low pH, <4.1, VFAs remain undissociated (pK_a values 4.7–5.0) and are highly lipid soluble. By penetrating the mucosal cells in which the pH is about neutral

they dissociate and the H^+ ions disrupt cellular Na^+ transport, leading to cell swelling, cell death and ulceration (Nadeau *et al.* 2001). Valeric acid even manifested this adverse effects at pH 7.0 (Nadeau *et al.* 2003). *In vitro* studies carried out by Andrews *et al.* (2008) indicated that acetic, propionic, butyric and valeric acids, especially the latter three (lipid solubility is likely to be greater for the longer carbon chains of butyric and valeric acids), in HCl, pH 1.5, caused disruption to barrier function and Na transport of non-glandular gastric mucosal tissues, whereas lactic acid in HCl did not.

Nadeau *et al.* (1999) had previously found that the gastric pH was higher, and there were fewer and less severe ulcer lesions in mares given a diet of alfalfa hay and grain cf. those given bromegrass hay during the initial 5 h after feeding. The high Ca and high protein contents of alfalfa have arrested an initial fall in pH, so that the VFAs remained ionised until they flowed into the duodenum, whereas in the high-Ca brome group there was a lower gastric pH and a greater concentration of butyric, propionic and valeric acids (Andrews *et al.*2005).

High fibre-vegetable oil diets provide a partial solution as corn oil supplementation (45 mL/day) in ponies was associated with a decrease in gastric acid production and increased PGE_2 concentration in gastric juice (Cargile *et al.* 2004), which should increase protective mucus secretion in the glandular region. Lorenzo-Figueras & Merritt (2006) observed no difference between 8% fat or high-carbohydrate (3% fat) meals on the accommodation of the proximal region of the stomach, and subsequent gastric emptying as influenced by of plasma cholecystokinin activity. The NRC (2007) and other workers (Metayer *et al.* 2004; Lorenzo-Figueras *et al.* 2005) conclude that for large cf. small grain meals, intragastric fermentation and VFA production will be greater and extended, because the rate of gastric emptying, measured as a per cent of total meal, will be lower for the larger meal. The gastric contents of roughage diets tend to be more fluid so that the gastric pH will fall more rapidly during a meal, arresting fermentation and limiting the production of VFA.

After ulceration, gastric acid prevents healing of the mucosa and therapy has successfully included H_2-receptor antagonists. Work in the US showed that omeprazole (a proton pump inhibitor) given to TBs at the rate of 4 mg/kg BW per os per day for 30 days followed by 2 mg/kg BW per day for the next 30 days led to a reduction in ulceration of the squamous region of the stomach and improved race performance cf. that from cimetidine (Nieto *et al.* 2001; Johnson *et al.* 2001).Ulceration adversely affects physiologic indices of performance (Nieto *et al.* 2009) and is associated with poor appetite, a dull coat, loose faeces, loss of weight and disinterest in exercise. Horses with gastric ulceration have significantly lower RBC counts and blood haemoglobin than those without the disease (McClure *et al.* 1999; Rabuffo *et al.* 2009).

The absence of some environmental stresses among horses involved in light work or pleasure, or those on pasture, contributes to a lower prevalence. Thus, the absence of frequent stress reactions, together with the lower DM and pH of gastric contents in horses receiving bulky feeds, could be important preventative factors. De Fombelle *et al.* (2003b) found high gastric concentrations of lactobacilli and lactate-utilizing bacteria after a large cereal meal. Although no association of these bacteria with gastric erosion has been demonstrated, it is prudent to reduce the rate of feed consumption and to provide several regular, small, daily meals of an open texture, allowing rapid penetration of the feed by gastric acid with its arrest of microbial fermentation. Moreover, intermittent deprivation of feed, given as hay, for periods of 24 h, causes erosion and ulceration of the squamous epithelium. Gastric ulcers in human subjects are associated with the presence of *Helicobacter pylori*, which is protected from an acid environment by urease secretion. Nevertheless, *Helicobacter* has not been shown to play any role in equine GI tract ailments.

Ill health associated with physical and microbial quality of feeds

The physical quality and hygiene of feeds used for horses are important in maintening good health. If the concentration of moulds, yeasts and bacteria in cereals is high, there is increased risk of digestive disturbances and respiratory disease. A high feed-yeast population might be associated with an increased risk of gastric colic and tympany, while a high feed level of LPS (Enterobacteriaceae) is associated with health disturbance, and *Salmonella* in silage can cause fatal colic (see ‘Silage and haylage and their safety’, Chapter 10, and this Chapter, for botulism). Chopping hay and straw too short can increase the risk of colic, and excessive stickiness of concentrate feeds (e.g. high gluten content) seems to cause gastric colic. Moulds, including ergot, can cause severe respiratory disease (Meyer *et al.* 1986).

Oesophageal impaction (choke)

Greediness, poor dentition, inadequate water, foreign bodies and the consumption of coarse bedding predispose the horse to oesophageal impaction: a foreign object, or feed, lodging in the oesophagus. Normally, the obstruction

will clear after a while, but choke is common in individuals possessing gullets with an abnormal structure. There can be enlargement of the cervical oesophagus. Long-standing impaction can result in chronic damage to the wall of the oesophagus and spasm, causing recurrence of impaction, dysphagia, coughing and regurgitation of food from the nostrils and mouth. Food withdrawal and immediate veterinary action is vital for successful management. In simple cases, sedation and nasal intubation with a small warm water lavage and external massage frequently works, followed by veterinary rehydration with i.v. isotonic fluids. Duncanson (2006) concluded that attempts to pass a nasogastric tube are not justified and that most cases respond to conservative treatment. For cases of more than 48 h duration a cuffed nasogastric tube is advocated, with the possible use of oxytocin.

The obstruction is sometimes caused not by physical impaction of the oesophagus, but by an absence of adequate *free* saliva in the throat. Thus, the faster a horse eats, the more likely is choke to occur. The implication is that softer feed pellets and sugar-beet pulp pellets are more likely to be causes owing to the absence of large amounts of free saliva, in comparison to the effects, in horses with good teeth, of a harder pellet that requires more chewing, or whole cereal grain that does not absorb much saliva.

It has been calculated that the intestinal tract of the average horse holds about 100 L fluid, that in water deprivation – as is induced by choke – are drawn on to maintain homeostasis. If choke prevents feeding for more than 6 to 7 days, dehydration precipitates prerenal azotaemia (raised blood urea). Once the obstruction has been removed, the horse should be watered and fed several times per day with small quantities of wetted nuts or other feed, and stones the size of tennis balls should be placed in the feed box to retard the rate of feed consumption. Some scarring of the oesophagus will have occurred, and this causes repeated trouble. These horses should be given soaked feeds.

Gastric impaction

There is little evidence concerning the causes of gastric impaction, although the ingestion of coarse roughage and inadequate water intake are contributory factors. A stomach tube should be placed to allow the expulsion of gases and permit the administration of antifermentatives such as chloral hydrate or turpentine (an oil obtained from various species of *Pinus*) in raw linseed oil, although surgery may be necessary. Liquid paraffin is given for impactions by nasogastric tube at rates of 2–6 L/500 kg horse once or twice a day for several days, and 0.5–1 L of raw linseed oil (acting as an emollient cathartic) is used in obstinate cases, together with warm salt water to stimulate thirst. For flatulent colic 30–60 mL turpentine is sometimes added to the oil.

Intestinal impaction

The aetiology of ileal impactions is also unknown, although ascarid impactions in foals and tapeworm infestations are less common causes. Large amounts of coarse roughage feeds or excessive cereal intake contribute to the risk, when ingesta might be present in the nostrils.

In cases of impactions of the large bowel, normal gut movement is encouraged by cold-water enemas and massage of any impaction at the pelvic flexure via the rectum. The general use of tranquillizers and pain-killing drugs removes the need for continued forced exercise to prevent the animal from damaging itself and they reduce the risk of a simple impaction becoming a volvulus (twisting of the intestine on its mesenteric axis). Walking sometimes helps to distract the horse's attention where drugs are unavailable or ineffectual. Quick action in mild colic can forestall a more serious derangement precipitating endotoxic shock and death. The most common cause of death in cases of colic is acute circulatory failure secondary to intestinal ischaemia. A strangulating intestinal obstruction was shown to be associated with a mean of 8.45 mmol lactate/L in peritoneal fluid (compared with 2.09 mmol/L in cases of non-strangulating obstruction) together with changes in the appearance of the peritoneal fluid and its chloride and pH values (Latson *et al.* 2005). Epicloic (omental) foramen entrapment is one of the most common causes of small intestinal strangulation in the horse. Tall horses and those with a previous history of colic are at greatest risk, and the condition is also associated with **crib-biting/windsucking**. A decreased risk was shown to exist where the owner provides the care (Archer *et al.* 2008).

Spasmodic colic

In spasmodic colic there is an increase in bowel movement, which can be precipitated by a sudden change of feed, work and chilling. Spasms last for a few minutes or up to a half-hour, and may occur repeatedly over a period of hours, the typical signs being those already discussed (see 'Colic and related disorders', this chapter). Recovery occurs without treatment, but relief of pain and the use of spasmolytic drugs are helpful in amelioration. The colic is associated with increased parasympathetic tone and, from experience, flighty horses are subject to spasms of this kind.

According to Meyer (2001) large intakes of concentrates per meal, little forage and a low standard of feed hygiene are major dietary causes. These conditions stimulate excessive microbial activity in the stomach and duodenum, with gas and organic acids playing pathogenic roles. High concentratrions of lactic-acid in the ileal chyme are pathogenic and are greatest following the feeding of whole grains as oats, followed by barley grain and least by maize grain, although from other evidence whole grain is preferable to ground cereal.

Parasitic worms

Strongyle larvae cause damage to the lining of blood vessels, particularly that of the anterior mesentric artery and its branches, and this can lead to various degrees of occlusion and inhibition of blood flow (ischaemia). Thromboembolism can be a major contributory cause of the complete loss of blood flow to, and death of, a portion of the intestinal tract, leading to obstructive colic. Where the blockage is incomplete, recurrent colic will be experienced. In acute cases, surgery is needed, but thorough worming at 30–60-day intervals will help to contain the situation. A defined dose of specific wormers (Table 11.1), given under veterinary guidance, will have some beneficial impact on the larval stages (which cause a rise in intestinal ALP activity of peritoneal fluid; see Chapter 12). In young horses, impaction of the small intestine with ascarid worms can occur where management is poor, necessitating immediate anthelmintic treatment.

Ileal impaction (ileus) and colonic impaction

Ileal impactions are defined as intestinal obstruction, with the accumulation of fluid and gas, as a consequence of loss of smooth-muscle activity and associated loss of peristaltic movement of digesta. Hillyer & Mair (1997) and Mair & Hillyer (1997) reviewed features of chronic and recurrent colic. Verminous arteritis was one of several causes of recurrent colic. Gastric and small intestinal ileus in the post-parturient mare (Hillyer *et al.* 2008) and colonic impactions are causes of, respectively, acute and chronic colic. The most common gastrointestinal causes of colic in pregnant mares were large colon impaction, displacement and torsion (Steel & Gibson 2001). About 30% of all colics are in the form of intestinal impactions, and, of these, most impactions occur in the large intestine. Impactions are typically located either at points where there is a change in the diameter of the colon or at flexures where it turns acutely (see Chapter 1). More frequent sites are the pelvic flexure and where the right dorsal colon empties into the small colon, but occasionally the sternal and diaphragmatic flexures and the ileocaecal valve are involved. The closer to the ileum the large-intestinal blockage occurs, the more dangerous it is, as it will severely restrict water resorption in the caecum and ventral colon, and this can lead to dehydration and hypovolaemic shock.

Duodenitis-proximal jejunitis is an idiopathic syndrome of the small intestinal ileus, associated with microscopic changes to the duodenum and proximal jejunum, characterised by copious nasogastric reflux. Horse with the condition are more likely to have been given concentrates or to have been grazed on pasture than not and mares are slightly more prone to the disease (Cohen *et al.* 2006). Clinical signs of post-operative ileus are related to small intestinal dysmotility with reflux of small intestinal contents into the stomach, moderate colic, absence of intestinal borborygmi, but with persistent tachycardia (Merritt & Blikslager 2008). Significant amounts of 5-HT can be released into the systemic circulation through massive release of platelet-stored 5-HT in horses with a compromised bowel. 5-HT is a potent proinflammatory, vasoconstrictive and immunomodulatory agent. This increased systemic release could play a role in the pathophysiology of ileus, as there is rapid and prolonged **tachyphylaxis** for the jejunal 5-HT_{IA}-like receptors (Delesalle *et al.* 2008).

The horse suffering colonic impaction will frequently look at its flank, emit no intestinal sounds and void small, mucus-covered droppings; palpation of the impaction is frequently possible. Inadequate water intake, excessive sweating and hard exercise together with excessive amounts of coarse roughage are also thought to be contributory factors. An association with positive *Salmonella* spp. faecal cultures has been observed, which can indicate a reaction to bowel inflammation. Impactions of the caecum are also related to endotoxaemia. Impactions are fairly common in old horses with bad teeth restricted to poor-quality hay with little water after experiencing lush grass. Impaction in the small colon also occurs in foals, 2 to 6 months of age, when roughage feeding is initiated. Veterinary treatment for small colon impactions includes introduction of gentle warm water enemas with a lubricated nasogastric tube through the rectum. Solid food or water, given orally, in cases of small-intestinal ileus, will aggravate gastric distension and should be withheld. Isotonic i.v. fluid treatment is indicated, together with intestinal lubricants. Surgical intervention will be necessary if obstruction is complete.

Sand colic

Sand impaction is a common cause of colic in areas with sandy soils. Sand build-up in the GI tract has been respon-

sible for up to 30% of colic cases in sandy areas of the Southern US. Husted *et al.* (2005) listed risk factors for sand-related GI disease in Icelandic horses: both excessively short and long grass in combination with a sandy soil, cf. a clay soil, and feeding on the ground where the grass was very short. Sand accumulation is associated with chronic inflammation and poor motility of the intestine. The cranioventral abdomen is the common location for its accumulation – a difficult area to evaluate other than by radiography or ultrasonography. The mean intake for eight individual mature horses varied from 2.5 to 272 g per day over 5 days when they were each offered sand in a bucket. Its elimination from the GI tract is assisted by the consumption of hay (Lieb & Weise 1999; Weise & Lieb 2001). The daily supplementation of clinically normal horses with a probiotic, prebiotic, and psyllium, enhanced faecal sand clearance (Landes *et al.* 2008).Psyllium is a hydrophilic mucilloid and is effective when given at the rate of 400 g/500 kg BW daily for three weeks, divided among three daily feeds. Flavoured psylliums are available for reluctant patients. Over the period it should remove most of the sand. This treatment might require repeating every 4–12 months. If psyllium treatment is not continued, complete recovery and normal motility may not occur, so that ultrasonography should be adopted in an assessment (Korolainen *et al.* 2003). Hotwagner & Iben (2008) observed a higher total ash output during a 5-day treatment when psyllium (0.5 kg twice daily) and mineral oil (2 L/day) were used cf. mineral oil (2 L/day) alone. The problem can frequently recur and is associated with periods of inappetence, diarrhoea, anxious pacing up and down with groans on lying down, pawing the ground or a crouched stance with a turned head. Sand can be present in the droppings. Horses likely to eat sand should be kept on a thick sward and hay should not be offered on the ground, but from racks, in order to reduce the risk of recurrence.

Intraluminal obstruction can also result from concretions of hair and of plant material, and combinations of these, and from the horse chewing objects in its environment. These concretions typically occur in the small colon and form as a mineral precipitate on the surface of the material during passage through the colon, over an extended period of time, before obstruction occurs. The clinical signs include abdominal discomfort, distension and straining to defecate.

If the horse has a predilection for chewing material in the surroundings a change in environment might reduce boredom and eliminate the habit.

Horses with clinical signs of colic and of endotoxaemia had high serum activities of CK and AST and were found to have severe ulcerative colitis, pyloric ulceration and stenosis with colonic sand impaction and obstruction owing to faecaliths. Their semi-membranosous muscles had scattered acute to subacute segmental necrosis of the myofibres, suggestive of endotoxin-induced muscle injury (Valentine & Löhr 2007). Occasionally enteroliths (large stones) are formed, apparently on a nidus of ammonium magnesium phosphate (struvite). This gradually enlarges, and its removal requires surgery. Hassel *et al.* (2004, 2009) detected increased alkalinity and increased levels of N, Mg, P, S, Na, Ca, and K in the colonic contents of horses with enteroliths. Horses fed alfalfa hay had lower colonic Na and K and higher Ca, Mg, P and S concentrations, and a more alkaline pH than those fed grass hay (Hassel *et al.* 2009). A higher pH and higher mineral content that might favour deposition of struvite, is associated with alfalfa hay (Hassel *et al.* 2004). NRC (2007) recommends that horses with a history of enterolithiasis are fed grass-, rather than legume-, hay, with the exclusion of wheat bran, and that 220 mL apple cider vinegar are given twice daily with a turnout to pasture.

Foal colic

Foal colic is very common in the first two days after birth, and is caused by the meconium blocking the large intestine at various levels. Lubrication of the impacted mass with orally administered liquid paraffin (200 mL) or glycerol, the use of enemata of soap and water and the relief of discomfort are normally sufficient remedies. If no response is registered within a few days, volvulus or intussusception is suspected. Abdominal pain can also occur at this age through rupturing of the bladder, which is effectively repaired by surgery, and in older foals discomfort coincides with the eruption of permanent teeth. Umbilical hernias can cause colic in young foals, but these usually correct themselves by 6–8 months of age. Inguinal hernias, especially in colts, have similar effects.

Lavoie *et al.* (2000) describe a transmissible enteric disease of foals and other species, caused by *Lawsonia intracellularis*, with faecal transmission. Signs include depression, rapid weight loss, subcutaneous oedema, diarrhoea and colic. The protection given by colostrum seems to be important.

Oral and dietary treatment

Unless one is very familiar with the sequence of events in a particular horse, veterinary help should be sought immediately signs of colic are observed. Feed should be removed until the cause has been determined, but clean water should be provided. If the animal shows signs of injuring

itself through violent actions, the horse could be walked; otherwise leave it alone in a box, free from projections or structures that might endanger it. In all colic cases the horse should be kept warm in cold weather and during recovery a warm bran mash is helpful. The general use of a mineral oil or a kaolin–pectin paste is safe and will accelerate the expulsion of offending masses. General treatment also has the objective of preventing rupture of some part of the GI tract, or displacement of its parts by control of pain and tympany, by evacuation of the bowels, by arresting rapid bacterial fermentation and by re-establishment of normal peristalsis. An enema of 9–13 L (2–3 gallons) warm soapy water is frequently given in addition to the lubricant mineral oil for intestinal impactions. A mild soapy-water enema is particularly helpful in cases of colonic impactions in foals.

Electrolyte disorders are common in horses with acute abdominal disease and are a cause of mortality. Borer & Corley (2006a,b) discuss the prevention and treatment of hypo- and hyper-kalaemia, -magnesaemia, -calcaemia, -natraemia, -chloraemia and -phosphataemia. Where there is impaction of the large colon, or caecum, oral fluids, including solutions of electrolytes and glucose, are allowed, but solid food should not be given until the impaction is passed.

From the dietary point of view, therapy includes withholding normal feed, but allowing access to water if there is no nasogastric reflux. Rehydration of the horse both per os (in the absence of reflux) and i.v. is essential, and laxatives are normally given by nasogastric tube. With severe intestinal impactions, a 500 kg horse should be given 6 L fluid every 2 h through an indwelling nasogastric tube. Mineral oil can be administered to facilitate passage after the impaction begins to resolve. Following resolution of the impaction, and in the absence of perforations, feeding should be resumed gradually to avoid an immediate recurrence, with the return to normal GI function. Bran mashes are given by preference for the first 24–48 h. Following this, leafy hay is introduced gradually, along with concentrate mashes, or short grazing spells on a halter. Forage chopped very short is said to increase the risk of ileal obstruction. With prolonged dysfunction, partial or total parenteral nutrition is required.

Gas or flatulent colic and its treatment

Gas colic is extremely painful and can be secondary to an obstruction or an impaction. Distension of the small bowel is rarely noticeable and if the abdomen is unusually large, tympany of the ventral colon might be present. Sometimes, on *post mortem* examination, several regions of the GI tract appear to be involved. Impaction and lack of movement of the intestines inhibit expulsion and minimize absorption of gas into the blood. The latter route of removal is more important than might be realized, as about 150 L carbon dioxide and methane can be absorbed daily from the intestinal tract.

If gastric tympany occurs, the condition becomes evident within 4–6 h of eating. Intubation with a nasogastric tube to relieve pressure is essential to prevent rupture of the stomach. A warm 4% salt solution, which decreases the viscosity of the fermenting mass and encourages water consumption, is sometimes administered in small quantities at intervals by allowing it to drain into the stomach through the tube. The horse may adopt a typical dog-sitting attitude, or stand without moving its feet, especially where a rupture or a serious intestinal infarct exists. Perforating ulceration of the small intestine was detected by paracentesis, yielding abnormal peritoneal fluid in 5 adult horses with signs of low grade colic, depression and inappetance (White *et al.* 2008). After stomach rupture, in particular, ingesta is sometimes observed in the nostrils. With different intensities of gas colic the horse can feel cold, despite experiencing a fever, it exhibits congested mucous membranes of the eyes and has a sour and vinegary breath. Powerful analgesics prevent violent rolling and self-inflicted trauma. Where there is risk of violent action the horse should, if possible, be kept on its feet, but quiet horses are best left alone.

Gas colic is frequently the sequel to a rich diet of cereals, or lush legumes, or even to the inadvertent consumption of a pile of grass cuttings. Quick action is essential, as again violent reactions on the part of the horse can lead to ruptures or to twisting of the gut, with a poor prognosis.

Colic associated with torsion, twists and rotations

Torsion (rotation on its own axis), volvulus (twisting on the mesenteric axis) and intussusceptions (infolding), usually of the terminal ileum into the caecum, all require immediate surgery, and the prognosis is often poor. The membranes of the eyes and lips are typically dry and pain induces a rapid rise in pulse and respiratory rates so that the loss of carbon dioxide causes alkalosis despite a raised blood lactate. The value and risks associated with post-operative parenteral nutrition have been assessed by Durham *et al.* (2004).

Other colics

Abdominal pain is not the reserve of the intestinal tract and can result from bladder and kidney stones,

urinary infections, pericardial effusions and sometimes liver disease.

Predisposing factors in colic

The following factors may predispose a horse to colic:

- 'overheating' – sudden access to large quantities of cereals or stands of green clover and lush grass;
- stress, caused by changes in routine or environment;
- irregular work, horses standing idle on full feed or changes in the timing of feeds;
- working a horse on a full stomach. Even during protracted slow work large feeds should not be given and bulk feeds should be excluded. Bulky feeds should be given in the evening, after the digestive powers have been restored by rest;
- work itself can precipitate colic, especially towards the end of an exhausting day – both feed and work should be regular. Extended work should be interrupted by short rests every 2–3 h when a few mouthfuls of concentrated feed and water are provided;
- the consumption of excessive amounts of cold water after severe and hot work before the horse has cooled down and/or providing heavy feed at this time;
- insufficient good-quality roughage or mouldy corn and mouldy silage;
- large quantities of cut green feed;
- unfit horses changing abruptly to an increased work rate and a concentrate-rich diet;
- failure to provide fresh clean water at all times;
- lack of teeth care. 'Quidding' is noticed in which small balls of partly chewed feed are dropped into the manger. This is usually associated with cheek teeth that require rasping;
- greedy feeders that bolt feed, or greedy bullies in group-fed herds; and
- inadequate work control.

Prevention

In general, adherence to the following should prevent occurences of colic:

- Each horse has its idiosyncrasies; before a new horse is placed gradually onto a rich working diet its habits and particular requirements should be studied and understood.
- Hard-worked animals should receive a small feed of concentrates at frequent and regular intervals, and the time of meals should be constant, even at weekends.
- Increased demands for energy should be met by an increased feeding rate of concentrates of no more than 200 g/day in a 500 kg horse and a proportionately lesser rate of increase in smaller horses.
- A sensible and regular exercise programme should be instituted.
- Where a horse is changed from one stable to another, its old routine should go with it and, if necessary, be changed gradually.
- All animals should be checked last thing at night.
- Only good-quality roughage, free from contaminating weeds, and, in the US, also free from blister beetles, should be provided.
- Stores of horse feeds and dangerous chemicals should always be held in rooms the doors of which cannot be opened by horses.
- No horse should be overworked and, following strenuous work, no substantial feed or water should be given until the animal is cool and rested and then should only be given in moderate amounts.
- Cribbers or wind suckers (Plate 11.1) should be fitted with cribbing straps, or, if cribbing is known to precipitate colic in the individual, surgery should be considered.
- Teeth should be inspected at regular intervals (see Chapter 1). It may be necessary to file the upper and lower molars and premolars or remove teeth with infected roots (decaying teeth can be inferred from excessive salivation).
- Individuals that bolt their concentrate ration should have it mixed with chaff or dry bran. Place stones the size of tennis balls in the manger to retard the rate of feed consumption.
- A proper worming programme is essential in all horses and a pasture rotation system should be instituted where horses have access to grass.

Overfeeding

In addition to the consequences discussed in this chapter, overfeeding can have a number of other deleterious effects in horses. Some of the more obvious are listed below:

- Obesity: said to present difficulties at foaling in mares, to affect the work rate, to accelerate the onset of fatigue and to be asociated with insulin resistance (see Insulin resistance and laminitis, this Chapter);
- Obese horses that suddenly experience food deprivation, can become anorexic secondary to colic, which sometimes causes hyperlipidaemia. Pony mares in late pregnancy or peak lactation and subject to pasture changes



Plate 11.1 A crib-biting mare – crib-biting (or wind-sucking) is a vice in which the incisor teeth grip a solid object, the horse pulls down and swallows gulps of air. Sometimes a leather strap is fastened snugly around the neck just behind the jaw to deter the horse from the practice.

are prone to this problem. Treatment can be problematic and certainly requires veterinary advice. Anorexia is also a sequel to acidosis;

- The overfeeding of young horses, in particular colts, where food is given in separate and discrete meals, and/or where the ration is unbalanced in respect of its mineral content, can cause bone disorders. The principal one is epiphysitis – typically of the distal epiphyses of the radius, metacarpus, tibia and metatarsus – recognized by bony enlargements and 'lipping' of the physes;
- Contracted tendons (see Chapter 7) may be associated with overnutrition (energy-rich feeds in late pregnancy producing too much milk), and/or overfeeding of the foal and yearling; and
- Enterotoxaemia: in young horses fed in groups, enterotoxaemia is occasionally precipitated in the largest and most aggressive individual. A flatulent colic occurs in which the intestines are loaded with rich feed and gas. Signs of dyspnoea and subcutaneous oedema are presented and the cause is often a rapid proliferation of the bacterium *Clostridium perfringens*.

Hyperlipaemia

Hyperlipaemia is a clinical condition of which the subclinical form is known as hyperlipidaemia. In the horse, most of the circulating fat is in the form of VLDL, of hepatic origin, especially in the post-absorptive state of hyperlipaemia. The clinical disorder is characterized by depression, anorexia, elevated plasma TAG concentrations, lipid infiltration of the liver and hepatic failure. In large horses azotaemia also occurs. Various physiological stresses, fasting, obesity, pregnancy, lactation and depressed feed intake predispose horses to the disease. Gupta *et al.* (1999) kept donkeys and mules off dry feed for 10 days. Serum became cloudy by the seventh day, although the donkeys were more adaptable than the mules and recovery occurred by the sixth day of re-feeding. The difference in susceptibility to the condition between the donkey and the pony on the one hand, and the horse on the other, could be caused by a greater frequency of insulin resistance amongst ponies and donkeys. Insulin is required for the activation of LPL to clear TAG from the blood into adipocytes.

Starvation depletes body stores of the minerals P, K and Mg, and alfalfa is the basis of a good diet for recovery. Whilst the addition of corn oil to alfalfa dampens the insulin response, the addition of excessive oil can jeopardise Mg status and recovery of Mg reserves (Stull *et al.* 2001). Tube feeding with readily digestible carbohydrate in relatively small amounts, initially, can begin if the animal will not eat voluntarily. Insulin therapy (monitoring blood sugar) is frequently used to promote

Table 11.3 Glucose and insulin therapy for hyperlipaemia.

	Day 1	Day 2	Day 3	Day 4	Day 5
Insulin (iu/kg) intramuscularly twice daily	0.15	0.075	0.15	0.075	0.15
Glucose (g) per os twice daily	100		100		100
Glucose (g) per os once daily		100		100	
Heparin (iu/kg) intramuscularly twice daily[1]	40–150	40–150	40–150	40–150	40–150
Sodium bicarbonate and fluid[2] i.v.					

[1]Blood clotting ability should be ensured and maintained, especially if larger doses are used. Pharmaceutical doses of heparin release lipoprotein lipase into the blood.

[2]Blood acid–base status should be checked first for a base deficit. PCV and blood urea should be measured and elevated values rectified with Ringer's solution.

carbohydrate metabolism and fat clearance and to reduce the activity of intracellular lipase, which mobilizes stored fat to form VLDL (Table 11.3).

The disorder can lead to organ failure and has mortality rates of 65–80%. The aetiology is apparently different from the condition in man, where the function of the enzyme LPL is impaired. In the horse, overproduction, rather than defective catabolism, of VLDL is the cause of hyperlipaemia. Plasma VLDLs are elevated in hypothyroidism (Frank *et al.* 2004). Treatment should therefore be directed towards use of lipid-lowering agents that reduce VLDL synthesis. Hyperlipaemia in the pony is accompanied by an elevation in plasma FFAs, without ketosis, which provides the stimulus for TAG synthesis and the secretion of the VLDLs. There is scope for the prevention and/or treatment of ponies by lowering this hepatic VLDL production with agents such as niacin, as this reduces adipose tissue lipolysis and FFA flux (Watson *et al.* 1992).

PASTURE AILMENTS

For equine motor neuron disease and equine degenerative myeloencephalopathy, see Chapter 4.

Botulism (forage poisoning in adult horses and shaker foal syndrome in foals)

Botulism amongst horses has increased this century in Europe, owing to the popularity of haylage. Characterized by dysphagia, weakness and progressive flaccid paralysis, botulism is caused by the exotoxin of *C. botulinum* that interferes with the release of acetylcholine at the neuromuscular junction. Wound botulism has rarely occurred in horses. In adult horses the disease is caused by the consumption of the toxin, but in young foals the toxin can be elaborated by organisms present in the GI tract. See also 'Silage and haylage and their safety' and 'Equine dysautonomia', Chapter 10.

Treatment of infected foals and adults is difficult and requires intensive care with the use of a specific antitoxin. This treatment will not reverse the effects of toxin already bound at the presynaptic membrane and it should be introduced before recumbency occurs. Tube feeding is necessary, together with measures to prevent aspiration pneumonia, constipation, corneal ulcers and gastric ulceration. Mineral oil can be added to feed if constipation occurs. Foals are optimally protected by vaccinating the pregnant mare, with the annual booster given one month before foaling. This will protect the foal until two to three months of age. The foal should receive the three-dose toxoid series starting at two months of age. A recombinant vaccine (HcBoNT/C) shows fewer adverse reactions, but induces similar concentrations of neutralizing antibodies against botulism neurotoxin C compared with that produced by a commercial toxoid vaccine (Frey *et al.* 2007).

Seasonal myopathy

A frequently fatal atypical seasonal **myopathy** has emerged in several European countries. It occurs, especially in autumn and spring, in horses that have been on pasture for at least a week, and it can develop during the following days in a box. Pastures with a stream/river containing dead leaves increase the risk – a piped water supply is protective. The mechanical harrowing of pasture to spread manure should be discouraged. Prevention is aided by giving regular anthelmintic treatment, supplementing pastured horses with a salt block and a concentrate rich in carbohydrate, and by avoiding the distribution of hay on the ground (Van Galen *et al.* 2008).

Yellow fat disease

Yellow fat disease, or steatitis, is a generalized disorder of fat depots characterised by extensive adipose cell degeneration and inflammation, causing hard painful swellings, with progressive peroxidation of unsaturated fatty acids. Lipofuscin pigment is responsible for the yellow discolouration. The disease occurs typically in anorexic pastured cold Shetland ponies and cold-blooded horses when the tissue concentration of antioxidants is insufficient. Elevated serum LDH, CK and AST activity is a typical occurrence and elevated rectal temperature and heart rate are frequent observations. Prognosis is poor, unless antioxidant treatment can be instituted at an early stage (de Bruijn *et al.* 2006).

Idiopathic colitis, malabsorption and chronic diarrhoea

The causes of malabsorption and maldigestion in horses were reviewed by Mair *et al.* (2006). The most common causes of malabsorption in adults are infiltration of the intestinal mucosa and submucosa by inflammatory or neoplastic cells, while in foals infectious agents causing enteritis are more likely. The most common clinical sign is chronic weight loss, associated with low plasma albumin and protein-losing enteropathy of either the small or large intestine. The loss of integrity of gut mucosa associated with protein-losing gastroenteropathy commonly causes chronic diarrhoea, and will reduce the net absorption of digestion products. Diet should be of the highest quality and rich in essential nutrients. Water and electrolyte losses in young stock suffering from diarrhoea can lead to a rapid decline in vigour unless lost fluid is continually replenished.

Severe colitis has been produced in ponies by oral treatment with the antibiotics clindamycin and lincomycin, followed by the intestinal contents of horses dying from naturally occurring idiopathic colitis (Prescott *et al.* 1988). Treatment of three further ponies with lincomycin on its own at the rate of 25 mg/kg BW twice daily for 3–5 days caused death. A clostridium closely resembling *Clostridium cadaveris* was isolated from the colon of each of these ponies and from one of six horses dying from idiopathic colitis, but not from horses with non-fatal diarrhoea. These data indicate the importance of maintaining a large mixed culture of symbiont bacteria in the large intestine by mixed feeding, which prevents potential pathogens achieving rapid unrestricted growth.

Chronic diarrhoea is normally of large-intestinal origin and results from some upset to the normal balance of the intestinal flora. It can follow stress, such as the prophylactic use of oxytetracycline or some other antibiotic. Where the diarrhoea is of small-intestinal origin, it can be connected with a lack of certain digestive enzymes detected by xylose and other tolerance tests (Chapter 12). It should also be remembered that the adult horse loses the ability to digest lactose adequatedly when about three years old. Large intakes of milk sugar after this time induce diarrhoea. Chronic diarrhoea can also be prompted by parasitism, mesenteric abscesses or some disorder of vital organs.

Salmonellosis, colitis and many other causes of diarrhoea are associated with rapid transit of digesta through the large intestine. This means that fibre digestion is impaired and that the efficiency of reabsorption of water and Na and K ions is depressed (see Chapter 1). Hyponatraemia, hypochloraemia and hypokalaemia occur. Blood should be monitored and blood acid–base balance determined, so that the appropriate electrolyte drinks and fresh water, can be given. Johns *et al.* (2009) examined adult horses with diarrhoea, finding 29% with positive blood cultures, which reduced survival rate. Organisms isolated included *Corynebacterium* spp., *Streptococcus* spp., *Pantoea agglomerans*, *Bacillus* spp. and yeast.

In the absence of these conditions and of inclement weather, it is feasible to turn the horse out to sheltered pasture, as green herbage is a good source of electrolytes. Remember that bacterial diarrhoea causes contamination of the pasture for a period.

Acute diarrhoea

Diarrhoea in the adult horse normally indicates colonic dysfunction and is typically absent from those with lesions limited to the small intestine (Chandler *et al.* 2000). Severe diarrhoea is frequently caused by *Salmonella* infection, precipitated by stress of transport, and, particularly, by strongyle worm infection. The prevalence of faecal shedding of *Salmonella* spp. by horses in the United States is apparently 0.8% and the prevalence of *Salmonella* spp. in grain or other concentrate used for horse feed is 0.4% (Traub-Dargatz *et al.* 2000). Antibiotic treatment to eliminate *Salmonella* is of questionable value and in fact oxytetracycline can trigger the onset of the infection. Salmonellosis occurs in closely stocked groups after mild winters and is associated with heavy worm burdens. It is transmitted to foals by adults that are asymptomatic carriers. Suspected cases should be isolated in a box with a very strict programme to ensure that contaminated faeces are not transmitted to other stock. The bacteria are not continuously excreted in the faeces, and careful veterinary examination is necessary to detect any carriers that are

shedding the organisms. Feed and rodents are also reservoirs of potential infection. (See also Chapter 7.)

All cases of acute diarrhoea are associated with a critical loss of fluid, K, Na and Cl, and unless replacement therapy is quickly instituted, the consequences are rapidly fatal in young stock. Appropriate fluids are listed in Table 9.3. Fluid losses of 20–50 L, and deficits of Na 92000–6000 mmol), K (700–3000 mmol), and bicarbonate (1000–2000 mmol) occur, and should be made good. In acute diarrhoea, where there is hypotonicity and dehydration, the use of hypertonic solutions yields an immediate response. Where the animal is dehydrated and hypertonic, hypotonic solutions are given. The normal plasma values for Na, K, Cl and bicarbonate in horses are, respectively, 139, 3.6, 99 and 26 mmol/L.

Foals experience absolute losses amounting to 15–20% of that of the adult. Neonatal foals become hypovolaemic extremely rapidly with gastrointestinal disease. The accompanying low arterial pressure is not always accompanied by an increased heart rate and response to fluid and electrolyte administration is different from that in adults, as it can lead to hypernatraemia, owing to immature fluid and electrolyte homeostatic mechanisms. Hollis *et al.* (2008b) showed that the hormonal responses of mature and neonatal horses are indeed different during hypovolaemia and following fluid resuscitation.

Foal-heat diarrhoea

Foal-heat diarrhoea typically occurs at the time the dam's first postpartum oestrus is expected, but no correlation has been established between it and the chemical composition, bacterial count or oestrogenic activity of mares' milk (Urquhart 1981). The diarrhoea is normally self-limiting in 3 or 4 days, and is probably a secretory diarrhoea. Hypersecretion in the small-intestinal mucosa overwhelms an immature colon unable to compensate by increasing fluid and electrolyte absorption. If a prolonged diarrhoea occurs, fluid and electrolytes should be replaced.

Dehydration and potassium status

Although the carcass of a 500 kg horse contains by weight only 15% as much Na or K as of Ca, on average the 1100–1200 g K are subject to a much greater flux than the Ca, owing to its higher solubility in tissue fluids. The volume and water content of muscle and red cells are modulated primarily by the control of their Na^+ and K^+ content. The cell membrane is relatively impermeable to small cations, which diffuse slowly, whereas small anions diffuse freely; haemoglobin, acting as a large anion, remains as an intracellular entity. However, the equilibrium distribution of charged particles between red cells and plasma differs from that predicted by normal diffusion processes. The slow passive movements of Na^+ and K^+ are balanced by an active outward movement of Na^+ and an inward transport of K^+ in each cell, mediated by several hundred discrete pumps fuelled by ATP. If the glycolytic mechanisms which yield ATP break down, or if the cell membrane is damaged such that diffusion leakage increases, there is a decline in resting membrane potential and the pump mechanism is incapable of maintaining a physiological cellular environment. An intact glycolytic pathway in red cells is necessary for the maintenance of a physiological cation distribution between cells and plasma, as has been amply confirmed in numerous experiments.

It is clear that the horse must maintain cellular K within strict concentration limits to maintain normal health. The measurement of plasma or serum K^+ concentrations, as a guide to body K status, is misleading. Measurements of large numbers of horses have failed to detect any correlation between serum and cellular concentrations of K^+ (Frape 1984b; Muylle *et al.* 1984b) and serum contains, on average, only 3.7–4.3 mmol K^+/L, that is, 3.8–4.4% of the mean concentration in red cells. In fact, the total body ECF contains only 1.3–1.4% of total body K. In maximal anaerobic exercise, serum K^+ tends to increase, whereas it has a tendency to decrease in endurance work without comparable changes in the cells.

In severe diarrhoea, the bodily loss of K by a 500 kg horse approaches 4500 mmol (175 g), associated with a fall in red cell K^+ from 97.5 to 75 mmol/L, without significant change in plasma K^+ (Muylle *et al.* 1984a). Protracted work in hot weather apparently leads in a horse of this size to losses of K^+ and Na^+ of 1500–1800 and 4000–5000 mmol, respectively. Thus, the measurement of red-cell K^+, the concentration of which appears to be well correlated with that of muscle-cell K^+ (Carlson 1983b), is recognized as a more reliable means of assessing K^+ status.

A fall in red-cell K^+ concentration below 81 mmol/L is associated with weakness of skeletal and smooth muscle, tremor, and, in severe depletion, with recumbency, cyanosis and eventually respiratory and heart failure. The K^+ ions released from muscle cells during hard exercise act as potent arteriolar vasodilators and stimulate cardiorespiratory reflex activity. Thus, there is a close correlation between the extracellular increase in K^+ and the increase in both muscle blood flow and oxygen consumption, and therefore in performance.

The blood flow is insufficient in K^+ depletion, precipitating hypoxia, anaerobic glycolysis and metabolic acidosis. This turn of events is pathological, and the ensuing

damage to muscle-cell membranes leads to further cellular K^+ loss, which cannot be restored by a Na^+/K^+ pump deficient in readily available energy. Investigations in Belgium (Muylle *et al.* 1984b) have revealed an anomalous situation in which about 10% of 436 horses examined possessed a normal red-cell K^+ concentration distributed independently of the remainder. Their mean red-blood cell K^+ concentration was 83.8 mmol/L, some 13.7 mmol less than the normal values for the other 90%, despite similarities in management and diet. Other studies by the same group (Muylle *et al.* 1983) indicated that, in a smaller sample of 43 horses, 11 were in this low red-cell K^+ range, and of these, nine were performing unsatisfactorily on the racetrack and had a more nervous temperament. An intriguing observation was made by Hess *et al.* (2003), who provided a concentrate low in K, cf. a K-rich mix, for horses subsequently participating in an endurance ride. This caused them to be less dehydrated, with higher plasma Na^+, during the ride, so maintaining plasma volume and blood pressure. Thus, the ideal composition of electrolyte mixtures for endurance rides needs further examination.

A dietary K deficiency is unlikely to be more than an occasional cause of K^+ depletion, although several investigations implicate dietary Mg deficiency as a cause. A 500 kg horse given a diet composed of a 50 : 50 mixture of grain and hay, absorbs daily 90–100 g K^+, well above the level required for maintenance. Excess dietary K is rapidly excreted so that a generous dietary content is of no avail in the acute K^+ depletion of diarrhoea, or of abnormally high sweat loss when, in any event, appetite is depressed. Dosing with an appropriate solution is the only reasonable approach to overcome the worst of the deficit.

K in excess is a moderately potent toxin to heart muscle so that only limited quantities should be given i.v., and cardiac action must be monitored. Intravenous infusion rates of 11.5–13.7 mmol KCl/min bring about plasma K^+ concentrations exceeding 8 mmol/L and consequential cardiac arrhythmias and abnormal electrocardiograms, through a transient but excessive alteration to the gradient of transmembrane K^+ transport (Epstein 1984). The bulk of such dosage must be given orally or by nasogastric intubation. By this route, Muylle *et al.* (1984a) administered a solution containing glucose (50 g/L), a commercial amino-acid mixture (0.05 L/L), KCl (5 mmol/L), $CaCl_2$ (3 mmol/L) and made isotonic with NaCl, which was partially replaced by Na acetate according to the acid–base balance of the horse. The quantities given daily were proportional to the deficit calculated from red-cell K^+ values (see Chapter 12 for assessment of red-cell K).

Malabsorption of fat-soluble vitamins

Occasionally the efficiency of absorption of vitamins A, D, E and K is reduced and the most frequent cause is an interruption to biliary flow by obstruction of the bile duct. The immediate effect is a failure in the blood-clotting mechanism, but this can be overcome by injections of vitamin K. Vitamin K administration is also successful in counteracting the bleeding syndrome caused by dicoumarol poisoning. Plant sources of this poison are referred to in Chapter 10.

Urticaria

The horse, especially the TB, is prone to urticaria, a common skin condition. Feed proteins, or peptones, especially from feeds containing gluten, are frequently implicated. They are absorbed from the gut in sufficient quantities to stimulate an immune response with circulating antibodies, although these are sometimes detected in otherwise healthy horses. A single insect bite can also cause generalised urticaria in a sensitised horse. Hypersensitivity to drugs (penicillin, clenbuterol and especially phenylbutazone, either the drug itself, the carrier or preservative) is a frequent cause. The clinical signs are raised oedematous plaques of varying size (0.5–10 cm diameter) over the body surface. The best treatment is avoidance of the allergen, or administration or dexamethasone i.v., 0.04 mg/kg BW (Pilsworth & Knottenbelt 2007). (See also Chapters 5 and 12.)

Mycotoxins

Depending on the species of the mould, mycotoxicosis may cause digestive disturbances, liver and kidney damage, nervous symptoms, infertility and abortions. The aetiology of several mycotoxin infections has been discussed in Chapters 5 and 10.

Grass sickness (equine dysautonomia)

Signs similar to those of choke are present in grass sickness, but stomach distension is frequently involved, as failure of the normal function of the stomach and of the oesophagus results from neural impairment of the muscles controlling the contraction of these organs. Nasal return of ingesta indicates such oesophageal impairment. Neurotoxins are apparently a cause (see Chapter 10 for current evidence).

Ammonia toxicity

Ammonia toxicity is much more likely to occur in the ruminant than in the horse, when the source of ammonia is urea taken orally. The reason for this is that the

breakdown of urea to ammonia requires the intervention of urease, which is not found in mammalian tissues, but is secreted by protein-degrading bacteria in the gut. In the horse, any dietary urea would be absorbed into the blood before it was significantly degraded by these bacteria. With grossly excessive protein (or urea) intake, or when intestinal haemorrhage occurs, protein (or urea) is bacterially deaminated to the extent that the portal system and the liver are overloaded, and ammonia spills over into the systemic circulation. Otherwise, the liver will 'mop up' ammonia in urea formation, or in transaminations. (See also Chapter 2 for treatment.)

Where there is hepatic dysfunction, as in pyrrolizidine poisoning and portocaval shunting of blood, the ammonia enters the systemic circulation. Its disposal through the kidneys is arrested by renal pathology. The kidneys would otherwise tend to combat metabolic acidosis by secretion of H^+ ions and ammonia into the tubules, where they combine to form ammonium ions, with reabsorption of Na ions:

$$H_2CO_3 \rightarrow HCO_3^- + H^+ \rightarrow Na^+ + Cl^- + H^+ + NH_3 \rightarrow NH_4^+ + Cl^- \text{excreted, with } Na^+ \text{ reabsorbed to neutralize } HCO_3^-$$

Ammonia toxicity has also been recognized in rare cases of unknown aetiology, where equine liver function is normal.

The signs of ammonia toxicity include head pressing, blindness, usually abdominal pain and varying degrees of maniacal behaviour, ataxia and depression. Encephalopathy is the major cause of the behavioural disturbance, which can return to normal following early treatment. Appropriately collected and quickly tested blood samples reveal metabolic acidosis, low plasma bicarbonate (10–15 mmol/L) and hyperammonaemia (150–400 µmol/L). Where liver function is normal, hyperglycaemia (15–24 mmol/L) and haemoconcentration have been observed, otherwise, with liver dysfunction (detected by raised liver enzymes, bile acids and bilirubin and depressed blood albumin) hypoglycaemia is likely. Blood urea might, or might not, be elevated and blood albumin can be depressed when there has been protein-losing enteropathy without liver dysfunction.

When the source of the ammonia is the intestine, bacterial action is partly arrested with nasogastric neomycin treatment, and polyionic fluids given to counteract dehydration. Sodium bicarbonate should be reserved for the most severely acidotic cases and given by slow i.v. infusion, as rapid correction of acidosis increases the intracellular movement of ammonia. K^+ treatment counteracts the toxicity of ammonia on neuronal cell membranes, and is given as i.v. potassium chloride (10 mEq/h) while monitoring heart rate.

Protein-losing gastroenteropathy

The condition of protein-losing gastroenteropathy is characterized by weight and muscle tissue loss, lethargy and diarrhoea, caused by leakage of plasma proteins into the lumen of the GI tract. There is a loss of integrity of the mucosa of the GI tract through gastric, or colonic, ulceration, GI parasitism and enteritis caused by various bacterial infections. It is crucial that the cause of the protein-losing condition is determined and that the appropriate veterinary therapy is instituted. Virtually all the albumin and fibrinogen in plasma and 60–80% of the globulins, are synthesized in the liver (the remaining γ-globulins are mainly formed in the plasma cells of lymph tissue). Globulin synthesis is faster than that of albumin, which has a longer half-life ($t_{1/2}$) of 19–21 days. Thus, in chronic cases, especially with hepatic malfunction, the albumin : globulin ratio and the colloid osmotic pressure of plasma both decline, owing to a failure of liver protein synthesis to keep pace with the loss (normal values are given in Table 11.4).

The rate of plasma protein synthesis can be very high, but depends, apparently, on the level of amino acids,

Table 11.4 Mean plasma protein concentrations (g/L).

	Birth	3 Weeks	Yearlings	2 Years	3+ Years	Mares at stud
TB						
Albumin	25	25	27–28	32	34	27
Total globulins	20	22	31–35	25–28	25–35	31
Non-TB						
Albumin			29	27	28	34–37
Total globulins			38–44	44	47–49	37–40

critically dietary essential amino acids, in the blood. Normal post-absorptive plasma free amino acid levels, even with adequate intakes, are extremely variable (lysine about 15–100 µmol/L, threonine 100–250 µmol/L and methionine 40–60 µmol/L). The ratio of tissue proteins to plasma proteins remains relatively constant at about 33 : 1. Amino acids from muscle tissues and from the diet are used by the liver in protein synthesis. Thus, losses are buffered, but the net loss depends to a large extent on the provision of good-quality dietary protein. Diets containing 140 g protein derived from soya and legume leaf are recommended.

Parenteral nutrition

Where detailed advice is required on this subject, readers are referred to Lopes & White (2002), who recorded a critical evaluation of the parenteral nutrition provided in 79 cases of gastrointestinal disease. Further references will be found in that report. It was noted that the most common complication of parenteral nutrition is hyperglycaemia and that, where possible, some oral or nasogastric feeding should continue to encourage normal GI tract function.

Protein-losing nephropathy

In severe kidney disease there can be considerable loss of plasma protein in the urine. Until the kidney disease has been resolved, a quantitative balance must be maintained between the protein intake necessary to stem the net loss and the amount that would tax the kidneys in urea disposal, causing azotaemia. Dietary protein with an ideal balance of amino acids will minimize urea production.

Urinary calculi (uroliths)

Urolithiasis, often with cystitis, is one of the common urinary disorders, clinically apparent in adult males, and the bladder is a frequently affected site (De Jaeger *et al.* 2000). Cystic calculi are 1.5–20 cm in diameter and weigh up to 6 kg. The crystal growth is driven by supersaturation of the urine, and desquamated epithelial cells or mucous collections serve as a nidus for urolith formation. The chemical composition of uroliths varies somewhat with diet.

A high content of oxalates in pasture is a major contributor, and a rise in urinary pH induces precipitation of Ca phosphates, carbonates and, occasionally, of Ca sulphates, particularly where water intake is restricted. When high urinary pH is an immediate cause, the dissolution of calculi is facilitated by decreasing the pH with acid salts such as ammonium chloride (NH_4Cl), or with ascorbic acid. Daily doses of NH_4Cl are 45–100 g. A dose of 0.33 g/kg BW has been shown to lower urinary pH from 8 to 6.2. A dose of 500 g ascorbic acid on each of two days, or 1 kg once, both by gastric intubation, effectively lowered the pH to 4.7.

By contrast, oxalate removal requires an alkaline pH. Potassium citrate (0.1 g/kg BW per day *per os*) will alkalinize urine and help with calcium oxalate stones, although surgical removal is often necessary. Subsequently, clean water consumption should be encouraged by administering 60 g NaCl/day in the diet for at least three weeks.

Intestinal stones

Excessive dietary Mg and P encourages stone (struvite) formation in the gut, and feeding 200–300 g NaCl daily assists in preventing the precipitation of Mg and PO_4 around the nidus of a calculus by stimulating fluid intake. In many species (including dogs and cats) struvite stones are a major cause of nephrolithiasis. (See also ‘Sand colic’, this chapter.)

LIVER DISEASE

The liver is central to the intermediary metabolism of both nutrients and non-nutrients, and it is the main line of defence in the detoxification of ingested substances. During browsing and grazing the horse ingests many chemicals requiring detoxification, some of which damage the liver, leading to clinical liver disease. A frequent example is hepatic cirrhosis, resulting from the ingestion of pyrrolizidine alkaloids present in *Senecio jacobaea* (see also ‘Seneciosis’, Chapter 10). The risk of damage is increased by several antecedent causes of injury. These include:

- hepatic necrosis caused by *Strongylus equinus* and *S. edentatus* larvae and mouldy hay;
- aflatoxicosis and hepatic lipidosis (fatty change of liver, hyperlipaemia and amyloidosis);
- hepatic metastases from carcinoma of kidney, stomach and pancreas;
- GI disease (obstructive conditions, strongylosis, malabsorption and grass sickness; West 1996); and
- biliary lithiasis.

Liver disease (Table 11.5) causes an inability to metabolize and excrete photodynamic substances in the bile. Reactions to ultraviolet light then occur in unpigmented skin and skin not covered by hair, e.g. the muzzle, eyelids and ears, especially in horses grazing green pasture (see also ‘Dried lucerne’, Chapter 5]. Liver disease occurs typically in pregnant mares of 5–14 years of age and Welsh and Shetland ponies are particularly prone. Hepatic

Table 11.5 Clinical signs and abnormalities of liver disease (West 1996). (Note: characteristics within rows, i.e. signs and clinical abnormalities, are not necessarily related.)

Signs	Clinical abnormalities
Weight loss	Increased serum glutamate dehydrogenase (EC 1.4.1.3)
Anorexia	Increased protein and neutrophil counts in abdominal paracentesis samples
Dullness and depression	Intravascular haemolysis and haemoglobinuria
Jaundice	Raised haematocrit, owing to dehydration
Tachycardia	Leucocytosis, owing to neutrophilia
Intermittent pyrexia	Elevated serum bile acids[1]
Abdominal pain	High serum GGT (EC 2.3.2.2) in biliary-tract damage[2]
Ventral oedema	Elevated plasma ammonia[2]
Muscle fasciculations	Increased prothrombin time[3]
Diarrhoea or constipation	Low plasma fibrinogen and platelet count
Dysphagia	Frequently normal values of:
Photosensitization	• plasma urea[4], but can be low, or high in renal failure
Encephalopathy	• plasma glucose, but severe hypoglycaemia terminally
Mucosal petechiation and nasal bleeding	• hyperbilirubinaemia[5]

[1]Serum bile acids are sensitive measures of early *S. jacobaea* toxicity and with glutamate dehydrogenase, GGT and liver biopsy are useful in diagnosing different types of liver disease.

[2]May be raised in both intra- and extrahepatic cholestasis.

[3]Delayed coagulation is a definite contraindication for liver biopsy, as haemorrhage into the abdominal cavity is likely to result.

[4]Normal ammonia : urea ratio (μmol : mmol) is 3 : 1. A ratio of 40 : 1 carries a poor prognosis.

[5]Frequently with acute hepatic failure. High concentrations are a terminal change. The conjugated form rarely exceeds 25% as the equine kidney is only permeable to conjugated pigment. A conjugated fraction of greater than 35% is normally terminal, whereas high total bilirubin, when most is unconjugated, follows a few days of starvation.

lipidosis is a feature of pituitary neoplasia with hirsutism, laminitis and high plasma cortisol.

The liver is able to regenerate, and the objective in cases of acute liver failure is to provide supportive care until its function is restored. Depending on the cause and development, sorbitol dehydrogenase (SDH) and AST liver enzymes are at increased concentrations in the blood. If biliary obstruction occurs (chronic cases), GGT and ALP are increased and conjugated bilirubin exceeds 25% of the total. In acute cases oral or i.v. glucose administration is frequently helpful and imbalances in electrolyte, acid–base and hydration status should be gradually rectified. In cases of hypoglycaemia, 10% glucose i.v. should be administered to establish normoglycaemia and then 5% glucose, at a rate of 2 mL/h per kg BW, for the initial 24 h. Blood glucose should be monitored and the post-absorptive value maintained approximately between 4.5 and 6 mmol/L plasma. Hyperlipaemia may be moderated by oral dosing with methyl donors such as choline chloride, or betaine and a general B-vitamin supplement. If signs of hepatoencephalopathy occur, a reduction in the absorption of toxic metabolites from the gut is aided by giving mineral oil (contraindicated in horses with GI reflux) carefully by nasogastric tube, as a laxative, at the

Table 11.6 Composition of wet mash (g/kg diet), 4–6 times daily, for acute liver disease.

Micronized barley	326
Crushed oats	400
Beet pulp	150
Beet molasses	120
Lysine-HCl	2
Vitamins* plus trace elements	0.5
Limestone	1.5
Grass hay	1 kg maximum

*Should include choline chloride, 1 g daily.

Table 11.7 Amino acid supplement (g/500 kg BW daily) for anorectic horses.

L-leucine	6
L-isoleucine	4
L-valine	5

rate of 2–4 L/500 kg BW once daily. Horses should be kept out of intense sunlight, to reduce the risk of photosensitization. High protein diets, legume hays and haylage, and silage, should be avoided. A mash, mixed with water, of the composition shown in Table 11.6 and allowed to stand for 30 min before feeding, is recommended. Anorexic horses can be force-fed a gruel by nasogastric tube.

Thiamin and folic acid can be given parenterally once per week. There is a case for raised, or pharmaceutical, doses of niacin to reduce adipose tissue lipolysis and FFA flux (see 'Hyperlipaemia', this chapter). Some benefit may be obtained by including synthetic sources of the BCAAs leucine, isoleucine and valine in the gruel (Table 11.7).

CHRONIC WEIGHT LOSS

Apart from the effects of an inadequate diet and bullying, there are many other causes of weight loss. GI diseases are among the most common. There are numerous recognized and putative causes of small-intestinal malabsorption, including inflammatory bowel disease (possibly an immunological response to chronic antigenic stimulation), alimentary lymphosarcoma, idiopathic villus atrophy, verminous arteritis, intestinal ischaemia, mucosal biochemical abnormalities, mucosal oedema and lymphangiectasia (dilatation of the intestinal lymphatic system, characterized by protein-losing enteropathy). Weight loss is the predominant clinical sign of intestinal disease, owing to inappetance, maldigestion, small-intestinal malabsorption, enteric protein loss and cachexia. Tamzali (2006) proposes a rationalized approach to the diagnosis of the causes of weight loss under the headings: reduced feed intake, malabsorption of nutrients and their decreased utilization, excessive loss of energy and/or protein, and inadequate peripheral tissues perfusion. Haematological evidence is normally limited to anaemia and/or neutrophilia, a response to inflammation. Hypoproteinaemia, hypoalbuminaemia and a marked increase in plasma ALP activity were noted by Chandler *et al.* (2000). In protein-losing enteropathy, all forms of serum proteins are lost into the intestines, but the effect is greatest for plasma albumin and γ-globulin concentrations. An oral glucose absorption test is useful in measuring absorptive function, with resting and 120 min post-administration blood glucose measurements. The 120 min value should be double the resting value.

At feeding time, the activity of the horse should be closely observed. If its behaviour is atypical, the mouth should be examined and the teeth checked. Primary disorders of cheek teeth seem to be dominant. These include abnormalities of wear, traumatic damage, infections and fractures (Chapter 1). Nasal discharge is more commonly present with caudal than with rostral (adjacent to the muzzle) maxillary cheek teeth infections. The response to treatment is good (Dixon *et al.* 2000a, b).

If the buccal cavity is normal, examine the remainder of the GI tract. Increased energy expenditure can result from chronic infections, neoplasia, or RAO (see this chapter). Therapy for most causes of inadequate feed intake includes correction of fluid deficits, hypoglycaemia and acid–base imbalances and the provision of a high-quality diet given in small amounts at frequent intervals, supplemented with micronutrients. Stable design is also important as, for example Fuller *et al.* (1998, 2001) observed that reduced daylight length leads to a decrease in feed intake 5–8 weeks later (see Chapter 7).

One of the following causes might be suspected in cases of chronic weight loss (protein-losing syndromes are discussed above under 'Protein-losing gastroenteropathy' and 'Protein-losing nephropathy'):

- jaw and dental abnormalities, including sharp points on the upper and lower molars and premolars, or abscesses below the teeth;
- roundworm infestation damaging the gut, which has interrupted the uptake of nutrients, and larval stages injuring mesenteric blood vessels that supply the intestines;
- diarrhoea;

- tuberculosis, which very occasionally involves the digestive system;
- liver disease or the presence of some chronic septic focus in the body;
- windsucking;
- shy horses, low in the social order and subjected to group feeding;
- heart abnormalities and anaemia;
- arthritis and other causes of chronic low-grade pain; and
- cancer, especially in older horses. Grey horses are prone to internal melanomas.

Houpt & Mills (2006) reviewed the relationship between behaviour and clinical condition.

THE MATURE SICK OR GERIATRIC HORSE

There is no exact chronological threshold for old age, but it is normally accepted to be 20 years of age. The following points are a guide to the nutritional management of the adult sick horse:

- Provide fresh clean pasture which is sheltered and within observation distance;
- Where concentrate feeds form a major portion of the diet remember that it is necessary primarily to meet the energy requirement. To this end those feeds offered should be edible without difficulty. Grains should be crushed and made palatable with a little molasses. The horse is likely to be fussy and so a range of feeds should be offered to find those most acceptable;
- Include readily consumed bulky treats, such as apples and sliced mould-free carrots;
- Vegetable oil is a useful addition to the feed as it will not cause colic;
- Give B vitamins, including vitamin B_{12} by injection;
- If digestive function is compromised, partial parenteral nutrition (PN) is useful to reduce the load on the GI tract. Total PN can be adopted for a few days. This should be introduced, and subsequently removed, gradually over several days. Where maintenance of fluid status is necessary, acid–base status should be assessed and Ringer's, or lactated Ringer's, solution given i.v. through a separate catheter. Graham-Thiers *et al.* (2005) propose older horses should receive supplemental dietary lysine (0.25% of DM) and threonine (0.2% of DM) to maintain muscle mass.

Many of these points apply to the old horse. Teeth are likely to be in poor condition and therefore small quantities of high-quality leafy forage will be required to supplement larger quantities of digestible concentrates. These can usefully include micronized cereals. Ralston & Breuer (1996) reported that a commercial feed containing 85 g protein, 2.7 g Ca and 2.2 g P/kg was inadequate when it represented 20%–100% of the total feed, the remainder being timothy and lucerne hay. A compound feed containing 140 g protein, 6 g Ca and 4–6 g P, given in similar proportions with the hay and providing similar amounts of DE, led to greater body weight gains, better condition, higher plasma total protein and P concentrations, and higher haematocrit and blood haemoglobin values. Renal function was not compromised by the larger protein intake, as assessed by plasma creatinine concentration. Where older horses are known to have poor renal function, the dietary protein quality, i.e. amino acid balance, should be ideal and the dietary protein allowance should be such as to prevent any excessive rise in plasma urea level.

With respect to immune function, the greater level of white blood cell apoptosis amongst 22- *vs.*12-year-old mares subjected to graded exercise tests indicates that older horses can be immune-compromised during exercise (Williams *et al.* 2008).

MUSCLE AILMENTS

There are several metabolic abnormalities connected with muscle ailments, for which many of the signs are shared, and the nomenclature used is frequently confused. Previous terms include 'tying-up', myositis, set-fast and azoturia.

Exercise-associated myopathy

There is a range of degrees of exercise-associated myopathy, from severe rhabdomyolysis with azoturia, to milder 'tying-up', although the terminology is variable. The onset of clinical signs of muscle disease usually occurs within 5–20 min of commencing exercise which is either mild or strenuous. Signs are also seen in racehorses after racing. Muscle damage is accompanied by an elevation in serum CK and AST.

Post-exhaustion syndrome

Muscle problems occur after severe, prolonged exercise which causes exhaustion. Signs usually appear 2–4 days after completion of the exercise. Damage involves skeletal and cardiac myopathy, hepatic lipidosis, renal damage, laminitis and GI ulceration. The onset of muscular malfunction may occur within a few initial strides, when the horse falters and stops, generally refusing to move. The muscles are not palpably abnormal, but are stiff and sore. The horse adopts a cramped stance and, if willing to move, it does so reluctantly and slowly. Lesser attacks

occur following cooling off. Muscular stiffness, which passes within 1–3 days, is associated with the muscular accumulation of lactate. The activity of serum CK returns to normal after 4–6 days, but that of AST can take 4 to 5 weeks to do so. In addition to a depletion of muscle glycogen (principally in the fast-twitch fibres), there is also a depletion of ATP and CP. Muscle glucose is raised, as well as lactate concentration, suggesting local hypoxia. A leakage of the muscle enzymes AST, CK and LDH is the cause of their raised blood concentration (N.B. Determine iso-enzyme concentrations to confirm origin, e.g. LDH_5 is present in both liver and locomotor muscles).

The horse should be kept warm. Treatment normally includes the i.v. administration of calcium gluconate, magnesium and phosphate ions and vitamin D. Prevention requires training to increase condition, more frequent rest periods, and the administration of electrolyte solutions during and after physical activity. The condition is occasionally noticed in fillies coming into season (oestrogens increase the activity of 1-hydroxycholecalciferase, which implicates vitamin D.

After hard work it is an advantage to trot or canter slowly as this stimulates the transport of lactic acid from the muscles to the liver and stimulate the flow of oxygen to the muscles, so that muscle and blood pH return to normal more rapidly, and the conversion of lactic acid back to glycogen is accelerated.

Exertional rhabdomyolysis (ER) and polysaccharide storage myopathy (PSSM)

An early study by Lindholm *et al.* (1974b) described morphological and biochemical changes in Standardbred horses with rhabdomyolysis. ER is a syndrome of muscle pain, necrosis and cramping associated with exercise and is categorised into 'sporadic' or 'chronic' ER (Valberg *et al.* 1999a). Chronic ER has been described in two forms: (1) recurrent ER, commonly affecting TBs, but also occasionally Standardbreds and Arabians, and (2) PSSM, predominately affecting Quarter horses and occasionally other breeds.

ER

The clinical differentiation of ER from intoxication by ionophores, aortic–iliac thrombosis, atypical myoglobinuria, and exhaustion, following protracted exertion, requires careful analysis of clinical signs and laboratory determinations (P.A. Harris personal communication, 2002). The clinical signs tend to occur during or after exercise, but also in horses at rest, or after they leave their box or field. Horses kept on lush pastures during the week and ridden only at weekends can be susceptible. The signs are presented as a slight shortening of stride to a complete inability to move and recumbency. The condition can be fatal. There is a fall in muscle pH and muscles tend to appear firm. Palpation is normally not resented. Sweating is excessive and elevated pulse and respiration rates are evident. Discoloured urine is voided in severe cases, although this is also seen, for example, in monensin (see Chapter 5) and acorn poisoning. Myoglobin is usually present in the urine when its plasma concentration exceeds 0.2 g/L and this can lead to nephrosis and uraemia. Simple diagnostic methods used to distinguish myoglobin from any urinary haemoglobin contain inaccuracies. The muscle enzyme CK (normal range in TBs in training, 200–800 iu/L) attains a peak concentration in the blood 6–12 h after a single severe episode of muscle damage and returns to normal frequently within a week if no further damage occurs. AST reaches a peak about 24 h after the episode, but normal values are not regained for 2 to 4 weeks. Very high plasma concentrations of CK and raised AST are also observed following strenuous exercise, although they might not represent muscle damage and normal values are achieved after 24 h. ER of polo horses is said to occur more frequently early in the season in those that are excitable and unfit (McGowan *et al.* 2002). Blood cortisol and glucose concentrations tend to be higher in horses with ER, although this can be an effect of stress, rather than a cause of the ailment. The aetiology of ER can indicate poor management of training and it commonly occurs within the first hour of exercise. The ailment usually does not follow a rest period of a single day, or as long as 14 days, but commonly occurs following 2 days' rest on full rations. Some forms of the disease that succeed extended exercise are related to a depletion of glycogen stores and an increased shift to mitochondrial β-oxidation. An interruption in regular exercise, with the maintenance of an immoderate dietary starch intake, seem to be frequently associated factors.

It is vital that the affected horse is not allowed to move at all, as recovery requires that a regime of complete rest is immediately instituted. It might remain on its feet, or become completely recumbent, and severe pain and distress are often accompanied by repeated attempts to rise. The horse should be removed to its stable as quickly as possible in a low-loading trailer, where every effort should be made to keep it standing by slinging, or other means, as this can prevent the development of uraemia.

If exercise and movement have been stopped and treatment instituted immediately the horse can recover in 2–4 days. Treatment has included narcotic drugs and corticosteroids administered i.v. to control the swelling and to

stimulate energy metabolism; Bailey & Elliott (2007) review the evidence that the use of corticosteroids increases the risk of the induction of acute laminitis in horses. Horses can be in a state of dehydration and metabolic alkalosis, or acidosis, and therefore solutions of sodium bicarbonate should not be given unless the acid–base and electrolyte status (see 'Laminitis', this chapter, for K status) has been established. In the absence of this knowledge, isotonic neutral solutions, such as Ringer's, should be given i.v. Some horses have been found to be alkalotic, in which case treatment with sodium bicarbonate would be harmful. Diuresis should be induced (and anyway would do no harm) to diminish the risk of nephrotoxic effects of myoglobinuria. The maintenance of proper kidney function is vital if health is to be restored. A high rate of urine flow at an alkaline pH, prevents myoglobin precipitation in the renal tubules. Although acidosis is a normal feature of azoturia this should not be assumed. Diets differing in DCAB value have similar effects on both ER and healthy horses, as measured by plasma electrolyte concentrations, CK activity, plasma and urine pH and renal fractional excretion values of Na, K, Cl and P (McKenzie *et al.* 2002). Intramuscular injections of 0.5 g thiamin repeated daily seem to be warranted, and the inclusion of pantothenic acid and riboflavin, also involved in oxidative energy metabolism, may be justified. If the painful swelling of the muscles is not reduced, pressure on the sciatic nerve can induce secondary degeneration of other muscles.

The risk of ER is reduced if horses are always warmed up slowly, their concentrate allowance is halved at weekends, or at other times when they are not worked and their work is reinstated gradually with a gradual increase in the consumption of starchy and high-protein feeds.

(1) Recurrent ER

Recurrent ER in TBs has been identified as an inheritable defect of intracellular Ca regulation (MacLeay *et al.* 1999) and a breakdown in Ca transport seems to be a cause. This is supported by the effective prophylactic use of dantrolene sodium (Holmes 2007), a skeletal muscle relaxant, which probably acts by interfering with Ca flux across the sarcoplasmic reticulum. It lessened the elevation of post-exercise serum CK levels and appeared to eliminate signs of ER amongst 77 TBs (Edwards *et al.* 2003). The myoplasmic concentrations of free Ca increase in recurrent ER, associated with damage to mitochondria and other organelles. One study (Valberg *et al.* 1993a) showed that horses suffering from recurrent ER had a higher ratio of type IIA to type IIB muscle fibres, but this could have reflected better training which caused a lower accumulation of lactate and ammonia during near-maximal exercise (see also 'Ammonia toxicity', this chapter). Horses in training with recurrent ER have moderately elevated blood AST and/or CK concentrations at rest, even if they have been free of clinical signs for weeks. There appear to be subclinical episodes of rhabdomyolysis after exercise. Muscle-fibre necrosis and increased plasma AST, CK and myoglobin occur with exercise more frequently than can be detected clinically (Valberg *et al.* 1993b). Wijnberg *et al.* (2008) detected a low plasma vitamin E concentration and myopathy in a Haflinger with the clinical signs of recurrent ER, but found no disturbances in carnitine, acylcarnitines, very-long-chain fatty acid or organic acid metabolism. Elevated plasma lactic acidosis is not causal (Valentine 2008) and the aetiology has yet to be fully explained.

(2) PSSM

PSSM is associated with enhanced insulin sensitivity leading to increased glucose uptake by skeletal muscles and lower resting insulin concentrations occur (De La Corte *et al.* 1999a,b; Annandale *et al.* 2004). Signs of PSSM includes muscle soreness, stiffness, muscle weakness and atrophy and occasional ER. Rhabdomyolysis, especially of the rump, thigh and back muscles is apparent in combination with raised muscle enzymes in plasma (AST, LDH, CK). This disorder is associated with increased muscle glycogen concentrations which at rest can be two to three times normal. The term 'polysaccharide' has been adopted as the diagnosis is achieved by assessing the amylase-resistant inclusions in muscle using the periodic acid Schiff reagent. Amylase digests normal glycogen by breaking down the 1–4 glycosidic bonds, so leaving abnormal glycogen deposits intact. Evidence indicates that the disease can progress from an amylase-sensitive state to amylase-resistant inclusions. Although it was first identified in Quarter Horses, Stanley & Piercey (2007) and McGowan *et al.* (2009) have recorded its diagnosis in more than 30 breeds, or breed crosses, including draught breeds and Votion *et al.* (2009) have determined pasture characteristics with which it is associated..

Amongst a group of Quarter horse foals, evidence of chronic intermittent ER polysaccharide accumulation in skeletal muscles was not apparent until 2 years of age (De La Corte *et al.* 2002). A glycogen storage disorder has also been described in Warmblood horses (Hunt *et al.* 2005). McCue & Valberg, (2007) concluded on the basis of examining 164 overtly healthy Quarter Horses that the prevalence of PSSM in the US is likely to be between 6%

and 12%. Neither sex nor age was associated with the disease. There is increased glycogen synthesis rather than decreased utilization in PSSM and a cause is a R309H *GYSI* mutation of glycogen synthase. Stanley *et al.* (2009) demonstrated this mutation in 65% of UK horses with grade 2 PSSM and in 21% of those with recurrent ER and so it is an important cause of the latter.

Belgian draft horses with PSSM often present with hind limb hyperflexion, which can progress to weakness and recumbency (Fisherman *et al.* 2005). Whereas with draft horses a clinical improvement in PSSM can be demonstrated with diet change alone (Valentine *et al.* 2001b), studies with Quarter horses (Fisherman *et al.* 2003) and Warmbloods (Hunt *et al.* 2005) have demonstrated that both a dietary change and daily exercise (including turnout) are necessary for a favourable response. For both recurrent ER and PSSM a reduction in dietary starch and sugar intake (<8% total DE from starch and sugar) and an increase in dietary fat (>10% of total DE from fat) are beneficial in management (Fisherman *et al.* 2003; McKenzie *et al.* 2003a,b; Hunt *et al.* 2005; NRC 2007), increasing lipid metabolism in muscles (Ribeiro *et al.* 2004). These guidelines include the removal of grain and molasses from the ration and the inclusion of a minimum of 1.5% of BW as grass hay, indicating the need for a high fat and fibre diet, low in soluble carbohydrate of less than 33% starch and sugar (Valentine 2003, 2005) with fat added as exercise rate increases (Valberg *et al.* 1999b).

Infectious myopathies

Myopathy can result from bacterial, viral or parasitic infection.

Hyperkalaemic periodic paralysis (HYPP)

HYPP is a genetic disease occurring in descendants of the American Quarter Horse, Impressive, but also occurring in Paint horses, Appaloosas, Palominos and detected in a Criollo Argentine horse (Diakakis *et al.* 2008). Recurrent muscle cramping, twitching and fasciculations of the flanks, lateral neck and shoulders are observed , occasionally with prolapse of the nictitating membrane along with facial spasms resembling a sardonic grin. In more severe episodes weakness is displayed by swaying, staggering, buckling of the knees, assumption of a base-wide stance, yawning and inability to raise the head, but without signs of pain. Spontaneous recovery occurs after 15–90 min. Other clinical signs include decreased tendon reflexes, sweating, normal or slightly elevated heart and respiratory rates, normal rectal temperature and signs of pharyngeal and laryngeal dysfunction (Spier & Hoffman 2008).

The hyperkalaemia is confirmed following KCl challenge: KCl is given at the rate of 100 mg/kg BW orally in 6 L of warm water via nasogastric tube following a 12 h fast; it is important to restrict the dose to this amount to reduce risks. A DNA test is available for the detection of the genetic abnormality,as a defect in the gene for Na channels in skeletal muscles occurs, with a substitution (cytosine to guanine), causing phenylalanine to be replaced by leucine. This modifies the protein on the intracellular face of the plasma membrane, altering its ability to close the channel. (An electrical gradient, of the order of 85 mV, is normally established across cell membrane in skeletal muscle cells as Na^+ is pumped out and K^+ is pumped into the cell. The gradient is in part maintained by the ability of the sodium channel to restrict entry of Na^+ into the cell. As long as the gradient is maintained the cell is at rest and no muscle contraction occurs. During excitation of the cell by a nerve impulse there is a conformational change in the Na^+ ion channel and the generation of an 'action potential'. During this Na^+ rapidly moves into the cell and K^+ moves out, so the gradient approaches 0 mV. This action potential, in a coupled reaction, causes the release of Ca^{++} from within the sarcoplasmic reticulum of the cell and the Ca^{++} ions move within the cell to the muscle fibrils: actin and myosin. The Ca binds to the myosin and the muscle cell contracts, remaining contracted until the Ca is resequestered in the sarcoplasmic reticulum by the action of a Ca pump.) In HYPP there is a constant movement of Na^+ ions back into the cell via the sodium channel, so that there is repetitive depolarisation of some muscle cells; when these become more severe the condition is clinically recognised as muscle fasiculations. In the most severe episodes the muscle contractions become tetanic and the patient sits or becomes recumbent. Death from respiratory failure is possible. During severe episodes serum K increases from 3–4 mEq/L to 12 mEq/L or more (Meyer *et al.* 1999; NRC 2007) with a decrease in serum Na concentration. The resting membrane potential of muscle fibres is less responsive to changes in extracellular K concentration, implying an increased transport of ions other than K. Muscle CK and AST activities are normal to moderately high and elevations in PCV and plasma protein can occur, indicating a fluid shift (Diakakis *et al.* 2008; Spier & Hoffman 2008).

An oncoming episode can sometimes be avoided with mild exercise, stimulating secretion of epinephrine and thereby increasing the Na^+/K^+ pump, or by giving a 5–10% dextrose solution i.v. that triggers insulin release, with K^+

following glucose into cells. Treatment of HYPP aims to limit increases in serum K by restricting K intake – the risk of episodes is heightened when dietary K exceeds 1% of total diet (Reynolds *et al.* 1998a,b). Horses positive for HYPP should be kept on pasture, where the high moisture content lowers the rate of K uptake. Alternatively in the stable they should be given small frequent meals, avoiding feeds high in K, e.g. alfalfa, brome hay, sugarcane or beet molasses, kelp (seaweed), soyabean meal and most electrolyte supplements (Diakakis *et al.* 2008). (See also 'Potassium', and 'HYPP', Chapter 3)

Nutritional myopathy

Horses with nutritional myopathy present with depression, weakness, dysphagia and dropping of the head and neck (see also 'Selenium', Chapter 3 and 'Vitamin E', Chapter 4, especially for prevention of EDM and EMND). Muscle enzymes are elevated in serum and there is severe degenerative myopathy with hyalinization and fragmentation of muscle cells. The condition occurs typically in neonatal foals, but also in adult horses, and is associated with deficiencies of Se and/or vitamin E.

Hypo- and hypercalcaemia and stress tetany

Extended exertion, particularly in hot weather, leads to dehydration, loss of electrolytes and to energy depletion. The signs of fatigue presented reflect a combination of these losses even though the diet is quite satisfactory. Ca losses in the sweat can amount to 350–500 mg Ca/h, and continued hyperventilation is associated with alkalosis (discussed in Chapter 9). Hypocalcaemia is acutely life-threatening in horses suffering from stress tetany, as total plasma levels can fall to 1.5 mmol/L, whereupon muscular twitching and cramps are manifested. The fall is largely the result of alkalaemia, but occasionally hypomagnesaemia is present. On the other hand, Standardbred trotters subjected to high-speed tests exhibited a decrease in plasma ionized Ca^{++} concentration, despite a fall in blood pH and a rise in plasma lactate, intact parathyroid hormone (PTH) and inorganic P concentrations (see Ca and P, Chapter 3); whereas low-speed exercise resulted in an increase in plasma pH, but with no change in lactate, ionized Ca^{++}, total Ca, inorganic P or intact parathyroid hormone concentrations (Vervuert *et al.* 2002). The authors suggested that intact PTH is a mediator in counter-regulation of exercise-induced hypocalcaemia.

As normal muscle function, including that of cardiac muscle, requires the concentration of Ca in the blood to remain within strict limits, the most immediate need is for careful i.v. administration of a Ca gluconate solution (Table 9.3) while heart function is monitored, as an excessive rate of administration can be fatal. Where the clinical signs of hypocalcaemia are clear, a greater risk is entailed in awaiting confirmation by laboratory determination so that immediate veterinary treatment of this kind is indicated.

A fall in plasma Ca and of Mg also occasionally occurs in lactating mares, although clinical signs are quite rare (see Chapter 7). Where tetany does occur this is usually precipitated by additional stresses of exertion, transport, weather or disease. Ca gluconate should be given. In the dairy cow hypocalcaemia causes paresis rather than tetany.

Electrolyte losses in extended exercise

Several days' rest are required for the regeneration of muscle-cell glycogen reserves after extended work. The exhaustion of this and loss of cellular K in sweat contribute to a sense of fatigue and, as suggested in Chapter 9, recovery during the next few days may be accelerated by providing KCl as well as common salt in the feed (10 g NaCl plus 5 g KCl/kg total feed). Isotonic dehydration depresses thirst so that rehydration is also stimulated by the provision of these electrolytes. Salt licks are supplied in most horse boxes, but many horses will not consume sufficient in this form and the licks normally contain NaCl only. Thus, a powdered feed supplement containing both salts, available from feed merchants, yields a more satisfactory outcome. In fact, during summer weather generally, many horses in training for flat races do not consume enough salt from licks and therefore have a higher PCV and plasma viscosity than should be the case. By encouraging water consumption, electrolytes can have the effect of improving performance. Similarly, during long endurance events, those horses that do not drink fatigue more easily and are less likely to finish. By satisfying a need for electrolytes at rest points, a thirst response is induced, water consumption is increased and dehydration is deferred or prevented (but see Chapter 9, electrolytes and gastric ulceration).

Losses of Ca, chloride and K throughout long rides cause 'thumps' or synchronous diaphragmatic flutter during or after exercise. Losses of these electrolytes and 'thumps' are also brought on by severe diarrhoea. A decrease in the plasma concentration of Ca, chloride and K is thought to change nerve irritability, initiating a contraction of the diaphragm muscles in unison with that of the heart beat. This is seen as sudden movements of the horse's flanks. Treatment consists of replacing lost electrolytes, with access to water. The quantities to be administered are given in Chapter 9. The exhausted endurance

horse is usually alkalotic, so that Ringer's solution, which is slightly acid, is preferred for immediate use. Sodium bicarbonate should not be used in these circumstances unless metabolic acidosis has been demonstrated.

Lameness

In one survey of 314 TBs, 53% suffered lameness at some time and in 20% of the cases lameness prevented subsequent racing (Jeffcott *et al.* 1982b). Undoubtedly, the condition represents a considerable embarrassment to the industry and is a problem in horses and ponies of all types. Kim *et al.* (2009) did not detect any negative influences of early conditioning exercise from birth to 18 months of age on the prevalence of gross defects in cartilage of the midcarpal joint or in the quality of the cartilage extracellular matrix. (see Chapter 8, Brama *et al.* 2009).

Lameness can be defined as a disturbance of gait, which reduces the weight on the affected limb. Although there are a multitude of causes, one study carried out by the author (unpublished observations) on horses in the Far East revealed that faulty mineral nutrition, as estimated by phosphate clearance, all too frequently was associated with vertebral fractures, and probably with fractures of other kinds. Unsoundness of joints results from sprains, strains and jarring forces causing inflammation, which can also result from osteoarthritis. Although this and many bone disorders, e.g. splints, ringbone, osselets, bone spavin, curb, capped hocks and thoroughpin, are unlikely to have specific dietary causes, the severity of response may well be affected by dietary imbalances or deficiencies (see 'Polyunsaturated fatty acids', Chapter 5). Abnormalities in mineral, trace-element and vitamin nutrition are frequently associated with various types of lameness, but little research (apart from investigations of OCD, Chapter 8) has been undertaken to make any objective assessment of the scale of that involvement. Other reasons for lameness include bruised feet, bowed tendons, navicular disease and spinal lesions, which might in part implicate poor hoof care: inspection of hooves daily often avoids long-term problems.

Physitis has already been discussed in Chapter 7 and some reference was made to contracted tendons (see Plate 7.1). The latter often occurs in foals that are doing well with mares having ample milk on good grass. The speed of onset in a foal is surprising, in that within 24–48 h the heel will rise and a slight concavity develop on the front wall of the hoof. Wear at the heel is decreased and increased tension occurs in the extensor tendons. These do not contract, but apparently fail to develop at a rate commensurate with bone growth, and the fetlock joint also tends to enlarge. It is essential to spot the aberration in the early stages so that it can be counteracted by weekly rasping of the heels, exercise, the removal of concentrates and reduction in milk intake. Ultimately, surgery is the only means of rectifying severe angular deformities of leg joints (see also 'Developmental orthopaedic disease', Chapter 8). Exercise is important in the prevention of leg-growth abnormalities, which are prevalent where mares and foals spend long periods in their boxes during adverse weather without any cut in feed intake. It might be preferable to allow mares and foals to remain out all the time in the summer, regardless of weather, so long as some form of shelter is available.

Navicular disease

Lameness and damage to the navicular bone of the hoof are possibly caused by thrombosis of the navicular arteries, abnormal stresses on the bone or by degenerative changes. Treatment includes corrective trimming of the hoof and fitting a wide, webbed egg-bar shoe. Drug treatment with isoxsuprine hydrochloride, a vasodilator, is a common practice, as it avoids the risk of haemorrhage encountered with warfarin. Warfarin (dicoumarol) is helpful, although its use is declining. This drug interrupts blood coagulation by extending prothrombin time. The dose has to be carefully titrated to extend clotting time by 2–4 s from the equine standard of 14 s. An excessive dose will lead to bleeding. If colic inadvertently occurs during treatment, warfarin treatment should be withdrawn and liver function stimulated with vitamin-K treatment.

It is essential that horses treated with warfarin are fed consistently, particularly in respect of the amount of green feed, which is rich in vitamin K, and as the level of work affects clotting time any change in activity must be imposed gradually. If practical, regular work should be commenced and blood samples taken at intervals of at least one per month immediately after exercise, during rest periods. Samples should also be taken 5–7 days after any change in work or feed routine. Veterinary treatment with warfarin must always err on the side of caution as excess can be fatal. The drug seems to act by reducing the viscosity and increasing the flow of blood, which improves the nutrition of the navicular bone, and this is probably more than just the prevention of thrombus formation.

Osteoclastic activity also plays a role in the pathology of navicular disease. Bone remodelling changes that occur are occasionally corrected by tiludronate, a bisphosphonate, that inhibits bone resorption. When given i.v. at the rate of 1 mg/kg BW daily for 10 days, tiludronate improved lameness two and six months post-treatment, so long as

treatment was commenced soon after the first clinical signs were observed (Denoix *et al.* 2003).

HOUSING

Any detailed consideration of housing is beyond the scope of this book, but the environment provided by housing has a profound impact on the well-being of horses. Aberrant behaviour, including wood chewing, wall licking and kicking, or pawing by Quarter Horse weanlings was seen to be much more frequent amongst those box stalled than those weaned in a paddock (Heleski *et al.* 1999). A series of studies by Houpt & Houpt (1988) at Cornell University, Ithaca, New York, has revealed environmental factors important to the well-being of horses. For example, mares in visual contact with other mares are less active and spend more time eating than do those without visual contact. Mares were also found to prefer an artificially lit environment to a dark one. An enrichment of the stable environment through provision of multiple forages reduces straw consumption and motivates foraging behaviour (Goodwin *et al.* 2002). Amongst *ad-libitum*-fed weanling horses, feed intake is increased by 0.2% for each 1°C decrease in barn temperature below 0°C (Cymbaluk *et al.* 1989a).

Ventilation

Little attention to the detail of ventilation is required in the construction of individual horse loose boxes. For reasons of savings in finance and labour there is a trend for housing racehorses in American-style barns (Townson 1992). The communal air and the aerial dust and ammonia levels that can develop at feeding and bedding-down times in barns dictate strict rules that should be followed for ventilation rates, as this is the principal route for evacuating the small particles that are the agents of respiratory distress.

In recommending ventilation rates, allowance has to be made for the fact that the air volume per horse in barns tends to be more than double that in loose boxes (mean values 98 cf. 43 m^3/horse; Townson 1992). Natural systems of air flow (Figure 11.2) should have a controllable inlet area of up to 0.3 m^2 per horse (Sainsbury 1981). If building design impedes natural air flow, fan assistance should be provided at the base of the outlet chimney. This will function as an aid to natural ventilation and is a more desirable solution than the installation of a pressurized system for which the costs are greater and the numbers of suspended air particles increased by greater air turbulence. There should, for similar reasons, be no recirculation of air as occurs in air-conditioned units, as seen in hot climates, where insulation, especially of the roof, is critical.

Abrupt changes in air flow as a result of 'on–off' regulators are to be avoided. Bottom-hinged air inlets are recommended, which deflect cold air up in cold weather and can be fully open in hot for cross-draughts. Their height above the floor should be such as not to interfere with the stock and they must, of course, be of safe construction. Additional inlets will probably be necessary for very hot weather. Where horses are grouped in barns and covered yards a copious air flow is essential at all times. This will be facilitated by a wide open ridge, 0.3–0.6 m wide, with a covering flap. Space boards at the top half of the wall are successful (150 mm wide boarding with 25 mm gaps; Figure 11.2) or, on exposed sites, narrow sliding boards in which one set slides over the other.

The air space in barns is shared, whereas that in loose boxes is either shared or isolated. Although it should be isolated, the tendency is for sharing to be more frequent in modern loose boxes. Probably the most successful housing in the UK consists of various forms of monopitch lean-to buildings (Figures 11.3 and 11.4), which contain single open-fronted boxes facing the warmest wind and the greatest sunlight. These buildings have a low back and high front with hopper-flap air inlets such that the air flows from back to front. An extension to the roof at the front gives cover and cross-partitions act as load bearers. These divisions should be solid as the partly open ones allow crossflow of air, the incidence of wall kicking increases and, where the division contains iron bars, even at quite high levels, shod horses can become ensnared (see also 'Ailments related to housing', this chapter).

Dust loads vary considerably, not only among stables, but also within a stable at various times in the routines of daily husbandry. The horse should be out of the environment when conditions are at their worst and ventilation should be encouraged at that time for the safety of the grooms. Dust can contain pathogens, allergens, irritants and nonpathogenic nuisance microbes. Townson (1992) found that 65% of racehorses in Ireland were bedded on straw, and all were given hay as forage. When this is provided in large round bales, and shaken out in the feed passage before feeding, it adds dust to the barn atmosphere. Where alternatives to hay are not available the hay should be of very top quality, or soaked prior to distribution. Clarke (1987) showed that the number of breathable particles released from moulded rye grass hay was reduced from 45 000 to 1650/mg fresh material after 5 min of soaking and to 525/mg after 24 h of soaking (cf. 44/mg from haylage). Wood shavings, used as bedding in barns, yield relatively few dust particles (Table 11.8); but the concentrations of gaseous NH_3, CO_2, N_2O and water

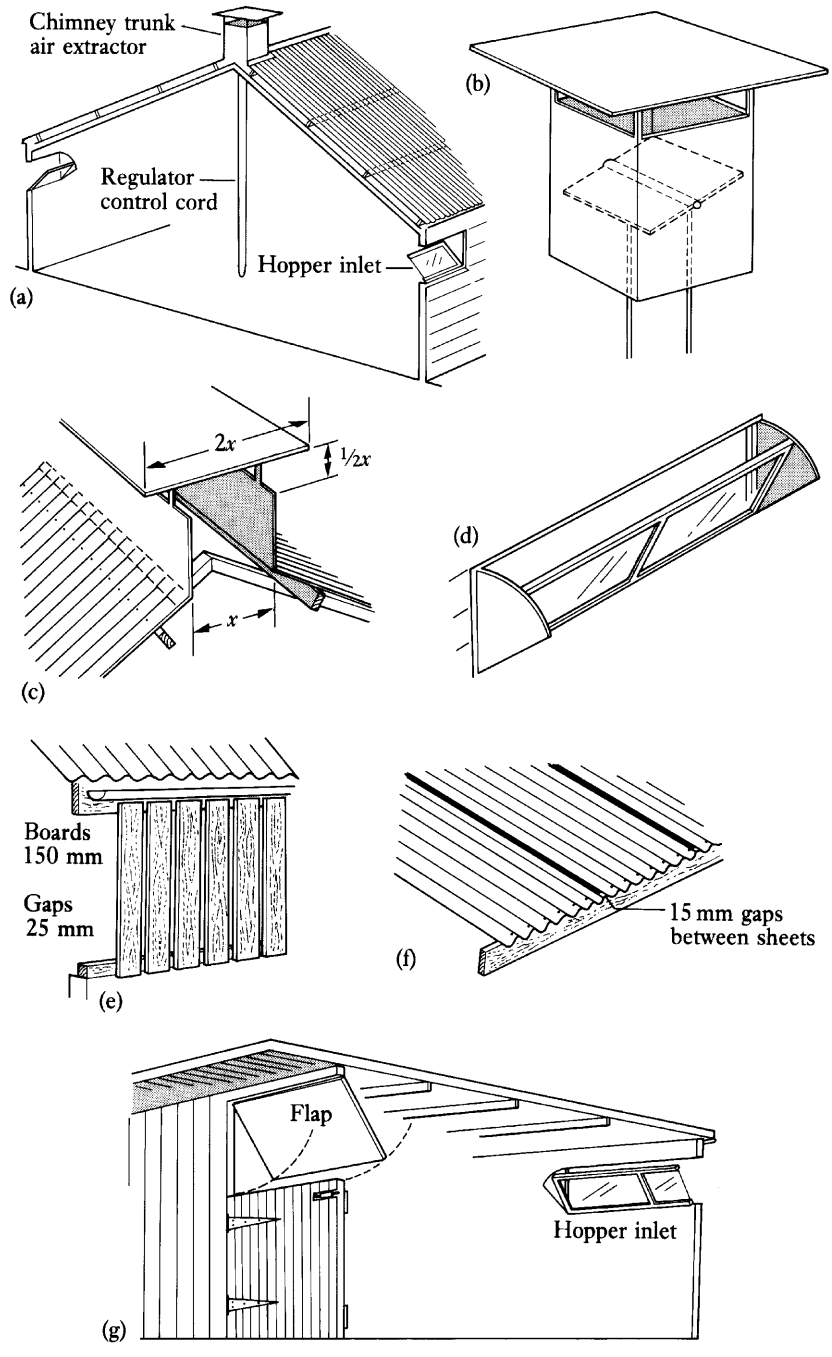


Figure 11.2 Arrangements for the natural ventilation of stables: (a) ventilation suitable for all stables using extractor chimney trunks and hopper inlets for fresh air; (b) detail of the extractor chimney trunk, which can have a regulator or electric fan placed in the base; (c) simple open ridge suitable for extraction ventilation of covered yards; × is normally about 300 mm in yards up to a width of 13 m and 600 mm in yards over 13 m and up to 25 m wide; (d) hopper window suitable as a fresh-air inlet; (e) spaced boards, giving draught-free ventilation: normally 25 mm gaps between 150 mm wide boarding; (f) 'breathing roof' – corrugated roof sheets fixed with 15 mm gaps between for extractor ventilation; (g) mono-pitch house showing hopper inlet at back and ventilating flap at front. Note overhang on roof to protect horses from rain, sun and wind (Sainsbury 1981).

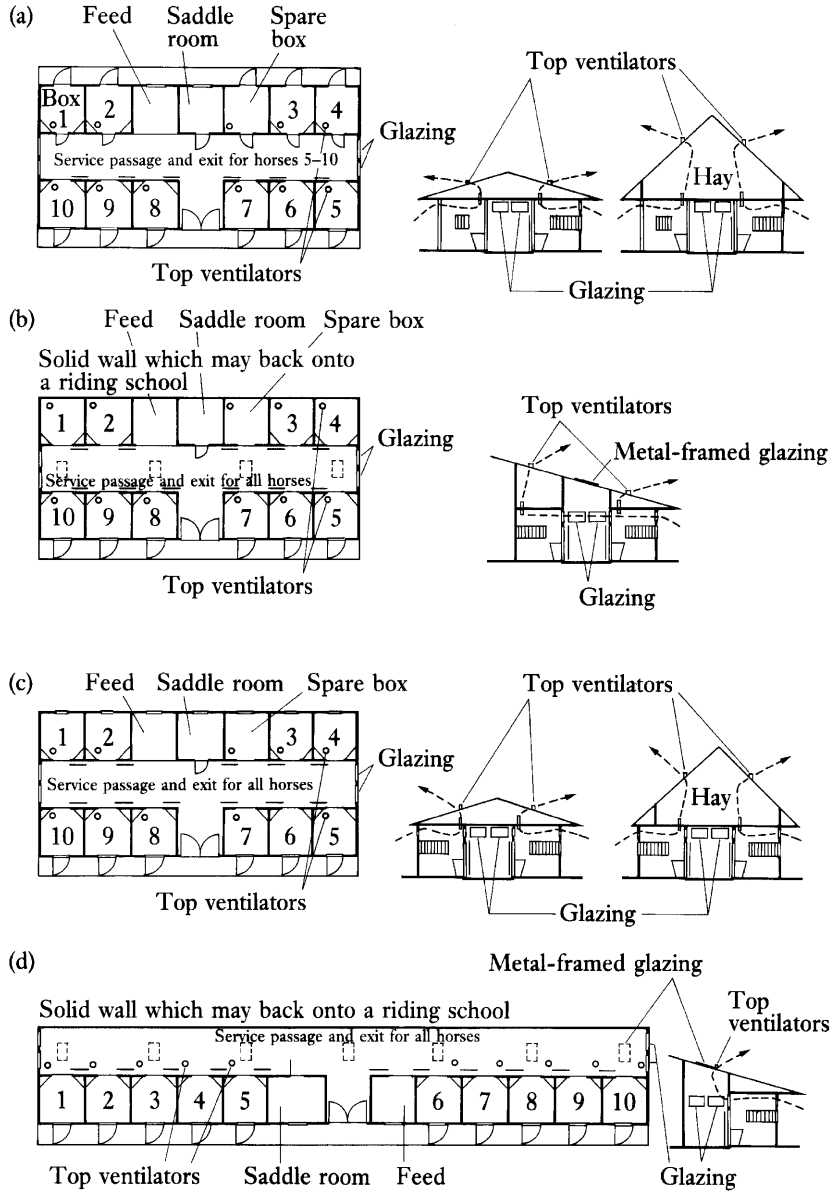


Figure 11.3 Four layouts of 10 horse boxes, which combine the advantages of indoor and outdoor boxes without draughts. (a) All the boxes open to the outside: boxes 1–4 have large exterior service doors; boxes 5–10 have interior sliding doors and shutters opening to the outside. (b) Stable with a solid back wall and interior service; boxes 5–10 have shutters opening to the outside. (c) Stable with a side exposed to bad weather and with interior service; boxes 5–10 have shutters opening to the outside. (d) Stable with a solid back wall and with interior service; all boxes have shutters opening to the outside (Ministère de l'Agriculture 1980).

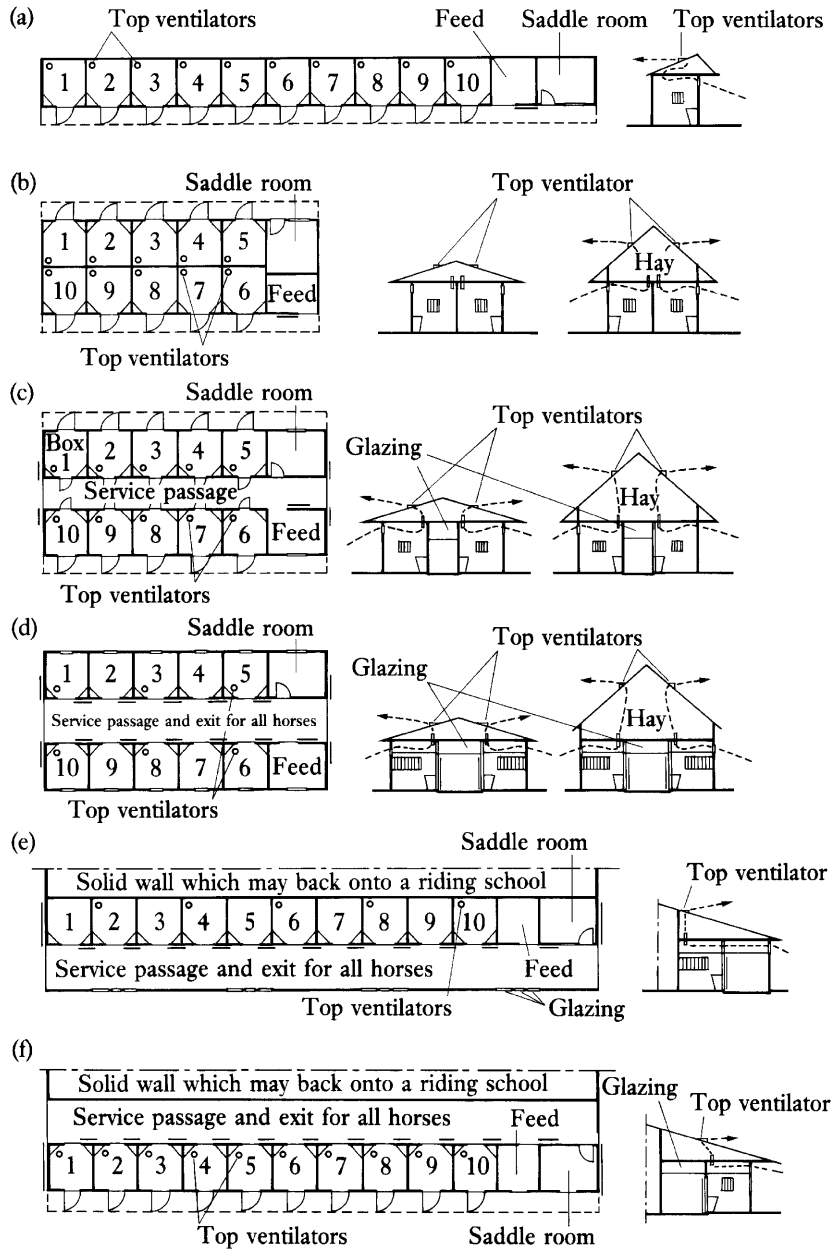


Figure 11.4 Simple layouts for 10 horse boxes: (a) a row of outside boxes with external services; (b) back-to-back boxes with external services; (c) boxes with external horse access and internal service; solid doors and walls in the passage keep draughts to the minimum; (d) boxes with internal (central) servicing without access for the horses' heads to the outside (a less satisfactory arrangement); (e) boxes with internal (lateral) servicing, without access for the horses' heads to the outside (a less satisfactory arrangement); (f) boxes with internal (lateral) servicing and with access for the horses' heads to the outside; solid doors and walls onto the passage keep draughts to the minimum (Ministère de l'Agriculture 1980).

Table 11.8 Number of dust particles/cm^3 stable air in well-ventilated boxes (Webster *et al.* 1987); mean weight of dust/m^3 air in 1166 stables of 26 Irish racing establishments (Townson 1992) and as described by Fleming *et al.* (2008a)[F].

	Number of airborne particles (diameter >0.5 µm/cm^3 air)*			
	Quiet	Bedding down	Weight of dust (mg/m^3 air)	
Wood shavings	8.8	30.5	0.19	0.14[F]
Paper	10.0	40.0	0.37	
Straw	11.7	75.9	0.57	0.23[F]
Straw pellets			0.11[F]	

*Particles of up to 5 µm diameter can enter the alveoli of the lungs. These can include antigenic *Aspergillus fumigatus* and *Micropolyspora faeni*.

Table 11.9 Ventilation rates required to achieve a TLV of 33 particles/cm^3 air at three rates of release of dust into the stable air (Townson 1992).

Dust release rates (particles/cm^3/hour)	Air changes required/hour
60	3
300	10
600	22

vapour above bedding treated with a horse manure/urine mixture tell a different story (Fleming *et al.* 2008b). Means of NH_3 were: wheat straw 178.0 mg/m^3, wood shavings 155.2 mg/m^3, hemp shives 144.6 mg/m^3, linen shives 133.7 mg/m^3, wheat straw pellets 60.3 mg/m^3, and paper cuttings 162.6 mg/m^3. Although straw pellets were the most suitable for stables, Fleming *et al.* (2008b) concluded that the high substrate temperatures generated in straw pellets could favour the growth of pathogens.

Too frequently an adequate ventilation rate depends on whether or not the top half of a loose-box door is open and whether or not barn doors are open. The problem tends to be greater with barns, as the top halves of loose-box doors are normally left ajar, but should have the facility for not being fully closed. The need for good ventilation should be gauged against the evidence that the thermoneutral zone (TNZ) for horses (see Chapter 6) is −10 to +10°C. Loss of body heat through concrete floors by conduction, when a horse is lying down, is greatly reduced by bedding, which also reduces floor draughts. Generally, ventilation rates should be 4 to 8 air changes/h. These rates should keep the number of aerial particles of >0.5 µm diameter to fewer than 33 particles/cm^3 (the threshold limiting value, TLV), a need that depends on the dust release rate (Table 11.9).

Table 11.10 Requirements for natural ventilation of a typical box and barn (Sainsbury 1981; Webster *et al.* 1987; Townson 1992).

Dimensions per horse	Box	Barn
Volume (m^3)	50	85
Surface area of building* (m^2)	41	43
Ventilation inlet area (m^2)	0.34	0.46
Ventilation outlet area (m^2)	0.17	0.23
Height from inlets to outlets (m)	1	2
Ventilation rate (m^3/s)	0.055	0.094
Air movement (m/s)	0.15–0.5	0.15–0.5
Ventilation heat loss (W/°C)	67	114
Ambient temperature (°C)	0–30	0–30
Relative humidity (%)	30–70	30–70

*Assuming 10 horses in a row of boxes or single barn. W, water vapour (kg).

Recommended dimensions for natural ventilation in boxes and barns are given in Table 11.10. Natural ventilation depends on both the area of inlets and outlets and the temperature difference between inside and outside. Insulation of a stable not only affects the temperature

gradient, and therefore the natural ventilation rate (stack effect), for a given area of inlets and outlets, but also decreases the fluctuations in temperature. Moreover, roof insulation can decrease the solar heating effect 10-fold compared with a single-skin roof.

Ailments related to housing

Weaving

Weaving behaviour involves repetitive lateral swaying of the head, neck, forequarters and sometimes hindquarters and is generally indicative of poor welfare. This behaviour is reduced by the visual presence of a healthy horse in a neighbouring box, or by the addition of a 1 m^2 acrylic mirror in the weaver's box and the presence of a hay net (Mills & Davenport 2002). A dietary supplement of virginiamycin has no effect on horses stereotypic behaviour (crib-biting and weaving), or on digestibility, but it increased the water intake of the crib-biters and decreased that of normal horses (Freire *et al.* 2008).

Coprophagy, geophagia, wood-chewing and stereotypies

The first three of these are not defined as stereotypic behaviours because they represent normal physiological or foraging response (Mills *et al.* 2005; NRC 2007). Wood-chewing appears to precede cribbing (see Glossary, under stereotypies) by some horses, infrequently causes small intestinal obstruction (Green & Tong 1988) and is minimized by a high roughage intake (Mills *et al.* 2005). High-concentrate diets, or pelleted feeds, increase wood-chewing activity (NRC 2007) and have been implicated in aggressive behaviour, eliminated by feeding at least 1 kg hay/100 kg BW (Zeyner *et al.* 2004) and can signal gastric erosion. Geophagia, or soil-eating, is not considered to be stereotypic behaviour and is not uncommon in feral and domesticated horses, but it can cause colic or diarrhoea (Husted *et al.* 2005; NRC 2007). Detection can be easier if horses are given their feed off the ground. Stereotypies are said to have breed, age, function, environment, feed management and disease associations (this does not leave much!) (Mills *et al.*2005; Nicol *et al.* 2005). Cooper *et al.* (2005) observed that all horses, whether prone to stereotypic behaviour or not, showed more of this behaviour as feeding frequency of grain increased from 2, to 4, to 6 times/day. These horses displaced resting time, rather than eating time, with cribbing behaviour (McGreevy & Nicol 1998ab). TBs are the breed most likely to be affected by crib-biting. It is rare for an affected horse to be copied (Albright *et al.* 2009). Cribbers given hay were found to have similar plasma concentrations of β-endorphin to non-cribbing horses, whereas the β-endorphin concentrations of concentrate-fed cribbers were less than half those of normal horses (Gillham *et al.* 1994). Crib-biting is probably mediated by neurotransmitters and where there is a high frequency of the vice among horses it can indicate stress caused by bad management. A similar vice apparently does not occur among feral horses. Ways to palliate it include increased handling and exercise, a change of environment and feeding strategies. Crib-biting increases saliva flow that buffers gastric acidity and gastric irritation could be a motivating factor (see Gastric ulcers, this chapter) (Moeller *et al.* 2008, the term cribbing is mistakenly used by the authors).

The following recommendations should reduce the tendency of horses to express stereotypies:

- avoid creep feeding pre-weaning,
- minimise concentrates and increase hay feeding,
- supplement with antacids (the author has found several more effective gastric treatments),
- manage horses on pasture,
- feed the affected horses before other horses,
- reduce stabled time,
- increase exercise, that also benefits training and handler safety (Freire *et al.* 2009),
- increase social contact, and
- use a stable chain instead of a solid door, giving the horse a more varied view (McBride & Long 2001; Mills *et al.* 2005).

Foals

The importance of hygiene where foals are concerned cannot be overemphasized. This includes the removal of pests and risk of acute diarrhoea. Severe liver damage and a lethal infection of young foals (from a few days to six weeks old) is caused by Tyzzer's bacillus (*Clostridium piliformis*). This bacterium is carried in the GI tract of many species of rodents and lagomorphs (rabbits and hares) and infection occurs by consumption of their carcasses, faeces or litter. The endospores can survive for very long periods in rodent litter and are sensitive to heating to 80°C for 15 min, or exposure to 0.015% sodium hypochlorite, 1% iodophol and 5% phenol.

Recurrent airway obstruction (RAO) and other respiratory diseases

RAO

This is now the accepted terminology for the disease entity in mature horses formerly known as chronic obstructive pulmonary disease (COPD), or heaves. RAO is a response

to exposure to organic dusts, principally those associated with stabling, resulting in inflammation and reversible obstruction of the lower airway. A survey of Great Britain showed that greater age, an urbanised environment and exposure to hay are risk factors and that respiratory infection in early life is associated with RAO in later life (Hotchkiss *et al.* 2007). More precipitins to mould antigens generally occur in the sera of horses housed in barns than in those in boxes. However, this does not necessarily relate to the risk or severity of RAO.

Viruses, bacteria (*Streptococcus equi*) and lungworm cause coughing, but the most frequently encountered equine respiratory disease in the UK is RAO. In a population of 300 adult horses referred for pulmonary examination in the north of the UK, Dixon *et al.* (1995) found that of 270 with pulmonary disease, 16.7% presented with infectious or post-infectious pulmonary disease, 2.6% with *Streptococcus zooepidemicus* pulmonary infection, 2.6% with lungworm infection, 5.9% with exercise-induced pulmonary haemorrhage, 3.3% with chronic idiopathic hypoxaemia, 14.1% with miscellaneous identified, or undifferentiated, pulmonary disorders and 54.8% with RAO. Ramseyer *et al.* (2007) determined that risk factors for the clinical signs of RAO in a group of horses by Warmblood stallions were: Sire, hay feeding, and age (in decreasing order of strength), indicating the importance of genetic predisposition in the aetiology of RAO. Soffler (2007) reviews evidence for oxidative stress in the horse and the evidence for current antioxidant therapy in relation to ischaemia–reperfusion injury of the gastrointestinal tract, RAO, exercise, osteoarthritis, EMND, and pituitary pars intermedia dysfunction. Horses affected with chronic and severe RAO tend to be thinner than healthy horses because of a 41% increase in resting energy expenditure (Mazan *et al.* 2004). RAO is a reactive, inflammatory, hypersensitivity response of the small airways (with fibrosis of the alveoli), to dust and mould (fungus and thermophilic actinomycete, especially two moulds, *Micropolypora faeni* and *Aspergillus fumigatus*). Biocompost, made from plant wastes, fermented by aerobic microbes and supplemented with peat, is not recommended as a bedding material, because its concentration of thermophilic actinomycetes was greater than found with wood shavings (Seedorf *et al.* 2007). The monthly prevalence of RAO is correlated with total pollen count measured 3 months previously to the clinical signs and total mould particle count occurring no more than 1 month before clinical admission (Ward & Couetil 2005). High pollen counts can also be associated with summer pasture associated allergy (Costa *et al.* 2000). The small size of respirable particles allows escape from the turbinate trap in the upper respiratory tract, and if the particles are not captured by the bronchial mucociliary mechanism they can reach the alveoli, causing from mild exercise intolerance to severe dyspnoea, even at rest. The threshold safe limiting value of spores for stables has been estimated to be 33 particles/cm^3 (Webster *et al.* 1987).Pirie *et al.* (2002) measured the response of heaves-affected and control horses to an inhaled hay dust suspension (HDS), principally mould spores, or to its supernatant and washed particulate fractions following centrifugation. Airway neutrophilia approached the magnitude of that found with HDS when the particulate and supernatant fractions were combined in comparison to the effect of either fraction separately. The soluble fraction seemed to be of importance in neutrophil recruitment, although HDS was important for pulmonary dysfunction.

Both genetic and environmental factors influence the IgE response to mould allergens from *Alternaria alternata* and *Aspergillus fumigatus*, causing RAO (Eder *et al.* 2001), indicating differences in risk amongst horses. The existence of RAO in a horse predisposes it to additional bronchial hazards. McGorum *et al.* (1998) observed that the total airborne endotoxin (LPS) concentration (standardised against *Escherichia coli* 0111:B4 endotoxin) in conventional stables is excessive. They suggest that the minimal total airborne LPS concentration causing bronchoconstriction in horses with pre-existing airway inflammation, such as occurs in RAO, is much lower than that for normal horses. There are many similarities between RAO in horses and asthma in man (which has shown increased prevalence over the last two decades). It has been proposed that air pollution with nitric oxide (NO) and related hydrocarbon combustion products predisposes patients to respiratory disease. The mechanism probably involves the induction of airway inflammation and increased epithelial permeability, with reduced clearance of allergens and other inhaled particles that can trigger RAO development. Two pro-inflammatory components of organic dust, LPS from Gram-negative bacteria and (1→3)-β-D-glucan from moulds in hay and bedding are contributary causes of RAO (McGorum 2003). Simonen-Jokinen *et al.* (2005) found evidence that airway matrix metalloproteinases increase after exposure to LPS in heaves-susceptible horses. These results indicated to Simonen-Jokinen *et al.* (2005) the potential value of protease inhibitors in attenuating the inflammatory response.

Breathing, in heaves, is characterized by a 'double lift', seen in the abdomen on expiration, when the normal abdominal contraction is followed by a second lift as the horse endeavours to expel more air. This RAO is known

as 'broken wind' or 'heaves' in which parts of the lung tissue lose their elasticity. Individuals between six and ten years old are most frequently affected. Many cases were formerly characterized by elevated maximal intrapleural pressure changes (max dPpl), although Dixon *et al.* (1995) found elevated max dPpl values in fewer than half of their 300 cases.

When clinical signs of dyspnoea occur the IgA : albumin ratio of bronchioalveolar lavage fluid increases and pulmonary neutrophilia of this fluid is frequently found. Relief is achieved by veterinary intervention with a cyclooxygenase blockade. Whether dietary fats rich in *n*-3 fatty acids would reduce the risk of inflammatory responses (see Chapter 5) has not been examined.

There is probably too great a concern that horses will catch a chill at night unless doors and windows are closed, and this often leads to inadequate ventilation, which, with inappropriate bedding, is the most important environmental factor in RAO. Inflammatory airway disease is twice as likely to occur in horses bedded on straw compared with those bedded on shredded paper (Holcombe *et al.* 2001). Straws and shavings were shown to have a similar complement of moulds to that of contaminated hays, whereas peat harboured few potential allergenic moulds (Clarke & Madelin 1987a,b) and all-natural materials were found to be inferior to cardboard bedding (Kirschvink *et al.* 2002a). Data are extremely variable. Fleming *et al.* (2008a) observed hemp and linen shives to have the highest airborne generation of particles, while the lowest generation was detected with straw pellets (111.2 ± 149.2 μg/m^3). Wheat straw had 227.5 ± 280.8, dry wood shavings 140.9 ± 141.9 μg/m^3, and unprinted newspaper cuttings an intermediate value. Horses prefer wheat-straw bedding to dust-extracted wood shavings, which are in turn preferred to shredded paper. Straw leads to more bedding-related activities, yet the welfare significance of this is unclear (Mills *et al.* 2000).

RAO typically presents with a chronic cough, nasal discharge and expiratory dyspnoea. The widespread endoscopic examination of airways, with cytological examination of respiratory secretions and other diagnostic tests has led to the conclusion that RAO is an important cause of poor performance in the absence of any other overt clinical signs. In addition, there has been the recognition, with increasing incidence, of summer-pasture-associated RAO.

Exercise increases oxygen consumption and the generation of reactive oxygen species (ROS), potentially reducing antioxidant defence and increasing cellular damage (Williams 2004). ROS occur normally in cellular metabolism, leucocyte-induced inflammatory response (respiratory burst) and exposure to environmental oxidising agents. Generated ROS molecules include free radicals (molecules containing and unpaired electron) and various peroxides (e.g. lipid hydroperoxides, singlet oxygen). These molecules are self-perpetuating and capable of damaging DNA, lipids, proteins and carbohydrates (Evans & Halliwell 2001). ROS propagation has been associated with joint disease (Dimock *et al.* 2000) and RAO (Deaton *et al.* 2004a). The upregulation of inflammatory genes has been linked to the redox condition initiated by the increase in ROS during inflammation (Kirschvink 2006, 2008) that exacerbates airway pathology. Acute organic dust exposure results in an antioxidant depletion in the trachea. This reflects inflammation and oxidative processes in peripheral airways (Deaton *et al.* 2006). Antioxidant supplementation (Deaton *et al.* 2002), providing vitamins C and E plus Se, given to RAO-affected horses in remission resulted in improved exercise tolerance and lower airway inflammation (Kirschvink *et al.* 2002b). Several enzymes and nutrients have antioxidant functions: GSH-Px (Se), SODs (Cu, Zn, Mn) and catalases (Fe). Other non-enzymatic mineral-dependent antioxidants include ceruloplasmin (Cu) and ferritin (Fe). The vitamins A, C and E and the carotenoids β-carotene, lutein and lycopene also function as cellular antioxidants and participate in the inactivation ROS, so reducing potential cellular damage (Frei 1994; NRC 2007). Horses in acute crisis with ROS have increased oxidised glutathione (GSSG) and glutathione redox ratio, GSSG : GSH, in epithelial lining fluid (ELF) of the pulmonary system (Kirschvink *et al.* 2002c), as indicators of **oxidative stress**. Horses with RAO, showing an inflammatory airway reaction, had lower ELF ascorbic acid concentrations than RAO-affected horses without airway inflammation, or unaffected horses (Deaton *et al.* 2004a, 2005a) with an elevation in cellular GSH-Px concentration (Tan *et al.* 2009), indicating an up-regulation of this redox system to combat oxidative stress. Lepene *et al.* (2009) are assessing the response to an antioxidant mixture of folic acid, GSH and bisamine poly(ethylene oxide), to target the activated macrophages.

Current advice is to:

- keep horses out of doors.
- use only mould-free feeds and bedding, or moisten feeds.
- improve stable ventilation.

Other respiratory diseases

RAO occurs in individual horses and is not infectious, but there are other respiratory diseases that spread from horse

to horse and cause impairment and damage to pulmonary function. The principal transmissible diseases are caused by bacteria, parasites and viruses. Air of contaminated stables in temperate latitudes is a major risk factor for *Rhodococcus equi* pneumonia in foals (Muscatello *et al.* 2006). Viral infections are of increasing incidence and include equine influenza, equine herpes viruses I and II, rhinoviruses I and II and adenovirus, particularly in foals. It is thought that the widespread occurrence of viral infections is associated with the trend to have larger numbers of horses in close proximity in totally enclosed buildings, greater national and international traffic in horses and, it must be said, an increasing awareness and understanding of viruses. Lung damage caused by these infections leaves horses more subject to the allergic responses of RAO: certainly one aggravates the other and both are influenced by building design and atmospheric pollution.

It is concluded that horse boxes should be not only well ventilated, but also sited distant from road traffic. Where haylage or damped hay is impractical to use, benefit is obtained from the use of mould-spore-extracted hay, or from the installation of ventilated hay racks, into which the air is drawn and extracted from the building by a fan. The system can be used as the ventilation system for the box, which should be bedded appropriately. Prophylactic use of sodium cromoglycate is effective in sensitive individuals. However, prevention is better and less expensive than a partial cure.

All hay contains moulds, but some is visibly mouldy and therefore presents a greater hazard than hard, stemmy, clean and shiny hay. Thus, there is reasoned justification for the use of such safe, though nutritionally poor, hay, the lack of nutrients and energy being made up with a high-quality concentrate. Where horses are affected they should be turned out to grass. If this is impracticable, hay should be soaked rather than merely dampened prior to feeding, or it should be replaced by silage, haylage or high-fibre compounded nuts. Dusty bedding should be replaced and the ventilation of the box improved. Symptomatic horses can become asymptomatic in 4–24 days when horses are bedded on shredded paper and routinely fed a complete cubed diet. The pulmonary function values of asymptomatic horses might then not differ significantly from those of normal horses. The ingestion of mould spores, rather than their inhalation, as occurs with the feeding of soaked hay, is *not* a cause of the problem as it depends on a direct reaction between the inhaled particle and the lung alveoli.

STUDY QUESTIONS

(1) How would you set about minimizing the risks of GI disturbances in a stable of working horses?
(2) Where hyperlipaemia has been diagnosed in several pony mares, what is the appropriate course of action?
(3) What are the causes of wasting disease in an elderly horse and what management should be instituted?

FURTHER READING

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Wohlfender, F.D., Barrelet, F.E., Doherr, M.G., Straub, R. & Meier, H.P. (2009) Diseases in neonatal foals. Part 2: Potential risk factors for a higher incidence of infectious diseases during the first 30 days *post partum*. *Equine Veterinary Journal*, **41**, 186–91.

12
Laboratory Methods for Assessing Nutritional Status and Some Dietary Options

If the urine of a horse be somewhat high coloured, bright and cleare like lamber, and not like amber, or like a cup of strong March beere, then it sheweth the horse hath inflammation in his blood. … Now for the smell of his dung, you must understand, that the more provender you give, the greater will be the smell, and the lesse provender the lesse the smell.

G. Markham 1636

Many methods of nutritional assessment are appropriate only for large stables, where random variation in values can be overcome by determinations carried out in a number of horses fed in a similar manner. Nonetheless, whether one is dealing with individuals or groups, it is essential to establish normal values. The normal value of a particular parameter in an individual can differ from the breed mean, as age, sex, time of year, system of management, stage of training, and diet all influence the norm. With knowledge of a norm it is possible to reach a conclusion as to whether a particular value is normal or abnormal. Thus, a disturbance might be uncovered in its early stages. Observation and a range of tests are undoubtedly necessary to fully interpret the clinical evidence.

Metabolic profiles have been adopted as measures of the nutritional and physiological health of dairy herds and similar methods have been applied in stables. In assessing nutritional status, it is essential to have a knowledge of the ingredient and chemical composition of all the feeds and the weights of each type fed over an extended period. Records and the retention of feed samples assist in subsequent solution of problems. This procedure facilitates an objective diet evaluation, which, with the monitoring of management and disease, should indicate the most appropriate laboratory measurements to make, and thereby avoid unnecessary expenditure.

The adequate nutrition of the horse implies the normal nutrition of the tissues and cells of the body, but nutrient-related abnormality of these units does not necessarily imply incorrect feeding. A knowledge of the physiological and health status of the horse must be accommodated in any assessment and, in a few cases, enteral or parenteral, nutrition may be needed to supplement or supplant normal diet for a limited period. One of the purposes of this chapter and the book is to assist in the assessment of a horse's nutritional state.

METABOLIC TESTS

Some of the more commonly used metabolic tests for gauging nutritional status are discussed in this chapter. Single determinants and, owing to analytical error, single determinations, have limited value, as few of the methods are specific to the determination of the status of a particular nutrient, or in understanding a diet-related abnormality.

Variability of measured values

Repeat samples obtained from an individual horse normally yield variable values, due to analytical error, variable sample handling, and variation within the horse without apparent variation in its health. Different analytical methods for the same determinant yield different means, so it is important that the method is stated. Delays in processing equine blood held at room temperature cause increases in the activity of several enzymes and a decrease in blood glucose concentration, but the majority of other changes are unlikely to affect interpretation of results. The immediate separation of the clot from the serum does not materially increase accuracy for many analytes but is of some value (Rendle *et al* 2009).

The normal range for a particular parameter is usually given. However, the most important criterion is not usually that the value falls within the accepted normal range, but any trend in values over time for an individual, which can

nevertheless be compromised by systematic error trends over time. This is overcome by control measurements.

Circadian variation in physiological characteristics has been well established as a phenomenon in man (Frape & Jones 1995, Frape *et al.* 1997b, 1998) and other animals. Several equine blood parameters show a biorhythmical pattern. Under natural conditions, horses feed throughout the 24-h day, but tend to reduce their feeding activities at night whereas, under domestic management, meals are given at set times. Feeding time is known to be a synchroniser of the glucose, NEFA and phosphorus rhythms. Some control of this effect can be achieved by sampling at the same time in the daily routine, preferably at a time when the rate of change is least and when environmental and metabolic factors of no concern have least influence. Meal-fed horses, show a fairly marked 24-h circadian rhythm in the plasma concentration of PCV, plasma total protein, haemoglobin, urea, glucose, insulin, neutral fat, NEFA, cholesterol, Ca, P and several enzymes (Greppi *et al.* 1996).

Enzymes

Nomenclature

Over the years the names of many enzymes have changed, sometimes more than once. So that it is known to which enzyme reference is made in any particular statement, the International Enzyme Commission has classified each identified and specific enzyme with its own code number, known, not surprisingly, as the EC number (International Union of Biochemistry and Molecular Biology on the Nomenclature and Classification of Enzymes 1992). There are at least 3500 enzymes on the list, which is increasing in length. The EC number is a four-part system, the first digit of which defines the class, the second the subclass of that class, the third the sub-subclass, and the fourth the number the enzyme has been given in that sub-subclass, e.g. the oxidoreductase alanine dehydrogenase is EC 1.4.1.1 and alanine aminotransferase is EC 2.6.1.2, (Table 12.1).

Specific tissue isoenzymes

The activity of many specific tissue enzymes is frequently measured as an aid to diagnosis. The plasma enzyme concentration rises during leakage from the cells in which the enzyme is active, either as a result of maximum activity of those cells, e.g. muscle cells during galloping, or as a consequence of damage to the tissue, e.g. toxic damage of liver parenchymal cells. The location of the relevant tissue/organ is helped by measuring the isoenzyme concentration relative to a total raised activity. For example, there are at least five forms (isozymes) of lactic dehydrogenase (EC 1.1.1.27), which are physically and immunologically distinct, but which all catalyze the same reaction.

Table 12.1 Classification of enzymes.

Group	Name
1	Oxidoreductases
2	Transferases
3	Hydrolases
4	Lyases
5	Isomerases
6	Ligases

Diet and enzyme activity

Tissue enzymes are proteins that function only when present as the holoenzyme, which consists of a protein (apoenzyme, that on its own has no catalytic activity) plus a heat-stable, dialysable non-protein coenzyme. This dialysable fraction contains a nutrient in the form of a water-soluble vitamer or vitamin A or K. Frequently, a metallic cofactor, e.g. zinc, is also needed. A dietary deficiency of one of these nutrients therefore depresses the activity of the specific enzyme(s) in which the nutrient functions.

The degree of saturation of several enzymes (the fraction present in the holoenzyme form), measured *in vitro* as the enzyme activity coefficient, can indicate the nutritional status in respect of the nutrient component of the cofactor (Table 12.2). The addition of the cofactor vitamer to the *in vitro* system will increase the activity regardless of the nutritional status, as a portion of the enzyme is normally present in the unsaturated apoenzyme form. Therefore the extent of the response is critical in assessing whether a dietary deficiency exists.

Serum enzymes and liver function

Clinical signs of hepatic insufficiency include loss of appetite, icterus, depression, weight loss, lethargy, yawning and, occasionally, wandering. Haemorrhages in oral mucous membranes can occur and photosensitization has been observed, especially in horses with unpigmented skin and hair, with exposure to bright light. Photodynamic substances in the feed are incompletely metabolized by a compromised liver and cause hypersensitivity reactions in the skin.

Measurement of the serum activities of several hepatic enzymes is used in a determination of a dysfunction of hepatic tissue. Increased serum activities of hepatic

Table 12.2 Cofactor vitamers of several tissue enzymes, with their EC numbers, of particular significance in equine nutrition and those* for which enzyme saturation measurements have been used to assess adequacy.

Enzyme	Cofactor vitamer	Nutrient in cofactor
Erythrocyte glutathione reductase, 1.6.4.2*	FAD	Riboflavin
Erythrocyte transketolase, 2.2.1.1*	Thiamin pyrophosphate (TPP)	Thiamin
Pyruvate dehydrogenase, 1.2.4.1*	Co-carboxylase (TPP)	Thiamin
Fatty-acid synthase[1]	Acetyl-CoA	Pantothenic acid
Glucose-6-phosphate dehydrogenase, 1.1.1.49	NADP(H)	Niacin
β-hydroxyacyl-CoA dehydrogenase, 1.1.1.35	NADP(H)	Niacin
Lactate dehydrogenase, 1.1.1.28	NADH and FAD	Niacin and riboflavin
Pyruvate carboxylase, 6.4.1.1	Biotin	Biotin
Erythrocyte alanine aminotransferase, 2.6.1.2*	Pyridoxal phosphate	Pyridoxine
Erythrocyte aspartate aminotransferase, 2.6.1.1*	Pyridoxal phosphate	Pyridoxine
Methionine synthetase, 4.2.99.10	5-Methyltetrahydrofolic acid	Folic acid
Methylmalonyl-CoA mutase, 5.4.99.2	Adenosylcobalamin	Vitamin B_{12}
α-carboxylase, 4.1.1.1	K hydroquinone	Vitamin K
Succinic dehydrogenase, 1.3.99.1	CoQ_{10}	Ubiquinone[2]

*In vitro measurement of enzyme activity coefficient to assess tissue concentration of the cofactor.

FAD, flavin adenine dinucleotide; TPP, thiamin pyrophosphate.

[1]Multi-enzyme complex which differs among species.

[2]Not generally considered to be a vitamer because the molecular species shares an antioxidant role with other nutrients.

Also see vitamin status in 'Fat- and water-soluble vitamins', this chapter.

ALP, AST (formerly serum glutamic–oxaloacetic aminotransferase, SGOT) and SDH (EC 1.1.1.14) result from reversible changes in hepatocellular membranes, structural injury of hepatic tissue (caused by ischaemic necrosis or cholestasis), or from microsomal enzyme induction. Determinations of GGT (EC 2.3.2.2), serum bilirubin, serum proteins, the clearance of bromsulphthalein (BSP) excreted in the bile, and the histological examination of liver biopsy samples will also be appropriate in suspected cases of hepatobiliary dysfunction. However, hepatobiliary disease is rare in horses, unless the common bile duct is obstructed.

Liver function is also assessed by plasma coagulation tests, including prothrombin time and partial thromboplastin time (platelet function tests, for which several instruments are available, should be evaluated in cases of bleeding problems). Increased liver enzyme activity in serum can arise from decreased hepatic perfusion, causing hypoxia. Cardiac failure, endotoxaemia, septicaemia, hypothyroidism and hyperthermia contribute to hypoxia of hepatic cells, but generally serum enzyme values do not rise more than two to three times the normal value, as a result of these extrahepatic causes.

Aspartate aminotransferase (AST, EC 2.6.1.1)

AST is a cytoplasmic and mitochondrial enzyme present in several tissues – liver and skeletal and cardiac muscles. The activity of this enzyme in serum/plasma is elevated most rapidly following acute hepatic necrosis and it can reach values 10–40 times normal, attaining a peak in 12–24 hours and declining over a two-week period. Activity increases after larvicidal treatment with thiabendazole and in the hyperlipaemic syndrome of ponies and adult horses, but in chronic hepatic fibrosis the serum activity can be normal. The author (unpublished observations) has found values chronically 2–10 times normal in Miniature Shetland mares with bile-duct injury, but without hyperlipidaemia.

Alkaline phosphatase (ALP, EC 3.1.3.1)

ALP is a membrane-bound enzyme synthesized in many tissues, notably active bone tissue, liver, kidney and intestinal mucosa (highest specific activity in the duodenum), During ischaemic colic of the bowel, intestinal ALP is released into peritoneal fluids, where this isoenzyme is shown to have elevated activity (Davies *et al.* 1984). This aids a decision over immediate action in acute cases.

Intestinal ALP is not bound by L-phenylalanine, whereas ALP from other sources is, providing a means of differentiation. Serum values are elevated during bone growth as a consequence of osteoblastic activity, and in horses with renal hyperparathyroidism or with NSHP in Ca or even in P deficiency. When hepatic injury accounts for the increase, it is frequently caused by cholestasis or primary bile-duct injury and the serum level often remains elevated with severe chronic disease. (See also 'Bone metabolism', this chapter.)

Sorbitol dehydrogenase (SDH, EC 1.1.1.14)

SDH is concentrated in hepatocytes and so is used to demonstrate acute hepatic necrosis; if it occurs along with a raised AST, this points more assuredly to an hepatic origin for the disease. The serum activity of SDH declines rapidly over 10 h following hepatocellular disruption, and it has relatively low stability in stored samples.

Gamma-glutamyltransferase (GGT, EC 2.3.2.2)

GGT is membrane-bound in hepatic cells, but it also occurs in kidney and pancreatic tissue. Elevated serum concentrations are accounted for almost entirely by release from bile-duct cells and serum increases result from intra- or extrahepatic cholestasis. Cholelithiasis is such a cause. The source of urinary GGT is probably the brush border of the proximal tubular renal epithelium. High urinary concentrations can indicate proximal tubular dysfunction. Rudolph & Corvalan (1992) found urinary GGT concentrations of 47.6 ± 27.3 iu/L in horses positive for proteinuria and 27.2 ± 17.2 iu/L in those negative (N.B. Some proteinuria is a physiological consequence of a high-protein diet.)

Serum enzymes and exercise

Aminotransferases and creatine kinase

AST, ALT (EC 2.6.1.2) and CK (EC 2.7.3.2) can be detected in the serum and tissues of the horse, although different isoenzymes of AST and ALT are found in the mitochondria and in the soluble portion of the cytoplasm of cells. In the absence of severe tissue injury, the cytoplasmic forms predominate in serum. These two enzymes require pyridoxyl 5′-phosphate as a cofactor for activity.

There is a large individual variation in the normal serum activity of these enzymes and individuals with apparently high circulating levels can achieve a good running performance. A wide margin of uncertainty must be allowed in assessing whether the peripheral concentration of a transferase is within physiological limits, unless there are good previous data available for a particular horse. Adequate circulating apoenzyme must be included in the total. For this, exogenous pyridoxal phosphate should be added to the assay medium (Rej *et al.* 1990). Serum concentrations increase slightly post exercise and are elevated in disorders of muscle soreness, azoturia or 'tying-up' myositis.

Equine rhabdomyolysis is a muscle disorder resulting in elevated serum activities of muscle enzymes, stiffness, loss of performance and/or lameness, with a slightly higher frequency in two- and three-year-old fillies than in other horses. Serum concentrations of muscle enzymes 24 h after exercise might more easily allow differentiation between muscle damage and the post-exercise physiological elevation. The 24-h upper limits of normality can be taken as 100 and 300 iu/L, respectively, for CK and AST. CK is relatively muscle-specific and has a $t_{1/2}$ of about 2 h, whereas AST has a $t_{1/2}$ of 7–8 days.

Plasma proteins

Albumin is the major plasma protein (Table 12.3) synthesized in the liver and it determines plasma colloidal osmotic pressure. Low serum values may indicate that over half the liver has been non-functional for several weeks, as albumin has a long half-life in plasma. Causes include trauma, malnutrition, parasitic-worm infestation, protein-losing enteropathy and renal dysfunction, with elevated urinary levels, or poor hepatic circulation through hypotension, hypovolaemia or chronic inflammatory processes. Insufficient dietary protein is more likely to be the cause of depressed blood haemoglobin than is a relatively scarce quantity of dietary iron, where normal dietary ranges are concerned.

Plasma minerals

The plasma concentration of minerals is used, with considerable reservations, to assess the status of: Mg, K (but

Table 12.3 Blood serum albumin concentrations in TBs determined by the sulphate/sulphite method of Reinhold (1953).

	Number sampled	Range (g/L)	Mean (g/L)
Foal	140	0.512–45	25.8
Yearling	70	0.512–42	28.5
Two-year-old	120	9.5–42	26.8
Three-year-old	90	13.5–49	30.3
Four-year-old	52	0.518–47	29.8
Mare	32	18.5–52	29.6

see p. 332), P, Zn, Se, Cu, and T_3 and T_4, reflecting I status (p. 60). Cr is a component of the glucose tolerance factor and there is speculative evidence that depressed plasma Cr, or a measure of its renal excretion rate, might relate to glucose tolerance and insulin sensitivity. Serum ferritin provides a good index of hepatic and splenic Fe and can be used to evaluate Fe storage in horses. Whereas a deficiency of Se is characterized by a depression in serum concentration of the element and a depressed activity of Se-containing serum GSH-Px (EC 1.11.1.9), the situation with Zn is unclear. In growing children, dietary deprivation of Zn causes a depression in the serum concentration of Zn only where there is sufficient dietary protein to promote a normal growth rate. In other species at least, the activity of serum ALP (EC 3.1.3.1) is sensitive to Zn status, as Zn is a cofactor. The relationship in the horse has not been adequately examined, and the activity *per se* is not specific for Zn status (see also Biting midges, Chapter 11). Plasma concentrations of inorganic phosphate reflect the dietary intake of P and in the deficient state there is an *increase* in the activity of plasma ALP. Plasma measurements of inorganic phosphate and Ca are necessary, together with the urinary measurement of P, in the clearance test for Ca adequacy, discussed under 'Urinary fractional electrolyte excretion (FE) test (creatinine clearance)', this chapter.

Intracellular minerals

When there is a dietary protein deficiency, some success has been achieved in detecting Zn inadequacy by measuring the more labile Zn content of leucocytes (Table 12.4). The reason for this is that Zn is a cofactor of cellular SOD (EC 1.15.1.1). There are two known forms of this enzyme: one contains Cu–Zn and is found in the cytoplasm of most cells and the other contains Mn and is present in the mitochondrial compartment of cells. The measurement of the leucocyte activity of SOD, or the cellular content of Zn and Mn, which tends to reflect the activity of the enzyme, is a measure of the adequacy of these elements. For similar reasons the measurement of leucocyte or platelet Cu, required as a cofactor in cytochrome c oxidase (CCO, EC 1.9.3.1), or the cellular activity of CCO, is informative as a measure of Cu status. Plasma Cu is largely present in caeruloplasmin (EC 1.16.3.1), an acute-phase protein, the concentration of which relates to the inflammatory process, so plasma Cu concentration is unrelated to status.

Table 12.4 Range in leucocyte Cu and Zn contents in seven Shetland ponies receiving poor hay only, before and after supplementation with Cu and Zn for 50 days (author's unpublished observations, by method of Williams *et al.* 1995).

	Cu ($\mu g/10^{-9}$ cells)	Zn ($\mu g/10^{-9}$ cells)
Before supplementation	0.11–0.18	2.57–6.25
After supplementation	0.40–2.86	2.57–10.57

Fat- and water-soluble vitamins

Among the fat-soluble vitamins, the level of plasma α-tocopherol in relation to plasma lipid is a measure of storage status, and elevated breath alkanes can indicate deficiency. A deficiency of vitamin E and/or Se can cause muscle and liver damage that non-specifically leads to an increase in the activity of certain plasma enzymes, particularly AST and CK, owing to leakage from the tissue cells concerned. A more specific measure of adequacy is, however, the determination of red-cell fragility in the presence of dialuric acid or hydrogen peroxide (see Chapter 4 for a fuller discussion).

The homeostatic control of many blood components and the interaction of many nutrients imply that variations in blood concentrations require careful interpretation. Plasma retinol (vitamin A) concentration is only marginally informative. However, the concentration of vitamin A in the liver varies more closely with the dietary intake of vitamin A. Liver tissue is not easily accessed and an alternative sensitive measure of vitamin A adequacy is the RDR, determined in the horse's jugular blood and discussed in Chapter 4. Vitamin D deficiency occurs only in horses confined indoors for long periods, e.g. pit ponies, or those in the higher northern latitudes in winter. Status is assessed by plasma concentration of 25-(OH)-D_3 , possibly in conjunction with serum ALP (EC 3.1.3.1) activity. The determination of blood prothrombin time as a measure of vitamin K status is discussed in Chapter 11. Elevated times would generally indicate antibiotic misuse, causing depressed intestinal synthesis of the vitamin.

Among the B vitamins, quick and routine methods are now available for assessing whole blood vitamin B_{12} (cyanocobalamin), or erythrocyte B_{12}, and leucocyte folic acid, which, in the experience of the author, clearly reflect diet. Serum assays of folic acid frequently do not differentiate between the oxidised and reduced forms, so that a deficiency of the active, reduced, form is masked. Piercy *et al.* (2002) overcame this by either measuring red-cell folate concentration, or by performing HPLC to determine

the relative proportions of the active and inactive forms (see Chapter 4).

In the human subject, the most sensitive indicators for detecting and separating B_{12} and folate deficiencies are the measurements of methylmalonate and homocysteine concentrations in urine and blood. Elevation of both these metabolites indicates a B_{12} deficiency, whereas an elevation in homocysteine indicates only a folate deficiency. Among other B vitamins, methods have been successfully developed for assessing thiamin, riboflavin and pyridoxine adequacy through measuring the activity coefficient of the appropriate enzymes discussed in 'Diet and enzyme activity', this chapter. The activity *per se* (not the coefficient) of a transaminase in red blood cells of sow pigs has been proposed as a good indicator of pyridoxine status. The *serum* activity of AST (EC 2.6.1.1), routinely measured in horses, is greatly influenced by the extent of leakage from liver and muscle cells and therefore the activity in horse red cells can reflect pyridoxine status. Biotin, a cofactor in carboxylases, is assessed in humans from whole blood or urinary biotin concentrations.

Tests for liver and kidney disease

Blood urea

Urea is derived from ammonia and, although the liver has a large over-capacity for its synthesis, blood urea values can be half normal with severe chronic liver disease. Blood values will also be greatly elevated when daily protein intakes exceed NRC requirements by a large margin, a not infrequent occurrence in TBs during training. Nevertheless, much ammonia produced during exercise has a different origin (see 'Ammonia and the alanine vehicle', Chapter 9).

Blood ammonia

Blood ammonia derives from the action of bacterial urease on urea in the gut, although much of this ammonia will be re-utilized in bacterial protein synthesis. Ammonia also results from the deamination of amino acids. Ammonia is converted to urea in the liver, but raised plasma levels have been noted in hepatic encephalopathy. The range of normal values is 80–160 µg/L. As blood samples must be placed on ice (not dry ice) immediately following collection and the plasma separated within 30 min, this measurement is problematic from a practical viewpoint.

Hepatic encephalopathy

Hepatic encephalopathy is a neurological dysfunction resulting from acute, or chronic, liver disease, characterized by depression, frequent yawning, muscle twitching, poor coordination, head pressing and loss of strength and posture. It is associated with high concentrations of blood ammonia, low blood glucose and increased levels of blood VFAs.

Bile acids

About 75% of bile acids in the horse are accounted for by chenodeoxycholic acid. Approximately 85% of these bile acids are conjugated to taurine and the remainder to glycine. The plasma content of total bile acids determined by HPLC rises slightly 2–6 h after feeding, primarily owing to an increase in glycocholic acid production. Liver failure, obstruction of bile flow, or vascular shunting causes a rise in plasma bile acids, as a proportion of those absorbed from the gut is not removed by the liver for resecretion. Normal serum bile acid concentrations can be up to approximately 12 µmol/L, whereas in horses with clinical signs of liver disease the serum concentration can be more than double this.

Bilirubin

Bilirubin is synthesized from the degradation of the haemoproteins of red blood cells in the reticuloendothelial cells of the spleen and liver. Bilirubin conjugated with glucuronic acid is excreted in the bile, except where hepatobiliary obstruction occurs and jaundice develops. Icterus is apparent when total plasma bilirubin concentration exceeds 34 µmol/L (20 mg/L). ALP and GGT are also usually elevated. Fasting hyperbilirubinaemia can occur in the horse, possibly caused by a reduction in hepatic blood flow.

Reticuloendothelial system function

The cells of the reticuloendothelial system have several functions. They act as a sieve, protecting the systemic blood circulation from some products carried to the liver by the portal system. These products include bacterial antigens of gut origin and both enterotoxins and endotoxins resulting from grain overload. It is thought that where there is a systemic acid–base imbalance and liver perfusion is compromised, the sieve is partly by-passed and clinical endotoxaemia results.

Crystalline urine deposits

Urolithiasis is uncommon in the horse despite the supersaturation of equine urine with calcium carbonate. The most common site of calculus formation is the bladder and cystic and urethral calculi have been reported, whereas renal and ureteral calculi are rare. Calcium carbonate is the predominant mineral of equine urinary calculi, although

oxalate and phosphate types exist. Calculi in the horse frequently result from the mineralization of a nidus, possibly provided by a prior disease, such as pyelonephritis, renal papillary necrosis or tubular necrosis. In other species, urolithiasis typically develops with diets having low Ca : P ratios and excessively high Mg contents (in cats and dogs, typically struvite crystals). Low Ca : P ratios activate calcitonin secretion, bone mobilization and soft-tissue calcification, leading to both nephrocalcinosis and urolithiasis. The situation in the horse differs in that the urine is normally supersaturated with Ca salts and persistent stone formers may lack some inhibitor of crystallization.

Urinary fractional electrolyte excretion (FE) test (creatinine clearance)

Electrolyte status is a function of intestinal absorption, renal tubule re-absorption, tissue deposition and mobilization, sweat loss and renal excretion. Serum or plasma concentrations of electrolytes are not used to detect electrolyte imbalance, owing to efficient homeostatic mechanisms which maintain relatively normal blood concentrations despite extreme body depletion. Homeostasis is primarily mediated by the kidney, so that the amount of an electrolyte excreted daily varies with the body's status. However, the daily urine volume varies considerably between individuals so the concentration of an electrolyte in the urine is an unreliable guide to nutritional status. The collection of urinary losses over a period of several days would indicate the quantities surplus to requirement, but such extended urinary collection is impractical. Urine concentration of an electrolyte is therefore related to that of a control substance. This substance should (1) have an excretion rate similar to the glomerular filtration rate, and (2) not be secreted or reabsorbed by the renal tubules. Creatinine, the excretion product of creatine metabolism, fulfils these requirements reasonably well and the fractional electrolyte excretion (FE) is measured as the creatinine clearance ratio (renal creatinine averages 1.15 ± 0.41 mg/h per kg BW; Meyer 1990b. Urine sample creatinine concentrations of <9000 µmol/L (1 mg/dL creatinine is 88.4 µmol/L) could indicate contamination of the sample or excessive salt consumption, causing polydipsia/polyuria, so the FE values obtained should not then be accepted, although low values in horses of less than 18 months old can indicate a physiological abnormality (P.A. Harris 1996, personal communication).

A urine sample and a serum sample are required. The urine sampling should be achieved without resort to the use of diuretics because they affect Na and, to a lesser extent, K and chloride losses. The clearance of an electrolyte equals the concentration in the urine times the urine volume divided by the concentration in the serum. The clearance ratio is the clearance of the electrolyte divided by the clearance of creatinine. In this ratio, the urine volume cancels out and so need not be measured. The equation for which the determinants are required is given below:

$$\text{Percent electrolyte clearance (\% creatinine)} = \frac{[E]_u \times [Creat]_s}{[E]_s \times [Creat]_u} \times 100$$

where $[E]_u$ is the concentration of electrolyte in urine, $[E]_s$ is the concentration of electrolyte in serum, $[Creat]_s$ is the concentration of creatinine in serum and $[Creat]_u$ is the concentration of creatinine in urine.

High-concentrate rations tend to give a raised phosphate clearance and a depressed K clearance, the reverse being the case for high-roughage rations. These effects are quite normal. K clearance, together with measurements of blood and urinary pH, is useful in evaluating the type of acidosis and in assessing K depletion in exhausted horses, as the urinary excretion of K and H ions tends to display a reciprocal relationship. K clearance is depressed in chronic laminitis. Note that even slight blood haemolysis increases plasma K and therefore yields falsely low FE values. A raised Na clearance can indicate excessive Na intake in the form of common salt, Addison's disease, dehydration or tubular malfunction. Stewart *et al.* (2004) indicated that 24 h urinary Mg excretion, or renal Mg clearance, gave more reliable indications of inadequate Mg intake than did serum total, or ionized, Mg concentrations (normal serum total Mg 1.6–2.0 mg/dL, 0.66–0.82 mmol/L).

Owing to the circadian rhythm of electrolyte excretion, referred to in 'Variability of measured values', this chapter, the FE ratio should be measured at least three times during a 24-h period. The serum and urine samples do not have to be taken simultaneously, but the samples should be taken pre-meal and pre-exercise. Decreased urinary Mg and Ca concentrations occur shortly before and after feeding with maximum values 4–8 h after feeding. The serum sample should be sterile and that of urine as close to sterility as possible. The urine sampling should comply with the following restrictions (P.A. Harris, Animal Health Trust, Newmarket, personal communication; Meyer 1990b):

- Samples should be freely voided, using a collection harness before exercise, to obtain the entire product of

micturition. Walking the horse slowly first thing in the morning while its stable is being cleaned and rebedded promotes urination on return. If catheterization is necessary, fillies and mares should first be bled and then given a short brisk trot before sampling. Catheterization will decrease the risk of contamination, and so be helpful. Plasma K concentrations also decrease during eating and rise to a peak several hours post-ingestion.

- Meyer (1990b found a lower mean urinary concentration of creatinine during the first two hours after eating. Moreover, Piccione *et al.* (2009) found a daily rhythm in serum creatinine and CK levels and differences in their amplitude between sedentary and athletic horses. Thus, for the assessment of dietary adequacy by measurement of the ratio, the measurements should be made before feeding or at the following times after a meal: 3 h for Na, 6–10 h for Cl, 3–8 h for K, and 4–8 h for Ca.
- There is little difference between the early, middle or late portions of a voided urine stream in the FE of Na, K, PO_4 and Cl, but for Ca, collect the entire voided sample. Owing to the supersaturation of urinary Ca, reliable urine sampling for Ca clearance is not achieved satisfactorily. The amount of Ca precipitate voided at any one moment is unpredictable. However, where a low urinary Ca value is supported by a low Mg value this is strong evidence of a dietary Ca deficiency and possibly a deficiency of Mg. Mg is more soluble so that urinary values are more reliable and Mg responds in a parallel fashion to that of Ca. The FE test is not used to monitor low phosphate intakes.
- If a delay between collection and analysis is unavoidable, there is little change in the Na, K or Cl concentration in plasma or urine on storage at 18°C, 4°C or –20°C. However, considerable changes in Ca, PO_4 and creatinine concentrations occur, especially at 18°C. Hygiene is important. Samples should be transported in capped sterile containers and stored at 3–4°C for short periods, or at –20°C for longer periods. Ca, PO_4 and creatinine should be analyzed as soon as possible, i.e. within 4 days.
- For urinary Ca determinations, mix well and use a flame atomic absorption spectrophotometer, as colorimetric methods are unsuitable for serum or plasma samples.
- Samples collected during, or shortly after, an episode of equine rhabdomyolysis do not reflect electrolyte status, owing to circulatory disturbances and raised plasma myoglobin concentrations which could affect renal function. Samples with a pH of 6 or below, and those that are positive for glucose, blood, myoglobin and/or haemoglobin, are unsuitable. A low urinary pH is accompanied by an increase in Ca and Mg excretion, giving falsely high Ca and Mg FE values. Blood and urinary pH can be decreased on a high-starch diet, yielding raised urinary Ca and P clearances, unrelated to changes in DCAB (see Chapter 9).

Savage (2008) reviewed evidence of urinary clinical pathology, and concluded that FE rates of electrolytes and minerals guided decisions over fluid and nutritional care.

Effect of diet on FE

Diets with a Ca:P ratio of 1:1, or lower, produce an elevated FE PO_4, whereas inadequate Ca and low Na intakes lead to low FE Ca and low FE Na. Normal clearance ratio values are given in Table 12.5. Frequently TBs in hard training in the UK appear to be receiving inadequate Ca and Na, reflected in low FE values for Na and high values for PO_4. However, it is unclear whether there

Table 12.5 Interquartile (25–75%) normal reference ranges for FE% (or creatinine clearance, % of electrolyte) of adult horses at rest and in work from spot samples (P. A. Harris personal communication) and a mean published range.

Electrolyte	Grain based diet* FE %	Balanced compound and hay diet* FE %	Published FE %
PO_4	0.0–0.5	0.0–0.2	0.04–1.19
Na	0.02–1	0.04–0.52	0.01–1
K	15–65	35–80	15–75
Cl	0.04–1.6	0.7–2.1	0.04–1.65
Ca		>7 (8–24)	
Mg		>15	

*Data provided by P.A. Harris.

is any relationship between these inadequacies and the frequency of muscle stiffness, as the appropriate definitive experiments have not been reported. (For bone mineral status, see Chapters 7 and 8).

DIETS FOR LIVER DISEASE

Dietary causes of liver disease include aflatoxin, from mould-infected grains and protein concentrates (especially groundnuts), and pyrrolizidine alkaloid poisoning from *Senecio* and *Crotalaria* species. Supportive therapy has the objective of allowing time for regeneration of hepatocytes. This could initially involve i.v. glucose administration, followed by enteral feeding of glucose. For horses suffering severe hepatic dysfunction, dietary management should ensure the following:

- The ration should be divided into at least three daily meals.
- The ration should contain the highest quality protein in adequate, but not excessive, amounts.
- An amino acid supplement of the glucogenic BCAAs, isoleucine and valine (1 g/kg diet of each) may help.
- Soluble fibre sources, such as citrus pectin and beet pulp, are useful, together with wheat bran, soya hulls and other insoluble fibre sources, and an adequate level of several cooked starch sources.
- Fat supplements should not be added to the diet (although there is a case for fats containing *n*-3 fatty acids, e.g. fish oils).
- Vitamin E (1500 iu/day) and a water-soluble B vitamin supplement, including 1000 mg choline/kg diet, are advisable.
- A supplement of 0.5 kg DL-methionine/tonne, has been recommended. There is, however, some evidence that excessive dietary methionine is converted by gut bacteria to mercaptans. Following absorption a diseased liver is unable to clear these bacterial metabolites adequately and, acting with ammonia, they can cause signs of encephalopathy. Unfortunately mercaptans are also derived from cystine, so, if the evidence is reliable, the basal diet should be relatively low in total sulphur amino acids, i.e. not more than 3.5 g/kg diet. If methionine is to be added it should ideally be the L-α-isomer (rather than the DL-), and included at a concentration of 0.25 kg/tonne. (The author has never observed encephalopathy to result from methionine supplements in horses with compromised livers, so the risk may be slight.)

Bergero & Nery (2008) reviewed the management of hepatic diseases. Diets should be low in protein, of good biological value, and high in non-structural carbohydrates, except in cases of chronic hepatic disease, for which a slightly raised dietary protein is recommended. The mortality of equine hyperlipaemia is around 70% and so for its management a positive energy balance should be maintained, providing lipotropic agents (choline, etc.), but avoiding dehydration and metabolic acidosis. (See also Chapter 11, Hyperlipaemia and Liver Disease.)

DIETS FOR KIDNEY DISEASE

Renal dysfunction and failure can be caused by any acute condition leading to a severe reduction in glomerular filtration rate. This can be induced by shock, impairing renal blood flow, trauma and haemorrhage, severe dehydration, obstruction of the urethra, hypersensitivity reactions, certain bacterial and mould toxins and consumption of particular metal salts, including those of mercury, lead, arsenic and hexavalent chromium (see also Chapter 3).

Renal failure can lead to metabolic acidosis, therefore the diet should have as an objective the prevention of a low urinary pH. Dietary protein should be as described, above, for liver disease with an emphasis on protein of excellent amino-acid balance, given in adequate, but not excessive, quantities. Thus, deamination and uraemia will be minimized. A cooked, high-carbohydrate diet should be given to provide an adequate energy supply, so avoiding endogenous protein catabolism. Dietary P should not be excessive and the Ca : P ratio should be 2 : 1. NaCl supplements should not be given in excessive amounts and diuretics should be avoided. K retention can occur, so that control should be exercised over dietary K. Grasses and other raw materials known to contain oxalates should be excluded, and a pyridoxine supplement might be given. Fresh water should be available at all times and blood urea and ammonia monitored in both kidney and liver dysfunction.

BONE METABOLISM

Skeletal ailments are not infrequent in young growing stock and chronic, non-specific lameness in adults is sometimes an expression of NSHP, the most common cause of which is faulty Ca and P nutrition and an improper balance between these two minerals. A simple means of evaluating Ca adequacy is required. Ca concentrations in serum vary to a small degree in relation to Ca intake, and where they are measured in a number of horses, significant differences can be detected between deficient and normal groups (Figure 3.2). However, the method is insufficiently sensitive to be of practical use. Greater sensitivity is achieved with creatinine clearance ratios. The horse regulates serum

Table 12.6 Reference ranges in serum from healthy horses for PICP, ICTP, bone ALP and total ALP (after Price *et al.* 1995).

Age group	PICP (µg/L)	ICTP (µg/L)	Bone ALP (u/L)	Total ALP (u/L)
<1 year	1216–2666	14–27	134–288	223–498
1–2 years	550–1472	8–23	33–125	134–238
3–4 years	248–925	6–15	25–70	101–203
5–20 years	136–394	0–9	13–47	91–352

Ca more by renal excretion than by controlling intestinal absorption. Intact proximal tubules are required for P reabsorption and Ca excretion and in renal failure serum Ca is increased and P is depressed. The fully functioning kidney excretes excess Ca in the urine and therefore an inadequate intake might be revealed by a reduced urinary excretion, if it were not for the fact that Ca sediments in horse urine, owing to its alkaline nature. Repeatable estimates of the Ca content of urine cannot be readily attained.

Phosphate clearance can fall to zero where the diet is only marginally adequate in P and the clearance will be increased when the intake is greatly in excess of need, or when the horse is suffering from NSHP. An inadequate dietary intake of Ca stimulates the secretion of parathyroid hormone (PTH, see Chapter 3, for method), which increases the reabsorption of Ca by the renal tubules, mobilizes bone Ca, and increases the loss of phosphate through the tubules by decreasing tubular reabsorption of phosphate (see Vitamin D, Chapter 4). The net effect of this is to stabilize the ionized concentration of serum Ca and depress serum phosphate. Concentrations of Ca in serum tend to be below average, but within the normal range. An increased phosphate clearance with a normal or slightly depressed serum Ca, is therefore frequently indicative of insufficient dietary Ca when other causes have been eliminated. Measurement of changes in the rate of bone metabolism are helpful in assessing bone modelling and remodelling, during training, in the detection of cases of OCD or early stages of other bone disorders and during fracture repair (see Later growth, Chapter 8). Bone ALP is located on the cell surface of osteoblasts and it plays an important role in bone formation. It accounts for approximately 60% of circulating ALP activity in horses of <1 year of age, whereas in those >5 years it accounts for about 20%. Separation of the bone isoform is necessary in the detection of changes in bone metabolism.

Price *et al.* (1995) reported procedures for the measurement of bone ALP in serum by precipitating it with wheatgerm lectin (N.B. Liver ALP binds with wheat germ in man, but not in the horse, while two-thirds of caecum ALP binds in the horse, but does not appear in the blood). This binding depends on the number of carbohydrate units in the side chain of ALP, and, although there are two or three isotypes within each tissue of origin, the procedure still segregates tissue sources. Using radioimmunoassay, Price *et al.* (1995) also measured serum carboxyterminal propeptide of type I collagen (PICP) and serum pyridinoline cross-linked telopeptide domains of type I collagen (ICTP). ICTP is liberated into serum during the collagen degradation of bone resorption in man and it is a precursor of pyridinoline cross-links in urine. These determinants are all associated with bone modelling and are quantitatively and inversely correlated with age, at least up to five years in the horse (Table 12.6). Deviations from these ranges probably indicate bone metabolism, related to bone disease or repair, and with the parallel determination of other parameters, particularly Ca and P clearance, the data should be of diagnostic value. Type I collagen also occurs in the skin, tendons and ligaments, but the proportion in the serum pool, derived from these sources, seems to be small, and moreover inflammation of tendons in the horse is frequently related to bone resorption.

Blood osteocalcin is a measure of bone metabolism, but the molecule is labile and assay values in different laboratories have varied considerably (see 'Later growth', Chapter 8).

OTHER TESTS

Hair analysis

The chemical determination of proteins, minerals, trace elements and toxic heavy metals in hair has been suggested as a means of assessing previous dietary presence. The author has found increased amounts of lead in hair in lead toxicosis. However, the mineral composition is influenced by hair colour (Hintz 1980b), and quantitative measures require other procedures. The bulb diameter of hair among horses has been used successfully in the assessment of dietary protein adequacy (Godbee *et al.* 1979).

Table 12.7 A sequence of procedures for investigating possible causes of signs indicating possible nutritional anomalies in horses, recorded descriptively and by photograph, with times and dates.

Sequence of initial actions	Records to be kept	Laboratory investigation sequence
(1) Examination of horses, social interaction, collection of tissue samples, immediate therapy	Abnormal signs and age distribution	Routine haematology and biochemistry, preserve samples for later examination
(2) Examine co-grazing ruminants	Associated signs	If necessary, routine blood values
(3) Examine stable/paddocks	Water, weeds, pollutants, bedding, poisons, surface materials, dust	Identification of plants, pollutants and poisonous substances
(4) Sample all feeds, including pasture	Physical examination, ingredient make-up, calculate nutrient levels and intakes	Feed analysis for heavy metals, drugs, trace elements, fats, natural toxins
(5) Collect previous feed samples and records	Calculate presumed nutrient intakes	Calculate chronic intakes of nutrients and toxicants Specific non-routine determinations on initial samples
(6) Test presumptive corrective treatments in sequence or in selected sub-groups of horses	Clinical results	Determine any change in fresh sample values, previously abnormal

Sugar tolerance (as distinct from the glucose tolerance test)

Glucose and xylose absorption tests have been used to measure the gross function of the small intestine. The recommended procedure is to give 0.5 g D(+)xylose/kg BW as a 10% solution by stomach tube. By measuring the peak plasma xylose concentration after 90 min it is possible to discriminate between normal and abnormal absorption. A normal control animal should be measured under the same conditions. An oral lactose tolerance test (1 g/kg BW, as a 20% solution) should help determine small-intestinal mucosal damage in diarrhoeic foals, when the continued ingestion of lactose can be detrimental (Roberts 1975a,b; see also Chapter 1).

Feed allergen tests

Feed allergen tests have been used by the author for determining whether feed entities are causal in certain oedematous skin and respiratory irregularities. The horse is prone to such reactions, but cross-reactions between proteins must be expected. Blood and skin tests are appropriate in the detection of dietary and mould allergens, although the intradermal skin test has shown poor repeatability (Lebis *et al.* 2002). Nevertheless, the method detected a positive response to *Culex pipiens* (common grey gnat, a mosquito) and to *Dermatophagoides farinae* (sarcoptiform mite) in horses with skin signs of allergic origin (see also Chapters 5 and 11).

Haematology

The measurement of numbers of red and white blood cells has been used in folic acid, vitamin B_{12} and protein assessments. For consistency, samples should be drawn immediately after strenuous exercise; lower erythrocyte values should be expected for ponies and cold-blooded horses. Normal values are influenced by sex, age, stable and season, and, among racehorses, by training. More sensitive methods of assessing vitamin B_{12} and folic acid status are suggested in 'Fat- and water-soluble vitamins', this chapter.

Potassium status

The measurement of $[K^+]$ in red blood cells should be undertaken within two hours of removal of the blood from the horse. The metabolic energy required for the active transport of K^+ is not generated in blood samples stored in the refrigerator and consequently K^+ leaks by diffusion

from the cells and Na^+ penetrates their membranes. This movement can be reversed by incubating the blood with glucose, or by reinjecting the cells into the circulation. (See also Chapter 11.)

PROCEDURES FOR DETERMINING CAUSES OF SUSPECTED NUTRITIONAL PROBLEMS

Procedures for determining causes of suspected nutritional and dietary problems must vary according to the amount and reliability of the information at hand and whether there is a justification for determining causes rather than simply replacing an inadequate feed system by a proven system. A proposed procedure is given in Table 12.7.

STUDY QUESTIONS

(1) Construct a 'decision tree' and procedure you would adopt for determining the causes of a diet-induced nutritional problem.

(2) How would you propose a horse with poor liver function be fed and managed?

FURTHER READING

Donabédian, M., Van Weeren, P.R., Perona, G., *et al.* (2008) Early changes in biomarkers of skeletal metabolism and their association to the occurrence of osteochondrosis (OC) in the horse. *Equine Veterinary Journal*, **40**, 253–9.

Greppi, G.F., Casini, L., Gatta, D., Orlandi, M. & Pasquini, M. (1996) Daily fluctuations of haematology and blood biochemistry in horses fed varying levels of protein. *Equine Veterinary Journal*, **28**, 350–53.

Appendix A
Example Calculation of Dietary Composition Required for a 400 kg Mare in the Fourth Month of Lactation

(1) To calculate the proportions of hay and concentrate in the total diet, divide the required daily energy intake (MJ/day) by the desirable daily total feed intake (88% DM) to give the average energy density of the total diet. Then form a simple equation containing the energy densities of the roughage and of the cereal available (88% DM).

Required daily energy	84.5 MJ DE/day
Desirable total feed intake	9.0 kg
Oats	12.1 MJ DE/kg
Hay	7.3 MJ DE/kg

Let x be the proportion of cereal in the diet and $1 - x$ be the proportion of hay.

$$12.1x + 7.3(1-x) = 84.5/9.0$$
$$12.1x + 7.3 - 7.3x = 9.39 \text{ MJ DE/kg}$$
$$4.8x = (9.39 - 7.3) \text{ MJ DE/kg}$$
$$x = 2.09/4.8 = 0.435 \text{ or } 43.5\% \text{ oats } (435 \text{ g/kg})$$

If 43.5% of the diet is oats then 56.5% is hay.

(2) Now calculate the protein, Ca and P contents of this simple mix from the information given in Appendix C.

Oats (12.1 MJ DE) contains (per kg):

95 g	crude protein
0.8 g	Ca
3.3 g	P

Hay (7.3 MJ DE) contains (per kg):

55 g	crude protein
3.5 g	Ca
1.7 g	P

Initial dietary composition (g)

	Crude protein	Ca	P
Contributed by oats:			
$\frac{43.5}{100} \times$ each oat value above	41.3	0.35	1.44
Contributed by hay:			
$\frac{56.5}{100} \times$ each hay value above	31.1	1.98	0.96
Total initial composition (g/kg)	72.4	2.33	2.40
Requirement (g/kg)	120.0	5.00	3.00
Deficit of minerals (g/kg)	—	*2.67*	*0.60*

Now calculate how much soya-bean meal is required to make good the protein deficit (other protein concentrates could be used in amounts that are inversely proportional to the lysine contents of the protein source under consideration and that of soya). About 2–3 g more protein than is required should be allowed for because minerals will also be added to the diet. Soya-bean meal from Appendix C contains 440 g crude protein/kg. As before:

$$72.4x + 440(1-x) = 120.0 + 3 \text{ g crude protein/kg}$$
$$72.4x + 440 - 440x = 123 \text{ g/kg}$$
$$-367.6x = -317 \text{ g/kg}$$

Change signs on both sides.

x = 0.862 or 86.2% oats plus hay, and therefore (100 – 86.2) soya forms the remainder, i.e. 13.8% soya-bean meal.

The soya contains more energy than oats and hay, but this will be approximately compensated for by the complete absence of energy in the mineral and vitamin supplement.

(3) Now 0.6 g P/kg is still required, being the deficit shown above. Dicalcium phosphate contains 188 g P/1000 g (from Appendix C). Therefore an addition of:

$$\frac{1000}{188} \times 0.6 \text{ g} = 3.19 \text{ g dicalcium phosphate } (CaHPO_4)/\text{kg}$$

total feed will provide the necessary P.

total feed will provide the necessary P.

Dicalcium phosphate from Appendix C also contains 237 g Ca/1000 g. Therefore 3.19 g provides:

$$\frac{237}{1000} \times 3.19 \text{ g} = 0.76 \text{ g Ca}$$

The original deficit was 2.67 g Ca and it is now:

$$2.67 - 0.76 \text{ g} = 1.91 \text{ g Ca}$$

This can be made up with limestone flour ($CaCO_3$) containing 360 g Ca/1000 g. Therefore:

$$\frac{1000}{360} \times 1.91 = 5.31 \text{ g } CaCO_3/\text{kg total diet}$$

(4) The diet should also contain 5 g common salt (NaCl)/kg and a proprietary vitamin/trace-element mixture suitable for horses.

(5) The complete diet is now as follows:

	g/kg	%
Oats	283	28.35 (43.5 – 13.8 – 0.32 – 0.53 – 0.5)
Soya-bean meal	138	13.80
Dicalcium phosphate	3.2	0.32
Calcium carbonate	5.3	0.53
Salt	5.0	0.50
Vitamins/trace elements	+*	+
Hay	565	56.50
Total		100

*A supplement may be provided as described in Chapters 6 and 8.

The composition of the concentrate portion of the ration is as follows:

		Per cent
Oats	$\frac{28.3}{43.5} \times 100$	65.1
Soya-bean meal	$\frac{13.8}{43.5} \times 100$	31.7
Dicalcium phosphate	$\frac{0.32}{43.5} \times 100$	0.74
Calcium carbonate	$\frac{0.53}{43.5} \times 100$	1.22
Salt	$\frac{0.5}{43.5} \times 100$	1.15
Vitamins/trace elements		0.1
Total		100

(6) The total daily ration is to be 9 kg and of this the above concentrate would form 43.5/100 × 9 = 3.9 kg daily. The remaining 5.1 kg hay could be given in excess, as some will be lost and horses would naturally consume their ration of concentrate before filling up on hay.

The concentrate ration should be divided into a minimum of two feeds per day and introduced gradually in increasing amounts until the full ration is provided. Small adjustments of the quantity can be made to allow for differences in condition between individuals. Where growing horses are being fed, particularly of faster growing breeds, the overriding concern must be the condition of the legs, and if there is any tendency towards contracted flexor tendons, epiphysitis or crooked legs the concentrate allowance should be reduced until the condition subsides (see Chapter 8 for details).

Appendix B
Common Dietary Errors in Studs and Racing Stables

The table below shows the range of dietary composition for foals and yearlings in ten studs where home mixes have been prepared and when restricted access to pasture of moderate quality is provided (% of total diet, air-dry basis; dashes imply none used).

	Dietary range		Typical poor-quality diets at specific studs			
	Weaned foals	Yearlings	Foals (1)	Foals (2)	Yearlings (1)	Yearlings (2)
Oats	37–70	11–58	48	—	57	9.5
Boiled barley	0–1.6	0–26	1.6	—	—	20
Flaked maize	0	0–6	—	—	—	2.5
Bran (wheat)	0–12	5–28	5	—	28	16
Coarse mix (sweetfeed) of low quality	0–43	0–43	—	42	—	—
Barley chaff	0–7	0–7	—	7	—	—
Cubes of Moderate quality	0–12	0–60	12	—	—	23
Soya-bean meal	0–4	0	—	—	—	—
Linseed (boiled)	0–7	0–9	1.6	—	5	7
Palm-kernel cake	0	0–3	—	—	—	—
Milk pellets	0–13	0	—	—	—	—
Locust beans	0	0–3	—	—	—	—
Egg	0	0–+	—	—	—	—
Molasses	0	0–2	—	—	—	—
Carrots	0	0–3	—	—	—	—
Grass hay	6–18	14–29	16	18	10	11
Lucerne hay	0–9.6	0	—	9.5	—	—
Limestone flour	0–2.2	0–2.2	—	2.2	—	—
Dicalcium phosphate	0–0.5	0	—	—	—	—
Vitamins, trace elements and minerals	+*	0–+	—	—	—	—
Pasture	12–36	14–40	16	22	—	11
Some chemical characteristics						
Crude protein (%)	10–16	10–17	11.1	10.0	11.8	11.7
Total lysine (%)	0.4–0.6	0.4–0.7	0.48	0.40	0.45	0.45
Ca (%)	0.32–0.9	0.22–1.2	0.33	0.65	0.23	0.44
P (%)	0.2–0.46	0.2–0.65	0.42	0.19	0.65	0.57

*Some sort of supplement was used in each of the ten stables.

General comments:

- widespread use of poor-quality hay for young stock;
- failure to rectify this by adjustment of concentrate composition;
- insufficient checks on rates of growth;
- failure to compensate for inadequacies of pastures.

Common errors:

- very variable protein and lysine intakes exacerbated by variable pasture quality and availability;
- sub-optimum protein : energy ratios for weaned foals;
- extreme variation in Ca intake;
- sub-optimum Ca : P ratios;
- excessive intakes of vitamins A and D, but possible inadequacies of several other vitamins;
- lack of control of growth curve leading to poor conformation, epiphysitis and abnormal alignment of legs;
- use of poorly formulated micronutrient supplements;
- use of more than one micronutrient supplement lacking complementary effects;
- pasture trace-element and other deficiencies, which contribute to metabolic and conformational problems.

Faults in diets (1) and (2) specified above
Foals:

(1) Insufficient protein, lysine and Ca.
Excessive use of vitamins A and D_3.
Deficiency of selenium and marginal zinc status.
(2) Insufficient protein and lysine.
Excessively wide Ca : P ratio.
Selenium depletion, zinc inadequacy and suspected induced manganese deficiency.
Poor conformation.

Yearlings:

(1) Marginal protein and lysine inadequacy.
Ca deficiency with adverse Ca : P ratio.
Epiphysitis evident.
Several vitamin inadequacies and suspect trace-element status.
(2) Lack of objectivity in ration formulation and unnecessary complexity.
Adverse Ca : P ratio.

Range of dietary composition for TBs in eight racing stables where home mixes are prepared (% of total diet, air-dry basis; dashes imply none used, or analytical value unavailable).

	Dietary range	Typical poor-quality diets at specific stables	
		(1)	(2)
Oats	32–59	49	51
Bran (wheat)	0–16	—	1.1
Coarse mix (sweetfeed)	0–20	—	—
Chaff	0–3.7	—	—
Cubes	0–24	—	2.0
Soya-bean meal	0–2.2	0.8	0.7
Linseed (boiled)	0–1.5	0.8	1.5
Molassed peat	0–0.5	—	0.4
Carrots	0–2	—	—
Grass pellets	0–6	—	—
Molasses	0–1.5	—	1.4
Grass hay	28–53	49	40.6
Limestone flour	0–1.1	—	1.1
Salt (sodium chloride)	0–0.1	—	0.07
Corn oil	0–0.57	0.11	—
Vitamins, trace elements and minerals	0–4	0.2	0.1
Some chemical characteristics			
Crude protein (%)	7.2–11.5	7.3	7.4
Total lysine (%)	0.35–0.5	0.36	0.40
Ca (%)	0.15–1.38	0.15	0.68
P (%)	0.24–0.43	0.25	0.28
K (%)	—	1.5	—
Na (%)	—	0.16	—
Mg (%)	—	0.18	—
Zn (mg/kg)	—	—	24
Mn (mg/kg)	—	—	46

General comments:
- oats and poor-quality grass hay frequently constitute over 90% of the diet and their composition is variable and unknown;

- several non-complementary micronutrient supplements are frequently used in the same diet;
- frequently insufficient common salt is consumed in hot weather;
- frequency of feeding is often insufficient;
- rate of ration change and of energy intake frequently inappropriate;
- notion that a rest on Sundays with changes in management and in feeding benefits the horse is misguided, in contrast to human benefits.

Common errors:

- variable protein and lysine intakes;
- sub-optimum protein : energy ratios;
- extreme variability in Ca intake;
- sub-optimum Ca : P ratios;
- excesses and variable amounts of vitamins A and D_3;
- inadequate allowances of folic acid and possibly of other water-soluble vitamins and of salt;
- incorrect quantities of vitamins, trace elements and minerals provided by most supplements.

Faults associated with (1) and (2) specified above:

(1) Lameness, metabolic upsets (e.g. azoturia, set-fast, gastric ulceration).
Dietary faults include excesses of vitamins A and D_3 and excess iodine.
Insufficient Ca and adverse Ca : P ratio.
Insufficient sodium and folic acid, marginal protein and probably too wide an energy : protein ratio.

(2) Abnormal blood characteristics.
Marginally low protein intake.
Insufficient allowances of zinc, folic acid and salt (for working horses).

Appendix C
Chemical Composition of Feedstuffs Used for Horses

Values for feedstuffs (a) assume 880 g DM/kg , apart from INRA data, (dashes imply no value available); values for forages (b) are typical rather than average values. Equations in (c), (d) and (e) can be used to estimate UFC values from the chemical composition of feeds.

(a) Chemical composition of feed stuffs (g/kg), assuming 880g DM/kg.

	Crude protein (g/kg)	MADC (g/kg DM)	MADC (88% DM)	Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)
Oats	96	103	91	45	100	170	240	40	0.7	3.0
Barley	95	92	81	18	50	70	167	25	0.6	3.3
Wheat	100	98	86	15	22	40	123	19	0.4	3.2
Maize	85	79	70	38	25	30	95	15	0.2	3.0
Sorghum (white)	10.6			25	27	60	201	18	0.3	2.7
Rice (rough)	73			17	90	—	—	52	0.4	2.6
Millet	88			13	277	—	—	81	—	—
White fishmeal	660			80	0	0	0	215	57.0	34.0
Dried skimmed milk (spray)	340			6	0	0	0	80	10.5	9.8
Linseeds	219			316	76	135	—	45	2.4	5.2
Exp. linseed meal	320	323	284	60	100	170	220	60	3.0	7.3
Extr. soya-bean meal	440	437	385	10	62	100	132	60	2.4	6.3
Extr. sunflower seedmeal	280–450	344	303	18	230	—	360–450	77	2.9	8.0
Exp. cottonseed cake	410			37	140	220	250	65	2.0	10.5
Solv. cottonseed meal	395	—	—	17	135	175	271	59	1.8	10.1
Groundnut meal extr. 48–50% CP	470	—	—	13	140	220	—	50	2.0	7.1
Maize gluten feed	210	191	168	25	70–80	—	—	70	2.5	7.5
Maize gluten feed (40%)	410	427	376	25	50	—	—	40	2.0	5.1
Maize gluten meal	572	—	—	22	35	72	98	29	0.5	5.3
Field beans	255	272	239	90	74	114	—	29	0.8	4.8
Peas	229	223	196	50	57	82	172	27	0.7	4.0
High-protein grassmeal	160			32	220	360	540	70	6.0	2.3
Lucerne meal	170	101–122	89–107	30	170–250	400	410	100	15.0	2.0
Brewer's yeast	450			10	30	—	—	65	4.4	13.3
Rapeseed meal extr.	350			24	130	—	210	70	6.5	11.0

K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC (g/kg DM)	UFC (88% DM)	α-Tocopherol (mg/kg)	Free folic acid (mg/kg)	Available biotin (µg/kg)	Thiamin (mg/kg)	Riboflavin (mg/kg)	Pantothenic acid (mg/kg)
5.0	3.2	10.9–12.1	0.99	0.87	9	0.12	50	17.0	1.7	12.0
5.0	3.1	12.8	1.16	1.02	7	0.11	12	5.0	1.8	14.0
4.2	2.8	14.1	1.26	1.11	9	0.12	4	4.0	1.1	11.0
3.1	2.6	14.2	1.33	1.17	9	0.06	65	2.0	1.5	6.0
4.1	2.4	13.0	—	—	7	0.13	—	3.0	1.1	12.0
—	2.5	11.1	—	—	—	0.20	15	2.5	0.9	—
—	—	—	—	—	—	—	—	—	—	—
8.3	48.0	14.1	—	—	13	0.22	100	5.0	5.0	8.8
16.0	29.0	15.1	—	—	10	0.60	330	4.0	20.0	30.0
9.4	7.7	18.5	—	—	2	—	—	7.0	2.5	—
11.0	11.3	13.9	0.92	0.81	1	—	—	8.0	3.0	12.0
23.5	26.0	13.3	1.06	0.99	2	0.57	280	6.0	3.3	14.0
14.0	13.0	9.5	0.79	0.70	10	—	415	34.0	3.0	—
15.0	14.0	12.8	—	—	5	0.22	250	7.0	4.0	9.5
14.4	16.3	11.0	—	—	—	1.5	100	6.5	4.8	9.9
12.0	15.0	12.5	1.01	0.89	2	0.5	300	8.0	3.0	40.0
9.7	5.8	12.8	0.96	0.85	7	0.20	85	2.0	2.2	14.0
1.0	11.2	13.1	1.14	1	7	0.2	90	0.4	1.8	12.0
4.0	9.7	13.3	—	—	—	0.22	110	4.4	1.3	11.7
11.0	17.0	13.1	1.04	0.92	—	—	—	—	—	—
11.0	15.8	14.1	1.15	1.01	8	—	—	—	1.4	—
21.0	8.0	9.6	—	—	25	1.80	300	3.0	15.0	25.0
22.0	8.2	9.0–10.0	0.57–0.68	0.5–0.6	30	3.00	400	3.7	14.0	25.0
18.0	29.3	12.2			2	2.31	300	100.0	39.0	105.0
—	20.0	11.5			—	—	575	1.0	3.6	9.0

	Crude protein (g/kg)	MADC		Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)	Ca (g/kg)	P (g/kg)
		(g/kg DM)	(88% DM)							
Wheatfeed	155	148	130	35	85	100	310	50	1.0	10.0
Wheat bran	155	130	114	30	110	120	380	70	1.0	12.1
Extr. rice bran	135			15	120	190	400	125	1.1	19.0
Oatfeed	50			22	250	400	635	70	0.8	1.2
Malt sprouts (culms)	250	257	226	20	140	—	390	65	2.0	7.0
Dried brewer's grains	180–250	251	221	62	140–170	180	405	38	2.6	5.1
Soya hulls	114	—	—	20	370	440	586	43	5	1.7
Beet pulp	100	45	40	10	174	340	392	50	7.0	0.8
Molassed beet pulp	90–120			1–6	130	—	300	60–80	6.0	0.7
Citrus pulp	61	—	—	43	118	195	213	63	16.9	1.1
Grass hay	42–80	40–60	35–53	25	330	380	670	50	2.9	1.7
Matua grass hay	90–170	—	—	40–80	—	270–350	520–700	60–140	—	—
Grass haylage	62	24	21	—	—	—	—	—	5.1	1.8
Big bale silage	98–100	52	46	35	232	273	—	62	4.1	3.3
Clamp silage	108–135	73	64	28	299	334	—	62	4.8–5.3	2.6–2.9
Lucerne hay mid-bloom	150–160	84	74	17	270	350	415	70	11.4	1.9
Maize silage	70	—	—	—	—	245	430	45	2.7	2.0
Clover/grass hay	60–100	50–70	44–62	27	330	380	600	65	4.0	1.7
Maize silage in cob	70	30	26	—	—	245	430–760	45	2.7	2.0
Spring cereal straw, barley	30	0	0	19	410	590	640	70	2.0	0.4
Vegetable oil	0	—	—	100	0	0	0	0	0	0
Molasses (cane)	45	34	30	0	0	0	0	85	7.2	1.0
Molasses (sugar beet)	110	83	73	0	0	0	0	83	2.5	0.3
Nutritionally improved straw*	45	—	—	12	340	490	—	—	4.0	1.0
Limestone flour	0	0	0	0	0	0	0	990	365	4
Dicalcium phosphate	0	0	0	0	0	0	0	100	238	187
Steamed bone flour	0	—	—	0	0	0	0	980	323	133

*Contains 30 g Na/kg.

Exp., expeller; extr., extracted; MAD, modified acid detergent; MADC, *matières azotées digestibles corrigées* (corrected digestible N matter); Solv. solvent extracted.

K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC (g/kg DM)	UFC (88% DM)	α-Tocopherol (mg/kg)	Free folic acid (mg/kg)	Available biotin (µg/kg)	Thiamin (mg/kg)	Riboflavin (mg/kg)	Pantothenic acid (mg/kg)
12.0	6.1	11.0	1.09	0.96	23	0.67	15	13.0	4.5	16.0
14.0	6.0	10.8	0.86	0.75	21	0.30	15	7.3	5.4	26.0
19.0	5.3	10.8	—		—	—	20	23.0	2.6	23.0
7.0	1.9	7.7			—	—	—	—	—	—
—	12.0	10.0	0.92	0.81	—	—	—	—	—	—
1.0	8.3	10.0	0.79	0.7	10	—	—	0.6	1.0	10.0
10	4.6	6.9	—	—	—	0.5	—	1.5	3.5	13
2.0	5.6	11.0	0.86	0.75	—	—	—	0.4	0.6	1.5
16.0	2.6	12.0	—	—	1	—	—	0.4	0.4	2
9.7	1.6	10.5	—	—	—					
17.0	3.0	5.0–7.4	0.41–0.55	0.36–0.48	7	—	—	1.5	10	—
—	—	—	—	—	—	—	—	—	—	—
—	—	8.0	—	—	—	—	—	—	—	—
—	—	8.7	0.65–0.72	0.57–0.63	—	—	—	—	—	—
15	—	9.2–10.5	0.60–0.68	0.53–0.60	—	—	—	—	—	—
16	—	7.6	0.5–0.63	0.44–0.55	—	—	—	—	—	—
—	3.8	4.49	—	—	—	—	—	—	—	—
19.0	5.2	6.0–8.0	0.51–0.57	0.45–0.50	8	—	—	2.0	15	—
—	3.8	4.49	0.85	0.75	—	—	—	—	—	—
19.0	0	6	0.36	0.32	—	—	—	—	—	—
0	0	35	—	—	—	0	0	0	0	0
27	—	11.4	1.07	0.94	0	0.08	100	0.8	1.8	25
45	—	12.0	1.06	0.93	0	—	—	—	1.4	8
18.0	—	—	0.46	0.40	0	—	—	—	—	—
0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0
0	0	0	—	—	0	0	0	0	0	0

(b) Forages

	Crude protein (g/kg)	MADC (g/kg DM)	MADC (88% DM)	Oil (g/kg)	Crude fibre (g/kg)	MAD fibre (g/kg)	NDF (g/kg)	Ash (g/kg)
Pasture:								
(1) First growth	167	103	91	38	176	194	400	97
(2) Second growth	180	130 to 92	114 to 81	35	195	215	425	90
Pure clover	200	112	99	31	176	229	275	97
Pure grass	176	98	86	13	264	290	450	85
(3) Blooming								
Pure clover	150	96	84	26	211	308	340	105
Pure grass	79	40	35	13	264	290	570	92
(4) Winter after close grazing until July, and free growth July–December	136			26	194	—		70

(c) Some factors necessary for the estimation of k_m, used in the calculation of UFC values and the estimation of UFC values from chemical composition of feeds.

Gross energy (GE, kJ/g) and k_m % of nutrients

	GE	k_m
glucose (GL)	15.65	85
acetate (C_2)	14.60	
propionate (C_3)	20.76	
butyrate (C_4)	24.94	
amino acid (AA)	23.44	70*
long chain fatty acids (LCFA)	39.76	80

*amino acid ME

So

$$k_m = 0.85E_{GL} + 0.80E_{LCFA} + 0.70E_{AA} + (0.63 \text{ to } 0.68)E_{VFA} - 0.14(76.4 - ED),$$

where E is the % of absorbed energy from glucose or lactate (GL) etc., ED is energy digestibility (%) of the feed and the last term of the equation corresponds to the cost of eating, a term not included for concentrate feeds. The percentage of absorbed energy from a typical horse diet of roughage and concentrate is represented by 9–41% GL, 45–82% VFA, 7–17% AA and 2–6% LCFA (Vermorel *et al.* 1997).

(d) Variations in molar proportions, energy content (E, kJ/g VFA mixture) and efficiency (k_m) of E utilization of VFA mixtures absorbed from the colon of the horse receiving diets of three crude fibre concentrations (Vermorel & Martin-Rosset 1997):

Crude fibre %	15	20	30
DM:			
C_2%*	65	68	73
C_3%	21	19	16
C_4%	14	13	11
E, kJ/g	17.91	17.70	17.24
k_m %	66.6	65.8	64.5

*The molar proportion of acetate (C_2) in the VFA mix increases with increasing dietary crude fibre (CF), C_2% = 0.54 CF (% DM) + 57. The energy expended in mastication also increases with increasing dietary crude fibre, Δk_m = –0.20 CF% + 2.50, so the above k_m values decrease further from 15% to 30% CF. Both the heat of fermentation and the energy wasted in metabolism are greater with C_2 metabolism than with the other VFA. Thus increasing fibre is associated with increased waste heat production and poor meadow hay has a k_m % of only 61, whereas that of maize is 80.

Ca (g/kg)	P (g/kg)	K (g/kg)	Lysine (g/kg)	DE (MJ/kg)	UFC (g/kg DM)	UFC (88% DM)	pH	NH_3N as % of total N
5.3	3.1	26	5.0	7.4–9.3	0.88	0.77	—	—
5.3	1.9	21	—	9.6	0.79	0.69	—	—
15.8	1.9	18	—	9.0	0.8	0.7	—	—
5.7	1.8	21	—	10.0	0.8	0.7	—	—
14.1	2.0	17	—	8.8	0.7	0.6	—	—
3.1	1.8	15	—	8.4	0.67	0.59	—	—
—	—	—		6.6			—	—

(e) Prediction of UFC value per kg dry matter (DM) of forages and concentrates from their cytoplasmic carbohydrate (CC), crude protein (CP), crude fibre (CF) and digestible organic matter (DOM), kg/kg DM, or digestible energy (DE, MJ/kg DM) content (Vermorel & Martin-Rosset 1997).

(i) Forages

$$UFC = -0.124 + 0.254CC + 1.330DOM,$$
$$\text{RSD } 0.012,\ R^2\ 0.988.$$

$$UFC = -0.056 + 0.562CC + 0.0619DE,$$
$$\text{RSD } 0.007,\ R^2\ 0.996.$$

(ii) Concentrates

$$UFC = -0.134 + 0.274CF - 0.362CP + 0.316CC + 0.0755DE,\ \text{RSD } 0.017,\ R^2\ 0.995.$$

NB: See Martin-Rosset *et al.* (1996c) for CC values, i.e. water-soluble carbohydrates, of feeds and reference to a comprehensive data source.

(iii) Compound feeds

$$UFCo = 1.333 - 1.684ADFo - 0.096CPo,$$
$$\text{RSD } 0.060,\ R^2\ 0.958.$$

NB: Martin-Rosset *et al.* (1996c) define UFCo as UFC per kg organic matter to allow for the high mineral content of some compound feeds. UFCo values of compound feeds must be increased by 0.02 UFCo for each 1% ether extract above 3.5% of feed.

(iv) Organic matter digestibility of forages (OMD %)
Three alternative methods are proposed for the determination of OMD % of horses, of which the second and third are the preferred methods.

(1) OMD % = 67.78 + 0.07088CP − 0.000045NDF^2 − 0.12180ADL, RSD 2.5, R^2 0.878, Martin-Rosset *et al.* (1996a); where NDF is neutral detergent fibre, g/kg DM and ADL is acid detergent lignin, g/kg DM.
(2) OMD % in horses predicted by near infrared spectrophotometry; RSD 1.80, R^2 0.93, Andrieu *et al.* (1996).
(3) $OMD\% = -29.38 + di + 2.3032CDMD - 0.01384CDMD^2$, RSD 1.90, R^2 0.927, Martin-Rosset *et al.* (1996b); where di = +4.12 for green forages, di = 0 for grass hays and di = −2.61 for legume hays; CDMD is the pepsin cellulase degradability (%) of DM.

Appendix D
Estimates of Base Excess of a Diet and of Blood Plasma

ESTIMATE OF BE OF A DIET FROM ITS POTENTIAL FIXED ION CONTENT

(See Chapter 9 for further details.)

$$(\text{Cations} - \text{anions})_{\text{absorbed}} - (\text{cations} - \text{anions})_{\text{excreted in urine}} - \text{H}^+ \text{ endogenous} = \text{BE} \qquad (1)$$

Account here is taken only of fixed ions absorbed from the diet and Eq. (2) shows the principal ones involved. (NB: fixed ions are those that cannot be degraded by metabolism.)

$$(\text{Cations} - \text{anions})_{\text{absorbed}} = \text{mEq}\,(0.95\,\text{Na} + 0.95\,\text{K} + 0.5^*\,\text{Ca} + 0.5\,\text{Mg}) - \text{mEq}\,(0.95\,\text{Cl} + 0.95\,\text{S} + 0.5^*\,\text{P}) \qquad (2)$$

*Approximate values which will be inversely related to dietary concentration.

In order to avoid a degree of arbitrariness a simplified balance of ions has been proposed and these are shown in Eq. (3) in relation to their optimum range in the diet of a horse in light work.

$$(\text{Na} + \text{K} - \text{Cl})_{\text{absorbed}} = 200\text{–}300\ \text{mEq/kg diet} \qquad (3)$$

(Note: $Na^+ + K^+ = 95\%$ of all cations in extracellular fluid and $Cl^- + HCO_3^- = 85\%$ of all anions.)

ESTIMATE OF BE OF BLOOD PLASMA FROM ITS BICARBONATE CONCENTRATION

Plasma bicarbonate (HCO_3^-) (mEq/L) at pH 7.4:

$$\cong [HCO_3^-]_{\text{measured}} - 10(7.4 - \text{pH measured})$$

BE of plasma at pH 7.4:

$$\cong [HCO_3^-] \text{ at pH } 7.4 - 24$$

(Normal bicarbonate of venous blood at pH 7.4 = 24 mEq/L.)

If the venous plasma of a horse was found to have a pH of 7 and $[HCO_3^-]$ was 30 mEq/L then $[HCO_3^-]$ at pH 7.4 would

$$= 30 - 10(7.4 - 7)$$
$$= 26\ \text{mEq/L}$$

Therefore

$$\text{BE} = 26 - 24 \cong 2\ \text{mEq/L}$$

NB: other organic acid anions could also be included in a BE calculation.

Glossary

Terms in the definitions that are themselves entries are shown in bold face.

aboral Away or remote from the mouth.

acidaemia An increased hydrogen ion concentration (acidity) and lowered blood bicarbonate, or decreased **pH** of the blood.

acidosis See **acidaemia**.

ACTH Adrenocorticotropic hormone (corticotropin) is secreted by the anterior pituitary gland, so controlling cortisol secretion by the adrenal cortex. The release of ACTH is in turn controlled by corticotropin-releasing hormone (CRH) secreted in the hypothalamus.

acute Applied to a metabolic upset, or a disease, which progresses rapidly to a climax followed by death or rapid recovery. Contrasts with a **chronic** condition or disease.

acute-phase proteins Specific serum proteins which increase rapidly in concentration (up to 100-fold) following infection, e.g. C-reactive protein, and which increase during inflammatory response, e.g. caeruloplasmin (ferroxidase), which also transports Cu (contains about 3% of the body's Cu).

adipose tissue The cells of this tissue readily store fat which is drawn on as a source of energy, especially when the blood levels of glucose and VFAs are low.

***ad libitum* feeding** A system in which feed supply is unrestricted at all times except during exercise. However, usually applies only to growing horses.

adrenal glands A pair of ductless glands, one situated near each kidney, and consisting of an internal medulla, which secretes the hormones epinephrine and norepinephrine (adrenaline and noradrenaline), and an external cortex, which secretes corticosterone, cortisol (glucocorticoids) and aldosterone.

aerobic In aerobic respiration, energy-yielding nutrients are broken down with the consumption of dissolved oxygen that has reached the tissue cells from the lungs. In anaerobic respiration the breakdown of these nutrients is incomplete, it yields less energy and occurs in the absence of oxygen.

afferent Afferent nerve fibres conduct impulses centripetally, e.g. from sense organs to the central nervous system.

agalactia Failure of the secretion of milk.

agglutination The clumping together of particulate antigen (foreign substance) in the presence of homologous antibody (defence substance).

air dry Under UK regulations, feed that has been allowed to dry without heating in the air contains 100–140 g moisture/kg.

aldosterone The main mineralocorticoid hormone secreted by the adrenal cortex, promoting the reabsorption of Na and therefore water by the kidney tubules.

alfalfa See **lucerne**.

alimentary canal/tract See **gastrointestinal tract**. In addition, it includes the buccal cavity and oesophagus.

alliesthesia The perception of an external stimulus as pleasant or unpleasant depending on internal stimuli. A particular stimulus may be perceived as pleasant at one time and unpleasant at another. For example, negative water alliesthesia occurs when there is reduced consumption concomitant with severe dehydration and hyperthermia, leading to hypovolaemia and further hyperthermia. This might occur despite no apparent inadequacy in the thirst mechanism.

anamnesis The faculty of memory of a previous environmental experience, including abnormal sensations.

alkalaemia A decreased hydrogen ion concentration, or elevated **pH**, of the blood, irrespective of changes in blood bicarbonate. Normal arterial blood pH is 7.5.

allergy A condition of exaggerated susceptibility, or sensitivity, to a specific substance, usually, but not necessarily, containing a specific protein. Exposure, especially to large amounts of the allergen, through inhalation, ingestion, injection or even by skin contact, may cause respiratory difficulties, sneezing, a skin rash or diarrhoea of increasing severity through repeated contact.

ALT (also **GPT**) Alanine aminotransferase (EC 2.6.1.2), formerly called glutamic pyruvate transaminase. The activity of this enzyme in the blood plasma shows a similar reaction to that of **AST**, particularly in respect of exercise and muscle damage.

amino acids These N-containing compounds are the building-blocks of proteins. There are some 25 different kinds, 10 of which are known as dietary indispensable (essential) nutrients, the most critical of which is lysine.

α-amylase An important digestive enzyme in the digestion of starch and other polysaccharides containing three or more α-1,4-linked D-glucose units. It hydrolyses the α-1,4-glucan links.

anabolism The process of synthesis of complex organic molecules in the body from simpler precursors (cf. **catabolism**).

anaemia A condition in which there is a reduced number of red cells and/or a reduced haemoglobin content of the blood. The volume of packed red cells is reduced when the equilibrium between blood loss, through bleeding or destruction, and blood production is disturbed.

analgesic A pain-relieving substance.

angiogenesis The development of vessels; in the text it refers to blood vessels.

anion gap The anion gap is the difference between measured cations and measured anions. It quantitatively reflects those ions that are unmeasured because of the principle of electroneutrality, in which the sum of all anions and all cations must be equal in both plasma and urine.

$$[Na^+]+[K^+]+UC=([HCO_3^-]+[Cl^-])+UA,$$

so

$$[UA]-[UC]=[Na^+]+[K^+]-([HCO_3^-]+[Cl^-])=\text{Anion gap},$$

where UA = unmeasured anions and UC = unmeasured cations, both in mEq/L. It is used to distinguish titration and secretion forms of metabolic acidosis. The gap increases as the pH falls in the titration form (e.g. increased lactic acid), whereas the gap is normal in secretion form. In acid urine (pH < 6.5) the bicarbonate concentration is negligible and there is normally a negative correlation between anion gap and urinary ammonium ion.

anorexia Lack, or loss, of appetite for feed.

anthelmintic A substance used to destroy parasitic worms.

antibiotic A chemical substance produced by and obtained from living cells, especially of lower plants such as moulds, yeasts or bacteria, that is antagonistic to, or destroys, some other form of life. It may be used to destroy infectious organisms.

antibody A specific substance (immunoglobulin) found in the blood, or in certain secretions, as a response to the antigenic stimulus of bacteria, viruses, worm parasites and certain other foreign substances. An antibody has a specific amino acid sequence and can combine specifically with the inducing foreign entity (antigen), helping to inactivate it.

antigen Any substance that is capable, under appropriate conditions, of inducing the formation of antibodies and of reacting specifically with them.

antihistamine A drug that counteracts the effects of histamine or certain other amines that cause inflammation.

antitoxin A substance found in blood serum or other body fluid that is antagonistic to a particular **toxin**. For therapeutic or protective use it may be injected into horses to neutralize the toxin of a particular disease, but as it does not stimulate the horse to produce its own antitoxin, its (passive) effects may last for only a few weeks.

arrhythmia Any variation from the normal rhythm of the heart beat.

artery A vessel containing smooth muscle through which blood passes away from the heart to the various parts of the body.

ascarids Roundworms. A group of large parasitic intestinal parasites in the phylum Nematoda.

They are 15–20 cm long, white, and infest primarily young horses, as those over three to four years of age have usually developed immunity (for life cycle see Chapter 11). Large numbers of adult worms in the intestines can cause impactions, intestinal perforations and colic.

ascites Effusion and accumulation of serous fluid in the abdominal cavity.

ash The ash content of feed is determined by ignition of a known weight of it at 500°C until the carbon has been removed. The residue represents approximately the inorganic constituents of the feed – principally Ca, K, Mg, Na, S, P and Cl. Some feeds, particularly those contaminated with soil, may contain a significant amount of silica.

AST (SGOT) Aspartate aminotransferase (EC 2.6.1.1), formerly known as serum glutamic oxaloacetic transaminase. This enzyme is released into the blood following damage to liver or muscle cells so that the blood level rises sharply. Plasma activity of the enzyme usually increases after hard exercise. The normal maximum blood plasma level in adult horses is 250 iu/l.

ataxia Failure of muscular coordination, or irregularity of muscular action, resulting in a staggering gait. It may result particularly from exhaustion or from pathological change in the nerves.

ATP Adenosine triphosphate. ATP mediates the transfer of energy from the breakdown of glucose and fatty acids (exergonic reactions), for the synthetic processes of growth, milk secretion, etc. (endergonic reactions) and for muscular action. ATP is split by the enzyme ATPase, with the liberation of inorganic phosphate.

autogenous Self-generated. It refers particularly in the text to antibodies produced by the dam to blood proteins of the foetus that happen to be circulating in her blood. If colostrum is then taken by the foal within the first 12 hours, there will be an antibody reaction with the foal's blood proteins.

autonomic nervous system This is the self-controlling part of the nervous system – it is not subject to direct influence by the conscious brain. The sympathetic system (thoracolumbar outflow) and the parasympathetic system (craniosacral flow) have largely antagonistic actions. The combined system is of importance in regulating the activities of many glands, the smooth musculature of the GI tract and elsewhere, and the heart and blood vessels.

availability of minerals The proportion of a nutrient mineral supplied in the feed which, at a stated level of inclusion and level of feeding, can be absorbed and utilized by the horse to meet its net requirement. It is not necessarily synonymous with true digestibility.

azotaemia An excess of urea and other nitrogenous compounds in the blood.

azoturia An excess of nitrogenous compounds in the urine. Considered synonymous with exertion myopathy. It frequently occurs within a short interval of beginning exercise after a rest of two or three days. It is characterized by a reluctance to move and muscular spasms, as excessive lactic acid accumulates in the muscles. The muscles of the hindquarters become tense and there is a tendency to knuckle-over at the fetlocks. At some stage the horse will pass quantities of urine from light-Burgundy-wine to dark-coffee colour. Sometimes the urine is retained and requires removal with a catheter.

base excess and base deficit Base excess is defined as the titratable base using a strong acid to titrate to a pH of 7.4 at a pCO_2 of 40 mmHg at 37°C. Base excess is not a measure of total buffering power and it may be derived, under certain circumstances, from pH and bicarbonate alone. Base deficit is negative base excess and is defined as the titratable acid using a strong base, also to titrate to pH 7.4 (NB: normal pH: arterial 7.347–7.475; venous 7.345–7.433).

β-carotene See **carotene**.

big-head A condition seen in horses and ponies given a ration based on cereals, bran and poor hay without adequate mineral supplementation. The bones of the upper jaw and face are particularly enlarged, owing to a replacement of their normal mineralized structure by fibrous connective tissue.

bile duct The duct carrying bile, synthesized by the liver, to the duodenum, where the bile facilitates emulsification of fats and contributes to the production of an alkaline reaction of the intestinal contents.

biological values The amount of N retained by an animal per unit of N absorbed from the GI tract and from a given feed protein.

biuret A simple organic compound containing three N atoms, which has been put forward as a source of dietary NPN. The structurally

related compound urea contains only two N atoms per molecule.

bleeders Broken blood vessels. This usually refers to the loss of blood through the nose after hard exercise. Blood is lost from small broken blood vessels in the lungs, or nasal passages, in about half the population of healthy TBs during hard exercise; the condition requires no treatment unless large blood vessels are involved.

blood counts These normally include the numbers of red blood cells (erythrocytes) per unit volume, the packed cell volume (haematocrit), or proportion of red blood cells by volume in the total blood measured after the blood has been centrifuged, and the haemoglobin content of the total blood. Three other characteristics of the red cells (MCV, mean corpuscular volume; MCH, mean corpuscular haemoglobin and MCHC, mean corpuscular haemoglobin concentration) are calculated from these basic data. The numbers of white blood cells (leucocytes) per unit volume and the differential count (proportion of each type of white cell) may also be measured.

borborygmus Rumbling noise caused by the propulsion of gas through the intestines.

bot fly The larvae, or maggots, of *Gastrophilus* spp. cause chronic gastritis and loss of condition in grazing animals. *Gastrophilus intestinalis* lays eggs of pin-head size on the horse's hair in the summer. When the horse licks itself, the eggs are taken into the mouth and hatch there or in the stomach. On rare occasions they cause perforation of the stomach and death (see Chapter 11).

botulism A rapidly fatal motor paralysis caused by the ingestion of the toxin of *Clostridium botulinum*, a spore-forming anaerobic bacterium, which proliferates in decomposing animal tissue and sometimes in plant material. The toxin (a di-chain protein, molecular weight 140 000) is the most neurotoxic substance known. In Chapter 10, botulism is referred to in cases in which horses have consumed **silage** that has been subject to an abnormal fermentation. The toxin seems to inhibit irreversibly the release of acetylcholine from peripheral nerves, and so impedes neuromuscular transmission. A flaccid descending paralysis develops.

brachygnathia Abnormally short lower jaw bone, creating difficulties, for example, in grazing.

bradycardia Slow heart beat.

branched chain amino acids (BCAAs) Three amino acids, valine, leucine and isoleucine, with aliphatic side chains.

broken wind, heaves These are outdated expressions applied to long-standing respiratory diseases in which doubled expiratory effort is a feature. The causes include bacteria, viruses and, rarely, lung tumours. A common cause is an allergic reaction, and that related to the inhalation of spores and particles of the moulds *Micropolyspora faeni* and *Aspergillus fumigatus,* discussed in Chapter 11.

bruxism Rhythmic or spasmodic grinding of teeth.

buccal cavity The cavity of the mouth between the cheeks bounded at one end by the lips and at the other by the pharynx.

buffers Substances that in aqueous solution increase the amount of acid or alkali that may be added without changing the pH, or degree of acidity or alkalinity. In general, a buffer is made up of two components: (1) a weak acid, e.g. H_2CO_3, and (2) its corresponding base, HCO_3^-. Arterial blood is well buffered around a pH of 7.5.

cachexia A profound and marked state of constitutional disorder; general ill health and malnutrition.

caecum A cul-de-sac intercalated between the small intestine and the colon. In the adult horse it is about 1.25 m long, with a capacity of 25–30 L (see Chapter 1).

calcitonin A hormone synthesized by the parafollicular cells of the **thyroid gland**. It is secreted when the serum concentration of Ca ions rises, promoting the deposition of Ca in bones, and so counteracting the action of **parathyroid hormone**.

calculi Urinary calculi consist of accumulations of mineral substances in the urinary tract. They form in the bladder, less frequently in the urethra and rarely in the kidneys. They are commonly rough and yellow-brown and are composed of calcium carbonate. These are seen more commonly in horses on high-roughage diets or on pasture. The less common phosphate calculi are smooth and white and occur with high-cereal rations. A low intake of water may predispose the horse to calculi and the signs include difficulty in urination and incontinence.

calorie A unit of energy, being the amount of heat required to raise 1 g water 1°C. 1000

calories = 1 kilocalorie (kcal). The joule (J) has now been adopted as the unit of energy in nutrition; 4.184 kJ = 1 kcal.

carbohydrates Compounds composed of carbon, hydrogen and oxygen and including the sugars, starches and other storage carbohydrates and the structural (fibre) carbohydrates – cellulose and hemicelluloses; also pectins, gums and mucilages. Lignin is included in structural fibre but is not strictly a carbohydrate.

cardiac output The volume of blood expelled by each ventricle per minute. The stroke volume is the volume of blood discharged by each ventricle at each beat (i.e. the cardiac output divided by the heart rate). The cardiac output of the left ventricle after birth is 2–8% higher than that of the right, so the value quoted represents the mean of these two.

carotene Green plants contain a number of yellow carotenoid pigments, the most important of which are α-, β- and γ-carotenes and hydroxy-β-carotene. The most potent of these, β-carotene, is converted to vitamin A by the intestinal wall.

carpus The 'knee' of the forelimb of the horse between the **radius** above and **metacarpus** below.

catabolism The breaking down of tissue nutrients and components to less complex molecules (cf. **anabolism**).

catecholamines A group of similar compounds having sympathomimetic action. They include epinephrine, norepinephrine and dopamine. The latter two serve as neurotransmitters while the former two have hypertensive effects, stimulating smooth muscles.

cathartic A purgative, or medicine, that quickens the evacuation of the bowels (intestines).

cation The positively charged elements of electrolytes, which include all metals and hydrogen ions.

cellulolysis The breaking down, or digestion, of cellulose. Horses and other mammals depend on certain bacteria in their intestinal tracts to carry this out as they do not secrete enzymes capable of it.

cellulose A structural carbohydrate (fibre) of plant cells.

choke Obstruction to the passage of food through the pharynx and oesophagus, either partial or complete. This is frequently caused by a mass of dry impacted feed.

cholestasis Suppression of bile flow. This may be unusually caused by gallstones and is then termed *cholelithiasis*.

chondrocyte A mature cartilage cell embedded within the cartilage matrix.

chronic Long-continued; the opposite of **acute**.

CK (or **CPK**) See **Creatine kinase**.

coenzyme A non-protein organic compound, which may be a vitamer, the presence of which is required by an enzyme for the catalysis of the particular reaction to occur.

coldblooded horse In Europe two types of horse evolved – the light short-legged Celtic pony and the Great Horse of the Middle Ages (the large powerful but slow heavy horse). Present-day breeds whose major blood lines derive from either of these types are termed 'coldblooded' (cf. **hotblooded horse**).

colic Abdominal pain. Where this originates from part of the GI tract it is termed 'true colic', but where it derives from one of the other vital organs or muscles it is defined as 'false colic'.

colon Made up of the great colon, which originates from the caecocolic orifice and terminates where it joins the small colon, and the latter, which continues to the rectum. In the adult horse the great colon is 3–3.7 m long with an average diameter of 20–25 cm, whereas the small colon has a diameter of 7.5–10 cm and a length of about 3.5 m.

colostrum Secreted by the mammary gland of the mare shortly before foaling (parturition) and for about the first 24 hours after the birth of the foal. It is rich in **γ-globulins**, which comprise the antibodies that the foal absorbs undigested into its blood during the first 12–18 hours of life, providing it with a measure of protection from disease.

compounded feeds Balanced mixtures of ground or otherwise processed feedstuffs, to which appropriate supplements of vitamins, minerals and trace elements have been added.

concentrates The portion of the horse's ration, or feedstuff, that is rich in starch, protein or both, and contains less than 15–17% **crude fibre**.

conductance Capacity for conducting and/or transferring energy (e.g., heat) from one medium, or body, to another. The unit of electrical conductance is the mho.

contagious Transmissible from one horse to another.

contracted tendons Hyperflexion or flexural deformity of limbs. The condition in foals ranges from uprightness of hind or forelegs to knuckling over at the fetlock and/or inability to extend knee joints. Not uncommon in TB foals. Correction of slight abnormalities may include the fitting of boots and in more severe cases severance

of fibres of the flexor muscles and tendons or desmotomy of the superior check ligament.

convulsions A violent involuntary contraction, or series of contractions, of the voluntary (skeletal) muscles.

coprophagy The eating of faeces by an animal. Within three weeks of birth, foals will eat their dam's faeces and thereby acquire the species of bacteria and protozoa necessary for the rapid development of a normal microbial population in their GI tract so that invasion by harmful microorganisms is partially inhibited.

cornea The transparent structure that forms the anterior part of the eyeball.

coronary band (coronary matrix) Runs around the horse's foot just below the hair line and forms part of the sensitive structures from which the wall grows. A permanent defect in the hoof wall usually follows injury to the coronary band.

corpus luteum 'Yellow body.' A yellow glandular mass in the ovary, formed by an ovarian follicle that has matured and discharged its ovum. It contains carotenoids and secretes progesterone.

cortical bone Compact bone forming the cylinder of the shaft, or diaphysis of long bones, and resulting mainly from peri-osteal ossification.

corticosteroids Comprise the natural glucocorticoids, cortisone and hydrocortisone hormones secreted by the **adrenal cortex** and synthetic equivalents, e.g. prednisone, prednisolone and fluoroprednisolone, used in the treatment of inflammation, shock, stress and, in other animals, ketosis.

cortisol rhythm The release of cortisol, caused by stress, leading to the mobilization of fats and amino acids, with the production of sugars by gluconeogenesis from certain deaminated amino acids and glycerol.

creatine kinase (CK) This enzyme (EC 2.7.3.2) is measured as an indicator of muscle damage.

The normal maximum plasma level in adult horses is 105 iu/L.

creatine phosphate (phosphocreatine) The fixation of energy in the form of ATP is a transitory phenomenon, and any energy produced in excess of immediate requirements is stored more permanently in compounds such as muscle phosphocreatine (PCr). As ATP becomes depleted, more is generated from phosphocreatine by a reverse reaction.

creatinine The normal excretory breakdown product of muscle creatine found in horses' urine. As the quantity produced daily is relatively constant for a particular horse, being proportional to muscle mass, its concentration in urine is used in the assessment of the level of other substances excreted in urine (see Chapter 12). Blood levels of creatinine rise dramatically following renal failure.

creep feed A feed, normally dry pellets, offered to nursing foals behind a barrier, which does not allow the mare access, but permits the foal to enter.

crib-biting The horse or pony grips a fixed horizontal surface (e.g., the edge of the manger, fence, etc., Plate 11.1) with its incisors, contracts the strap muscles of the ventral throat and typically emits a grunting sound. Young idle animals are said to acquire the habit from individuals in their company with a confirmed habit. It can initiate repeated bouts of mild colic, while excessive wear of the incisor teeth may compromise grazing powers. Sometimes a crib strap is fitted around the upper neck of persistent offenders (see also **wind-sucking**).

crimping A term used for the pressing of cereal grains between corrugated rollers to rupture the kernels and to increase digestibility slightly.

croup That part of the horse's hindquarters lying immediately behind the loins. The 'point of the croup' is its highest part and corresponds to the internal angles of the ilia.

crude fibre The feed residue identified after subjecting the residual feed from ether extraction to successive treatments with boiling acid and alkali of defined concentrations. The crude fibre contains cellulose, hemicellulose and lignin, but it is not an accurate measure because it underestimates the structural components of vegetative matter (see **NFE**).

curb A swelling about 100 cm below the point of the hock, owing to undue strain causing a sprain of the calcaneocuboid ligament, or of the superficial flexor tendon.

cyclic AMP cyclic Adenosine monophosphate. An intracellular hormonal mediator formed from ATP under the influence of stimulating hormone.

DE See **Digestible energy**.

deamination When amino acids are present in excess of needs for tissue protein synthesis, or when the horse is forced to catabolize tissue to maintain essential functions, amino acids may be degraded to provide energy. This occurs mainly in the liver and to some extent in the kidneys. The first step in the oxidative degradation of amino acids is the removal of the amino group, a process called deamination.

This group is then either transferred to a keto acid to produce another amino acid or is incorporated into urea.

decarboxylation In the text, this term is principally restricted to reactions of amino acids. Bacteria – e.g., in the intestinal tract and in silage during the early stages of fermentation of the clamp – elaborate enzymes (called carboxylases), which act upon amino acids to yield amines and carbon dioxide. This implies a loss of dietary protein value and the amines, including histamines and tryptamine, may have toxic effects following absorption.

dehydrated Something from which most of the moisture has been removed. Dehydration extends the shelf-life of feed and greatly retards the rate of, or inhibits, mould spoilage.

dialysis The process of separating crystalloids from colloids in solution according to the difference in their rates of diffusion through a semipermeable membrane; colloids pass very slowly or not at all.

diaphragm A thin muscular partition or membrane that separates the thorax (chest cavity) from the abdomen. During its contraction air is drawn into the lungs and when it relaxes air is expelled.

diaphysis The shank of a long bone between the ends, or epiphyses, which are usually wider and articular.

dicoumarol An anticoagulant with similar properties to warfarin except that its action has a slower onset, a longer duration and a less predictable response. When ribbed melilot or yellow sweet clover (*Melilotus officinalis*), or white melilot or white sweet clover (*M. alba*) plants are damaged by weather, badly harvested, or when, as hay, they become mouldy, coumarin contained in the sweet clover is broken down to dicoumarol. Both yellow and white sweet clover are found in pastures in the UK, and white sweet clover is grown as a forage crop in North America and the former USSR.

digestible energy (DE) The gross energy (or heat of combustion) of a feed minus the gross energy of the corresponding faeces, expressed as MJ or kJ/kg total feed. Synonymous with apparent digestible energy.

dispensable amino acids Amino acids that are synthesized in the tissues of horses, and/or are made available from synthesis by gut microorganisms, in amounts sufficient to meet tissue requirements of horses without a dietary source.

distal Remote, farthest from the centre, or origin, as opposed to **proximal**.

diuresis Increased secretion of urine. A diuretic drug induces diuresis.

dopamine A neurotransmitter and an intermediate in the synthesis of norepinephrine.

duodenum The first (proximal) part of the small intestine, connected to the stomach. In the adult horse it is 1 m long with a diameter of 5–10 cm.

dyschondroplasia Disordered, or abnormal, cartilage formation.

dysphagia Difficult in swallowing.

dysplasia Abnormality in the development of cells.

dyspnoea Difficult or laboured breathing.

dystrophy Dystrophy of muscles causes their atrophy and degeneration.

electrolytes Substances that, in water solution, break up into particles carrying electrical charges. The principal electrolytes from a nutritional point of view are Na^+, K^+, Cl^-, HCO_3^-, Ca^{2+}, Mg^{2+} and HPO_4^{2+}.

emphysema A swelling, or inflation, of the chest which is caused principally by the presence of air in the intra-alveolar tissue of the lungs following rupture of the alveoli.

endochondral ossification Bone formation within cartilage, cf. periosteal ossification.

endocrine gland, hormones and related secretions An endocrine gland secretes a specific substance (hormone) that is released directly into the circulatory system (blood, lymph or neurosecretory channels) and which influences metabolism at sites in the body distant from the gland. Two other types of hormonal secretion are: *autocrine* secretion, which influences the function of the cells that produce it; and *paracrine* secretion, which is produced by one type of endocrine cell and influences the secretions of nearby cells that are not its target. An *exocrine* gland is one that secretes outwardly via a duct opening onto an internal or external epithelial surface.

endogenous Arising from within the horse, i.e. excluding the lumen of the GI tract.

endotoxaemia Presence in the blood of endotoxins, which are non-protein, lipopolysaccharide fragments of the cell wall of Gram-negative bacteria. Per milligram they are much less toxic than are exotoxins, but appear to play a crucial role in certain diet-related disorders of bowel origin, including forms of **colic** and **founder**.

enema A fluid for injection into the rectum or small colon for cathartic or diagnostic purposes.

enterotoxaemia Presence of toxins in the blood, produced and secreted in the intestines by certain bacteria,

e.g. *Clostridium perfringen*. Enterotoxins produced in large quantities by this organism are specific for cells of the intestinal mucosa and cause enteritis. This anaerobic organism is found in soil and the consumption of relatively small numbers of the spores in herbage is usually without remarkable effect (see also **botulism**).

epinephrine See **catecholamines**.

epiphysis A head of a long bone joined to the shaft, or diaphysis, during growth by a cartilaginous growth plate, or metaphysis.

epiphysitis Inflammation of an epiphysis or of the cartilage that separates it from the shaft during growth.

epistaxis A nose bleed. Blood may be present in the nostrils through being coughed up from broken blood vessels in the lung. Small losses are a normal phenomenon after a race, but persistent losses may occur in chronic bronchitis.

epithelial cells All the body surfaces, including the external surface of the skin, the internal surfaces of the digestive, respiratory and genito-urinary tracts, the inner coats of vessels and ducts of all secreting and excreting glands, are covered by one or more layers of cells called epithelium or epithelial cells.

ergot A fungus that infects and finally replaces the seed of a cereal or grass, especially the sclerotium of *Claviceps purpurea* (ergot of rye). This ergot is small, hard, black and resembles mouse droppings. Ergot contracts the arteriolar and other unstriped muscle fibres. Its toxins are used to arrest haemorrhage after parturition and internal injury. The persistent consumption of ergot of rye sufficiently decreases blood flow to cause gangrene (typically of the ears, tail and legs). Several ergot toxins cause abortions. After eating large amounts of ergotized hay, horses become dull and listless, a cold sweat breaks out on the neck and flanks, the breathing is slow and deep, the body temperature is subnormal and the pulse weak. Death occurs during a deep coma within the first 24 hours. Lesser amounts over a longer period may cause diarrhoea, colic, trembling and loss of condition.

erythrocytes See **red blood cells**.

essential amino acids See **indispensable amino acids**.

ether extract Chemical substances that are soluble in, and extracted by diethyl ether, or 40:60 petroleum ether (which one is used should always be specified.

eutrophication The accidental or deliberate promotion of excessive growth (multiplication) of an organism to the disadvantage of other organisms in the same ecosystem by oversupplying it with nutrients. In the case of rivers and ponds/lakes, principally by run-off or seepage of excess P and N from the surrounding land, leading to excessive plant (principally algal) growth and decay.

exertion myopathy **Azoturia** myohaemoglobinuria. An acute condition in which affected muscles, especially of the hindquarters, become hard to the touch, and the hind limbs rapidly become stiff and weak or staggering. There is a tendency to knuckle-over at the fetlocks. The risk is greatest in horses that have been in continuous work, abruptly rested for a few days on full feed and then returned to work.

expansion, extrusion This relies on the cooking effects of superheated steam injected into a slurry compressed against a die face by a revolving worm and the subsequent rapid fall in pressure during extrusion. Material is subjected to a temperature of around 120°C for about a minute.

extracellular space The fluid space, or volume, within the body that is external to the cells.

fasciculation Local contraction, or bundling, of muscles.

fatty acids Composed of a hydrocarbon chain of one to more than 20 units attached to a carboxyl group. In the formation of storage fats, fatty acids are neutralized by the trihydric alcohol glycerol. Both neutral fat and fatty acids circulate in the blood (see also **free fatty acids** and **volatile fatty acids**).

feed That which is given to the animal to consume, whether a single feedstuff or a mixture, but excluding water. Not synonymous with **ration**.

fermentation Decomposition of organic substances by microorganisms. In the horse's GI tract this refers especially to bacteria, yeasts and ciliate protozoa; the first being the most significant.

fertilizer Inorganic fertilizers are plant nutrients prepared synthetically or mined as minerals. Organic fertilizers are sources of the same nutrients of animal and vegetable origins bound in **organic** form.

fetlock The horse's 'ankle' joint in hind- and forelimbs between the metacarpus or metatarsus (cannon bones) and the first phalanx (long **pastern** bone).

FFA See **free fatty acids**.

fibre See **crude fibre**.

fibrin The insoluble protein formed from fibrinogen by the proteolytic action of thrombin during normal clotting of blood.

filled legs Oedema or puffiness of the legs caused by abnormally large amounts of fluid (exudate) in the intercellular tissue spaces beneath the skin. The most common cause in healthy horses is a period of inactivity in a box following a season of hard exercise, particularly where corn or other concentrated feeds are being given. However, it is not protein poisoning. Oedema can also arise from diseases of the heart, liver or kidneys, or from long-standing malnutrition involving a diet impoverished in protein.

flexor tendons Muscles are attached to the long bones of the legs by extensor tendons, which extend or straighten the leg when the muscle contracts, and by flexor tendons, which flex joints by contraction of the appropriate muscles.

founder, laminitis A painful disease of the feet in which there is apparently a transitory inflammation followed by congestion of the laminae of the hooves. It is most frequent in ponies but can be induced readily in both horses and ponies by a sudden increase in the starch or protein content of the diet. Sometimes all four feet are affected, sometimes only the forefeet, and occasionally only the hind feet, or a single foot. Affected feet feel hot and the body temperature may rise. The stance is unnatural, affected forefeet are thrust forwards and the horse is reluctant to move.

free fatty acids (FFAs) During work, storage fats are mobilized when lipase enzymes catalyze the production of fatty acids, splitting them from glycerol and leading to a rise in the blood plasma concentration of both components.

frog Horny central part of the lower surface of the foot, subject to thrush, an infection by bacteria and fungi occurring in wet unhygienic stables when there is poor routine foot care.

fundus gland region of stomach A large region of the stomach containing glands of two types of cells and lying distal to the oesophageal and cardiac regions and proximal to the pyloric gland region.

fungal units Fungi contaminating feed produce a mycelial mat of fine threads and fruiting bodies from which spores are shed. When badly affected feed is disturbed, the mycelial threads may break up into small particles and new growth can be initiated by each of these particles or by germination of spores. The fungal unit is any particle from which new growth can be started.

furlong Forty poles, one-eighth of a mile. Originally the length of the furrow in the common field. The side of a square of 10 statute acres. Equals the Roman stadium, one-eighth of a Roman mile.

β-galactosidase Neutral or brush-border lactase. This enzyme is normally present in the intestinal juice of young horses. It is necessary for the cleavage of milk sugar (lactose) to glucose and galactose, which can then be absorbed into the blood.

gamma globulin A protein fraction of the plasma globulin, which has a slow moving electrophoretic mobility. Most antibodies are gamma globulins.

gastro-intestinal tract Stomach and intestines. Sometimes used synonymously with alimentary tract, which is the entire tube extending from the lips to the anus.

Gastrophilus See **bot fly**.

GGT Gamma-glutamyltransferase (EC 2.3.2.2) is released into the blood following liver damage. The blood activity of this enzyme is elevated in liver cirrhosis, pancreatitis and renal disease. The normal range of plasma activity is 0–41 iu/L and seneciosis may cause a rise up to 80–280 iu/L.

ghrelin Hormone produced mainly by P/D1 cells lining the fundus of the human stomach and epsilon cells of the pancreas that stimulates appetite. Plasma ghrelin levels increase before and decrease after meals in humans, but levels are less variable in horses, possibly due to the greater role of the large intestine. It is the counterpart of the hormone leptin. Ghrelin is also produced in the hypothalamic arcuate nucleus, where it stimulates the secretion of growth hormone from the anterior pituitary gland. Receptors for ghrelin are expressed by neurons in the arcuate nucleus and the ventromedial hypothalamus. The ghrelin receptor is a G protein-coupled receptor, formerly known as the growth hormone secretagogue receptor). Ghrelin is only biologically active in its acylated form and physiological concentrations of plasma insulin might not be involved in the regulation of plasma ghrelin in horses.

glossitis Inflammation of the tongue. Observed in folic-acid deficient horses treated for protracted periods with sulphonamide drugs (see ‘Folic acid’, Chapter 4).

glucagon Polypeptide hormone secreted by the alpha cells of the islets of Langerhans in the pancreas in response to hypoglycaemia. It stimulates glycogenolysis in the liver and opposes the action of **insulin**.

glucogenic Giving rise to, or producing, glucose. According to a long-used classification, amino acids are ketogenic if (like leucine) they are converted to acetyl-CoA and produce ketones in the blood when fed to a starved animal. Glucogenic amino acids such as valine promote the synthesis of glucose and glycogen when fed to a starved animal.

gluconeogenesis The formation of glucose from amino acids by various routes within the body that include pyruvic acid and lactic acid, but not via acetyl-CoA.

glutathione peroxidase ($GSH\text{-}P_\infty$) The activity of this enzyme (EC 1.11.1.9) in horse erythrocytes is used as an indication of selenium status. It is determined as µmol **NADPH** oxidized in 1 min by 1 mL erythrocytes. In TBs, the activity should be approximately 25–35 units/mL red cells.

glycolysis The major pathway whereby glucose is metabolized to give energy is a two-stage process; the first stage, called glycolysis, can occur anaerobically and yields pyruvate.

glycosuria The presence of abnormal amounts of glucose in the urine. It arises from failure of renal tubular reabsorption or from an abnormality of hormone status, as in diabetes mellitus.

goitrogenic A term applied to substances in certain feeds, derived, for example, from members of the plant genus *Brassica* (family Cruciferae) which, if consumed persistently in large quantities, cause goitre (an enlargement of the **thyroid gland**). A deficiency of iodine in the diet will cause a similar condition, especially in young horses.

gossypol A toxin found with two other toxic pigments in the pigment glands of cotton seeds. It is a polyphenolic binaphthalene derivative. It is inactivated by heating, but in this procedure it combines with lysine, reducing the protein value. Selective breeding has produced glandless seed, not widely available.

grass sickness A disease of horses, ponies and donkeys that seems not to be contagious. It occurs mainly in the summer months among grazing animals on certain pastures in Europe in a peracute form in which death occurs within 8–16 hours of initiation after some periods of great violence. In a subacute form the horse is dull, listless and salivates. The bowel becomes impacted and distended. Food material may appear in the nostrils. There seems to be some loss of motor control of the GI tract. Muscles of the back are hard, the horse is 'tucked-up' and twitching over the shoulders occurs. Recovery is uncommon (see Chapter 10).

growth plate See **metaphyseal plate**.

haem-agglutinins Now called lectins, contained in *Phaseolus* spp., including *P. vulgaris.* Severe GI disorders can be caused by these substances when given in high dietary concentrations. They are proteins of which concanavalin A from jack beans is used medically as a mitogenic agent to preferentially agglutinate cancer cells.

haematocrit The proportion of blood by volume made up of cells, especially red cells, and expressed as a percentage. It is determined by centrifugation of blood samples containing an anticoagulant. Also called packed cell volume (PCV).

haematopoiesis The formation and development of blood cells.

haemoglobin The oxygen-carrying red pigment of the red cells (erythrocytes) of blood. A conjugated protein consisting of the protein globin combined with an iron-containing prosthetic group (haem).

haemolysis The rupture of red blood cells.

haemolytic icterus of foals Caused by the absorption of colostral antibodies that destroy the foal's red blood cells (see Chapter 7).

haustral Referring to the haustra, or sacculations, of the colon.

haylage Originally registered trade name for a silage now containing a high proportion (50–65%) of dry matter made from wilted forage, precision-chopped to approximately 12–25 mm nominal length and ensiled in a Harvestore tower silo. The name has acquired a more liberal definition to include unchopped vacuum-packed material.

heating feed A concentrated feed, which is readily digestible and fermentable and which leads to a rapid rise in blood metabolites, waste heat production and probably to some increase in metabolic rate.

heaves See **broken wind**.

Heinz body anaemia Heinz bodies appear as small round inclusions, composed of denatured haemoglobin, within red blood cells. This irreversible damage is usually caused by oxidation. These red cells are attacked by macrophages in the spleen, where the precipitate is removed and the cells eliminated, leading to Heinz body

anaemia. This anaemia can be caused by the consumption of onions in cats, dogs and various primates, and apparently by excessive garlic given to horses. Thiosulphate compounds in the bulbs have been identified as the cause.

hepatic encephalopathy A degenerative disease of the brain resulting secondarily from advanced disease of the liver, or where a portocaval shunt occurs.

hepatitis Inflammation of the liver.

hind-gut The large intestine, consisting of the caecum and colon.

hives Large numbers of small bumps, or raised areas, about 0.5 cm diameter under the skin, which eventually form scabs. They appear suddenly and are caused by an allergic response to specific components of feed, to drugs or to insect bites. The cause (**antigen**) can usually be determined by allergy tests, and recovery follows removal of the source from the diet or general environment. The condition sometimes arises when 'rich' feeds are suddenly introduced to the diet.

hock The tarsal joint between the **tibia** and **metatarsus** (cannon bone) of the hindlimb.

homeostasis Stability of the normal body states. Refers frequently to the constancy of pH and chemical composition of extracellular fluids.

hormone A discrete chemical substance secreted into the body fluids by an endocrine gland which influences the action of a tissue, or organ, other than that which produced it.

hotblooded horse Hot- and warmblooded horses (cf. **coldblooded** horses) are those derived to a significant extent from breeds originating in Mediterranean countries, which came to be called Arabian, Barb and Turk. Modern breeds of this type include TB, Arabian, Standardbred, American Saddle Horse, Morgan, Quarter Horse and Tennessee Walker. The principal North European warmblooded breeds are the Hanovarian, Trakehner, German Holstein, Dutch Warmblood and Oldenburg breeds.

hyper-, hypo- Prefixes: hyper- signifying above normal or excessive and hypo- meaning below.

hyperaemia Engorgement with blood.

hypercalciuria Abnormally large amounts of Ca in the urine; hypocalciuria is abnormally small quantities of Ca.

hypercapnia Excess carbon dioxide in the body fluids.

hyperglycaemia An elevated blood glucose concentration.

hyper- and hypokalaemia Abnormal level of plasma potassium. Hypokalaemia may be caused by a combination of inadequate dietary K with excessive losses from the body. The causes of both hypo- and hyperkalaemia can be metabolic derangements, when there are abnormal shifts of K^+ between intracellular and extracellular space, as in acidosis.

hyperlipidaemia A general term for elevation of any, or all, of the lipids in the plasma, including hyperlipoproteinaemia and hypercholesterolaemia, whereas hyperlipaemia refers specifically to an elevation of the triacylglycerols (TAGs), previously known as triglycerides.

hyperparathyroidism Abnormally increased activity of the **parathyroid** glands, causing loss of Ca from the bones.

hyperplasia Abnormal multiplication, or increase, in the number of normal cells in normal arrangement in a tissue.

hyperplasmia An excess in the proportion of plasma to cells in the blood.

hyperpnoea Abnormal increase in the depth and rate of the respiratory movements.

hypersensitivity An exaggerated reaction to a foreign agent. These immune responses are classified as immediate or delayed, or as Types I–IV.

hypertension Usually refers to high arterial blood pressure, which may be confined to a specific circulation such as pulmonary or renal.

hyperthermia Abnormally elevated body temperature.

hypertonic A body fluid with a concentration, or osmotic pressure, above normal (more than isotonic).

hypertrophy The enlargement or overgrowth of an organ or tissue by the increase in size of its individual cells.

hypervolaemia An abnormal increase in the volume of circulating blood, which may be of plasma and/or of red-cell mass. In studies referred to in Standardbred trotters, the term refers to red cell hypervolaemia, as plasma volume was within normal limits. The elevated volume of red cells should not be confused with an elevated mean cell volume (MCV), which is an abnormal mean increase in the volume of individual red cells, typically observed in alcoholism.

hypocalcaemia Abnormally low plasma concentration of Ca.

hypocupraemia Abnormally low plasma concentration of Cu.

hypokalaemia Abnormally low plasma concentration of K.

hypoglycaemia Abnormally low plasma concentration of glucose.

hypoglycaemic shock, insulin shock Occurs when blood glucose falls below the normal range, causing nervousness, trembling and sweating.

hypomagnesaemia Abnormally low plasma concentration of Mg.

hyponatraemia Abnormally low plasma concentration of Na.

hypotension Abnormally low blood pressure.

hypovolaemia Abnormally decreased volume of circulating blood plasma.

hypoxaemia Deficient oxygenation of the blood.

hypoxia A reduction below physiological limits of the oxygen supply to tissues despite adequate perfusion by blood.

icterus Jaundice. A yellowish discoloration of the visible mucous membranes – eyes, mouth, nostrils and genital organs. It can also be detected in blood plasma of stabled animals receiving a diet low in pigments and can be caused by certain infections resulting in destruction of the red blood cells and release of the haem pigment (haemolytic jaundice) or by liver damage.

IGF-1 See **insulin-like growth factor-1**.

ileum The distal portion of the small intestine extending from the jejunum to the caecum.

ileus Functional obstruction of aboral GI transit.

immunity Resistance to infection by an organism, or to the action of certain poisons. Immunity can be inherited, acquired naturally or acquired artificially.

immunoglobulin Specific proteins, found in blood, **colostrum** and in most secretions, produced by plasma cells in response to stimulation by specific antigens, which in turn are inactivated. The antigens may be carried by, or released from, bacteria, viruses or even certain parasites.

imprinting An inborn tendency of a neonatal animal to attach itself to a group of objects, or a single object, such as its mother.

indispensable (essential) amino acids Those amino acids that are not synthesized or otherwise made available in the tissues of the animal, for example, synthesis by gut microorganisms in amounts sufficient to meet tissue requirements and which must therefore be supplied in the feed.

infarct An area of tissue **necrosis** due to local anaemia resulting from obstruction of the blood circulation. In the text this refers to the effects of migrating **strongyle** larvae on the mesenteric blood vessels, which causes death of a segment of the intestines.

inorganic Not a precise term but can be taken to refer to the ash content of the body remaining after it has been incinerated, which removes H, C and N as oxides. The **minerals**, in an oxidized form, remain, together with a very small proportion of carbonates.

insulin A protein hormone synthesized by the islet cells of Langerhans in the pancreas and secreted into the blood where it regulates glucose metabolism. It is deficient in diabetes mellitus.

insulin-like growth factor-1 (IGF-1 or somatomedin) A protein with a topology resembling insulin and associated with its function, but produced by the liver and other tissues. Plasma concentration of IGF-1 in man appears to depend on growth hormone (GH) secretion by pituitary tissue and on nutritional status. It stimulates protein synthesis and functions in cell differentiation and growth. IGF-1 acts as an **endocrine** hormone via the blood and locally as a paracrine/autocrine factor, suppressing GH secretion and sensitizing insulin action.

international unit (iu) As applied to vitamins, is the internationally agreed unit of potency for a particular vitamin that may have several molecular forms. The unit is now being displaced in favour of gravimetric measurements of each molecular form or vitamer.

intracellular space The fluid volume within the cells of the body as distinct from the extracellular fluid space. Movement of ions, water, glucose, etc. from one to the other is under metabolic control.

intussusception Prolapse of one part of the intestine into the lumen of an immediately adjoining part.

ischaemia A deficiency in the blood supply to a tissue, organ or part of the body.

isoantibody An **antibody** generated in the body in reaction to an **isoantigen**. An example is one found in the dam's blood and **colostrum** in response to a foetal protein that has entered her bloodstream.

isoantigen An antigen in the body that will induce the production of an antibody against itself.

isoenzymes Isoenzymes, or isozymes, are physically distinct, e.g. immunologically so, but they all catalyse the same reaction.

isotonic solutions Those solutions that have the same concentration or, more specifically, the same osmotic pressure.

iu See **international unit**.

jaundice See **icterus**.

joint-ill A disease in which organisms enter the body by way of the unclosed navel causing

abscesses to form at the umbilicus and in some of the joints.

joule SI (Système Internationale d'Unités) unit of energy: 4.184 J = 1 calorie. The joule is defined as the energy expended when 1 kg is moved 1 m by a force of 1 Newton (1 Nm); 1 N = 1 kg.m.s^{-2}, or J.m^{-1}; a watt is power, 1 W = N.m.s^{-1}, or kg.m^{2}.s^{-3} = J.s^{-1}. The kilojoule (kJ) = 10^3 joules and the megajoule (MJ) = 10^6 joules.

keratinize To become horny. Keratin, the principal protein of epidermis, hair and the hoof, is very insoluble and contains a relatively large amount of sulphur. Increased keratinization of epithelia can occur under physiological conditions and under pathological conditions, e.g. vitamin A deficiency.

knuckling over Usually refers to flexing of the **fetlock** joint owing to contraction of muscles and tendons or ligaments behind the cannon.

Kupffer's cells Phagocytic cells lining the walls of the sinusoids of the liver and which form a part of the reticuloendothelial system.

labile Unstable (chemically).

lactase An enzyme that hydrolyses the milk sugar lactose to form glucose and galactose. The enzyme accomplishing this in the intestine of the horse previously known as β-galactosidase.

laminitis See **founder**.

latent period The period or state of seeming inactivity between the time of stimulation and the start of the response, e.g. the interval between the injection or absorption of antigen and the first appearance of antibody.

lathyrism A condition characterized by sudden and transient paralysis of the larynx, with near suffocation of the horse, caused by β-aminopropionitrile in *Lathyrus sativus* (Indian pea) and other *Lathyrus* spp., including *L. odoratus* (sweet pea).

LDH Lactic dehydrogenase (EC 1.1.1.27). A tissue enzyme that has several different forms or isoenzymes. It catalyses the transfer of H^+ with the formation of pyruvic acid and the reduction of pyruvate in the presence of NADH. The activity of LDH in the blood is elevated during and following strenuous exercise and following tissue damage.

legumes Plants of the family Leguminosae or Fabaceae, which include useful forage species (e.g. the clovers and **lucerne**) and seed species (e.g. soya beans, peas and field beans).

leptin (Greek *leptos* meaning thin) is a 16 kDa protein hormone that plays a key role in regulating energy intake and expenditure through appetite control. It is one of the most important hormones produced by adipose tissue. It interacts with six types of receptor (LepRa–f). LepRb is the only isoform that contains active intracellular signaling domains. This receptor is present in a number of hypothalamic nuclei. Leptin binds to the ventromedial nucleus of the hypothalamus, known as the 'appetite centre' and signals to the brain that the body is satiated, when it is present in the blood at relatively high levels. In the horse the plasma leptin response seems to occur 6–7 h after an observed increase in plasma insulin concentration. In humans, and other domestic and laboratory animals that have been investigated, these receptors may be down-regulated by excess food or up-regulated by a scarcity of food. Down-regulation apparently increases the circulating levels of leptin (i.e. reduced receptor sensitivity) and the propensity to later obesity. The circulating level also changes diurnally with feeding, and, assuming a standard sensitivity, a raised level decreases energy intake. Genetic changes in the leptin molecule affect hypothalamic receptor sensitivity to leptin. In the text, low plasma leptin concentrations are correlated with anoestrus in mares.

leucocytosis A transient increase in the number of white cells in the blood.

level of feeding Weight of complete dry diet eaten daily, not confined to energy. Strictly it should be given as a proportion of metabolic body size ($BW^{0.75}$), or, more crudely, per 100 kg BW.

ley Grass and clover seeds sown as part of a cropping rotation, and usually ploughed-up after one to six years. The distinction from permanent pasture is not absolute.

ligament A tough fibrous band supporting viscera, or which binds bones together. In different situations, ligaments are cord-like or in flat bands, or, in forming the joint capsule, they are in sheets, preventing dislocation.

ligand An organic molecule that donates the necessary electrons to form coordinate/covalent bonds with metallic ions. Heme is the ligand in haemo- and myo-globin. The term is also used to indicate a molecule that reacts to form a complex with another molecule, e.g. ligand–enzyme complexes, where the ligand influences the conformation and activity of the enzyme.

light horse A loose term for small and large 'lady's' hacks, usually of mixed breeding, which may include TB and English Arabian. An alternative definition includes all riding horses of the present day in this classification, which excludes heavy draught horses and ponies.

lipases A class of enzymes, members of which are found in digestive secretions and in body tissues. Individual lipases have as major functions the hydrolysis of fats to yield fatty acids, monoglycerides, glycerol and cholesterol, and the hydrolysis of phospholipids.

lipids A group of substances found in plant and animal tissues, insoluble in water but soluble in common organic solvents, including petroleum, benzene, ether and chloroform. The crude fat of feed is the material extracted using light petroleum.

lipolysis The decomposition of fat into glycerol and fatty acids and, in the case of, e.g., the phosphoglyceride lecithin, into phosphoric acid and choline as well.

lipopolysaccharides (LPS) Large molecules, also known as lipoglycans, consisting of a lipid and a polysaccharide joined by a covalent bond; they are found in the outer membrane of Gram-negative bacteria, act as endotoxins and elicit strong immune responses in animals.

lucerne (alfalfa) A legume, *Medicago sativa*. A perennial forage crop with a strong tap root, which grows well on light alkaline soils in warm climates. Weeds must be kept at bay during its establishment.

lymphatics A system of vessels that drains lymph from various body tissues and conveys it to the bloodstream and that conveys neutral fats from the small intestine after they have been absorbed.

lysine An **essential amino acid**, the concentration, or frequency, of which in most vegetable proteins limits their biological value. L-Lysine hydrochloride is sometimes used as a feed additive to make good any deficit in the dietary protein.

MAD fibre Modified acid detergent fibre. A fibre fraction of feed determined by the MAD fibre procedure isolates principally the lignocellulose complex. This complex is the fraction of plant material that has most influence on energy digestibility of feed. In comparison, during the chemical procedure for **crude fibre** determination, a considerable amount of the lignin may become soluble and hence lost from the residue, so leading to an overestimation of the digestible fraction of feed.

maintenance At the maintenance level of feeding, the requirements of the horse for nutrients for the continuity of vital processes within the body, including the replacement of obligatory losses in faeces and urine and from the skin, are just met so that there is no net gain or loss of nutrients and other tissue substances by the animal.

maize (corn) *Zea mays*. A member of the grass family, the Gramineae, the seeds, both cooked and uncooked, of which constitute an excellent high-energy cereal grain for horses. The above-ground vegetative parts, at the milky-grain stage, can be made into a good silage for horses.

mandible Lower jawbone. In the adult horse this bone has sockets for three incisors, one canine, three premolars and three molars on each side in the male (in the female the canines are usually absent, or rudimentary). Its grinding movements are controlled by powerful muscles.

ME Metabolizable energy. The digestible energy (**DE**) of a unit weight of feed less the heats of combustion of the corresponding urine and gaseous products of digestion.

meconium A dark-brown, viscid, semi-fluid or hard material which accumulates in the intestines of the foal prior to birth. It should be discharged soon after birth. The **colostrum** has a natural purgative action on it.

mesentery Membranous peritoneal fold attaching the intestines to the dorsal wall of the abdomen.

metabolism A term embracing the chemical processes of **anabolism** and **catabolism** in the body.

metacarpus The part of the forelimb lying between the carpus (wrist) and the digit (**cf. metatarsus**). There are three metacarpal bones: the central, or third, metacarpal (cannon) is the largest; the other two are rudimentary metacarpals (splint bones).

metalloproteinase Enzyme proteins binding a metal, including caeruloplasmin, cytochrome oxidase, lysyl oxidase and the superoxide dismutases.

metaphyseal plate The region of linear growth of the long bones of growing horses lying between the epiphysis, or head, and the diaphysis, or shank.

metaphysitis Inflammation of the metaphysis, seen as swelling or bumps adjacent to the joints of leg bones.

metatarsus The part of the hind limb lying between the tarsus, or hock, and the digit. It is similar in

layout to the **metacarpus**, with a central large metatarsal bone (cannon) and rudimentary metatarsals (splint bones).

methaemoglobin A modified form of haemoglobin in which the iron has been converted from the ferrous to the ferric state in which it can no longer combine with, and transport, oxygen. The lesions can occur following the administration of large doses of certain drugs or after the consumption of nitrites in feed or water.

micronization The cooking of cereal and legume seeds under ceramic burners that emit infrared irradiation in the 2–6 µm waveband, resulting in a rapid internal heating of the seed and a rise in water-vapour pressure, during which the starch grains swell, fracture and gelatinize.

minerals The essential elements in the diet other than carbon (C), hydrogen (H) and nitrogen (N). Of particular interest are the macrominerals calcium (Ca), phosphorus (P), magnesium (Mg), potassium (K), sodium (Na), chlorine (Cl) and sulphur (S), and the microminerals iron (Fe), zinc (Zn), manganese (Mn), copper (Cu), cobalt (Co), iodine (I), selenium (Se) and fluorine (F). The microminerals are also known as trace elements. Some other elements, such as chromium (Cr), nickel (Ni) and molybdenum (Mo), are required in very small amounts. The mineral elements are required for incorporation into compounds different, in the main, from those in which they appear in the diet, whereas C, H and N are required as constituents of preformed organic nutrients. A crude approximation to the mineral content of the diet is obtained from the measurement of the **ash** content of the feed.

mitochondria Minute bodies occurring in the cytoplasm of cells (except bacteria and blue-green algae, or cyanobacteria). They exert important regulatory functions both on catabolic and biosynthetic sequences. They are the seat of the citric acid-cycle, the β-oxidation pathway and of oxidative phosphorylation.

MJ 1000 kJ or 10^6 joules.

modulus The Young Modulus, *E* is a material property that describes stiffness, used here to measure biomechanical properties of joint cartilage. It is the ratio of stress, in units of pressure, to strain, which is dimensionless. The SI unit of modulus of elasticity is the pascal; the practical units are megapascals (MPa) or gigapascals (GPa or kN/mm^2), where N = the Newton. Poisson's ratio is the ratio of the relative contraction strain, or transverse strain (normal to the applied load), divided by the relative extension strain, or axial strain (in the direction of the applied load).

mycotoxins Substances produced under specific conditions by certain fungi or moulds. Their chemical form and their effects on animals are wide ranging. These include hormone-like effects, disruption of intestinal, renal and hepatic function, tumour induction and antibacterial effects. Most antibiotics are mycotoxins. The best-known mycotoxins include: aflatoxin, produced by *Aspergillus flavus*; T-2 toxin, produced by *Fusarium* and *Myrothecium* spp.; ergotoxin, produced by *Claviceps purpurea*; zearalenone (F-2), produced by *Fusarium* spp.; dicoumarin, produced on melilots or sweet clover by *Aspergillus* and *Penicillium* spp.; and vomitoxin produced by *Fusarium* spp. Several mycotoxins are harmful to the horse in normal husbandry conditions.

myoglobin A small oxygen-carrying protein of muscle tissue containing the pigment haem with an atom of iron at its centre. Severe muscle damage leads to its appearance in the urine in **azoturia**. Being a much smaller molecule than haemoglobin, it passes the glomerular filter much more readily, causing a dark-brown stain to the urine. Precipitation of myoglobin in the renal tubules, as with haemoglobin, may contribute to terminal **uraemia**.

myopathy A disease of a muscle.

natriuresis The urinary excretion of abnormal amounts of sodium.

N-balance (N-retention) The net gain of nitrogen (N) by the animal. N in feed (N in faeces + N in urine) − loss of ammonia expired in air per unit time.

NDF Neutral detergent fibre (NDF) is a measure of the indigestible structural carbohydrates of plant cell walls defined according to the method of determination of Van Soest (1963) and Van Soest *et al.*(1991) and described in Point 3 of Annex 1 to EC Directive 73/46/EEC(b). The procedure is also used in methods described in the booklet *Prediction of Energy Values of Compound Feeding Stuffs for Farm Animals* published by the Ministry of Agriculture, Fisheries and Food Publications, London SE99 7JT.

NE Net energy, the energy value of animal product formed, or of body substance saved at or below maintenance, per unit weight of feed consumed. NE = metabolizable energy − heat increment.

necrosis Death of a cell, or group of cells, which is in contact with living tissue.

NEFAs Non-esterified, or free, fatty acids produced from the hydrolysis of triacylglycerols, or triglycerides.

nematode The Nematoda are a class of tapered cylindrical helminths, the roundworms, of the phylum Aschelminthes. Not all are parasitic.

nephritis Inflammation of the kidney; a focal or diffuse proliferative, or destructive, process which may involve the glomerulus, tubule or interstitial renal tissue (cf. **nephrosis**).

nephrosis Any disease of the kidney, especially one characterized by degeneration of the renal tubules (cf. **nephritis**).

nephrotoxic Toxic or destructive to kidney cells.

neurotransmitter A chemical, e.g. norepinephrine, acetylcholine, dopamine, etc., released from the axon terminal of a presynaptic neuron on excitation, that travels across the synaptic cleft to excite or inhibit the target cell.

NFE Nitrogen-free extractive. Measured in g/kg feed, this is numerically evaluated as 1000 – (moisture + ash + crude protein + ether extract + crude fibre). NFE may include some cellulose, hemicellulose, lignin, sugars, fructans, starch, pectins, organic acids, resins, tannins, pigments and water-soluble vitamins if these components were present in significant amounts in the original feed.

non-protein nitrogen (NPN) The determination of the crude-protein content of a feed assumes that the protein content is generally 6.25 times the determined N content. However, there are many compounds in feed, including nucleic acids, creatine and others, that have no protein value, but which are included in the calculation. Young herbage is rich in amino acids and nitrates that are components of NPN, although amino acids have an equivalent protein value.

nuts, cubes Compounded horse feed mixtures that have been compressed into solid cylinders 3–10 mm in diameter and two to three times as long by forcing the mixture through the holes of a metal die. The mix may or may not have been previously steamed in a kettle.

nutritional secondary hyperparathyroidism (NSHP) The increased secretion of **parathyroid** hormone as a compensatory mechanism directed against a disturbance in mineral homeostasis induced by nutritional imbalances. A loss of Ca from the bones is induced, resulting in a condition marked by pain, spontaneous fractures, muscular weakness and osteofibrosis (see Chapters 3 and 11).

obligatory loss Usually refers to the minimal inevitable loss of a nutrient, by excretion from the body, at low dietary intakes of that nutrient.

oedema The presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body. In the text, applied especially to an accumulation in the subcutaneous tissue.

oesophagus The gullet, a muscular membranous tube or canal extending from the pharynx to the stomach.

oestradiol The most potent naturally occurring ovarian and placental oestrogen which prepares the uterus for the implantation of the fertilized ovum and which induces and maintains the female secondary sex characteristics.

oestrous cycle A cycle in the mare typically lasting 21 days in the breeding season in which there is a pattern of physiological and behavioural events under hormonal control. The cycle, which forms the basis of sexual activity, has two components: oestrus (heat) in which the mare is receptive to the stallion and the egg is shed, and dioestrus, a period of sexual quiescence.

OM See Organic matter.

omentum A fold of the peritoneum extending from the stomach to the adjacent organs in the abdominal cavity.

oncotic pressure As used in the text this refers to oncotic pressure of plasma, which is the osmotic pressure owing to the colloids present, principally albumin, that counterbalance the capillary blood pressure.

open knees A dished concave appearance to the front of the 'knee' or carpus joint caused by **epiphysitis** immediately above the knee.

orad Toward the mouth.

oral Of the mouth.

organic Complex molecules containing at least C and H and synthesized by living tissue.

organic matter (OM) Organic matter: **DM** (g/kg) –**Ash** (g/kg DM).

osmolality A solution that has 1 osmole solute dissolved in 1 kg water has an osmolality of 1 Osm/kg (1/1000 Osm dissolved/kg water has an osmolality of 1 mOsm/kg; cf. **osmolarity**, the more practical measure, which is the osmolar concentration expressed as osmoles per litre solution rather than per kilogram water. For dilute solutions, as in the body, the quantitative difference is less than 1%.

osmolarity The total number of dissolved particles, or osmoles, in water solution. Fluids with an

osmolarity greater than that of body fluids are hypertonic while those with a lower osmolarity are hypotonic. Osmolarity depends on molar concentration and not upon equivalents per litre (mEq/L). So, a Mg^{2+} concentration of 30 mEq/L, is 15 mmol/L, or 15 mOsm/L, while 100 mL 0.6 M $NaHCO_3$ provides 60 mmol Na^+ and 60 mmol HCO_3^-, or 120 mOsm, on the assumption that the salt is completely dissociated. The osmolarity of body fluids is about 285 mOsm/L.

osmotic pressure The pressure that can be exerted when water moves from one solution to another of higher concentration through a semipermeable membrane, such as that of red cells. Solutions that have the same osmotic pressure, i.e. are isotonic with the osmotic pressure of red cells, are used for injections; otherwise the cell membrane would become distended and burst (haemolysis). High-energy compounds are consumed when a nutrient is required to move against an osmotic pressure gradient.

osteoarthritis Chronic, multiple, degenerative joint disease characterized by degeneration of the articular cartilage, hypertrophy of the bone at the margins, and changes in the synovial membrane.

osteoarthrosis Chronic arthritis of a non-inflammatory character.

osteocalcin A non-collagenous protein secreted by the osteoblast. It plays a role mineralization and calcium ion homeostasis and is used as an indicator of bone formation. It may also be a hormone acting on the pancreatic beta cells.

osteochondritis Inflammation of both bone and cartilage.

osteoid The organic matrix of young bone.

osteomalacia Softening of bones in adults from a deficiency of vitamin D or minerals. There is an increased amount of uncalcified bony matrix (osteoid).

oxalate An organic acid anion that combines with Ca and some other positively charged dietary minerals to form a very insoluble precipitate that inhibits digestion and absorption (see 'Tropical grassland', Chapter 10). Oxalates circulating in the blood also react with ionized blood Ca and form a crystalline deposition in the kidneys.

oxidative stress The cellular injury and pathologic changes that occur when there is an imbalance favouring oxidants over antioxidants within a living organism. Evidence for oxidative stress in ischemia–reperfusion injury of the gastrointestinal tract, recurrent airway obstruction, exercise, osteoarthritis, equine motor neuron disease, and pituitary pars intermedia dysfunction are reviewed by Soffler (2007).

oxygen deficit O_2 deficit is defined by the O_2 equivalent of the difference between ATP supplied oxidatively from pulmonary O_2 consumption and ATP utilized in the exercising muscle during intense exercise. A major portion of O_2 deficit in the horse arises from anaerobic glycolysis. Thus, the deficit is the O_2 required to fully metabolize the products of anaerobic glycolysis, supplied when intense exercise stops.

Oxyuris equi Pinworm. A nematode intestinal worm, which is not a serious hazard to horses. It causes intense irritation of the anal region and this encourages tail-rubbing and biting. Piperazine compounds are effective.

pancreas A gland in the abdominal cavity that secretes a juice containing digestive enzymes into the duodenum (see Chapter 1) and secretes the hormones **insulin** and **glucagon** into the bloodstream.

Parascaris equorum A nematode intestinal worm that commonly infects foals, yearlings and horses under three years old. It causes intestinal problems, colic, coughing and nasal discharge (see Chapter 11).

parathormone Parathyroid hormone synthesized by the **parathyroid gland**.

parathyroid gland An endocrine gland located in the upper neck adjacent to the thyroid gland. When serum concentration of calcium ions falls, parathyroid hormone is secreted into the blood. It induces mobilization of bone Ca, increases Ca absorption in the intestine, reabsorption of Ca by the renal tubules and urinary excretion of P. Its effects are counteracted by that of **calcitonin**. See also **secondary nutritional hyperparathyroidism**.

paresis (general) A condition short of complete paralysis in which certain muscles are relaxed and weak. If it is more generalized the animal cannot support itself or stumbles. Sometimes there is slight paralysis (if there has been some injury to or effect on certain motor nerves) and an inability to make purposeful movements.

paresis (parturient) **Paresis** following **parturition** is associated with **hypocalcaemia**, although it is not a normal characteristic of mares. It involves incomplete or slight paralysis of motor functions; whereas **tetany** is sharp flexion of the motor muscles, a spasm or twitching, owing to hyperexcitability of nerves and muscles caused by decreased extracellular ionized

calcium, usually with parathyroid hypofunction.

parturition The act, or process, of foaling.

pastern The region of the leg between the fetlock and the hoof in both fore- and hind limbs, formed by the long and the short phalanges which create the pastern joint. The third phalanx is in the hoof.

pelage The hairy coat of the horse and other mammals.

peristalsis The rhythmic involuntary muscular contractions of the alimentary canal by which it mixes and propels ingesta towards the rectum.

petechial haemorrhage Small, subcutaneous blood spot caused by the effusion of blood from a capillary. Numerous spots are frequently present in tissue reacting to toxins, such as endotoxin.

pH The symbol used to express the H ion concentration of water solutions. It signifies the negative logarithm of the H ion concentration in gram molecules (moles) per litre (the logarithm of the reciprocal of the H ion concentration). pH 7 is neutral. Above this value alkalinity increases progressively and below it acidity increases.

phosphagens A group of compounds, including phosphocreatine and ATP, that occur in tissues and which yield inorganic phosphate with the release of energy on cleavage. They are classified as high-energy phosphate compounds.

photosensitization The development of abnormally heightened reactivity of the skin to sunlight. In the text, reference is made to a skin reaction of horses following the consumption of certain plants. Reports exist of domestic animals reacting to St John's worts (*Hypericum* spp.) or bog asphodel (*Narthecium ossifragum*). White, grey or piebald horses or those with liver damage are most susceptible.

physitis Inflammation of the physis: the tissue between the metaphysis and epiphysis of the growth plate of long bones. Whether this presents a clinical problem during growth should be determined.

phytase Phytase (EC 3.1.3.8) is produced by *Aspergillus niger* (CBS 114.94). Phytase is produced by many microbial cells, but, as far as it is known, does not occur in any of the digestive secretions of the horse.

phytates Much of the phosphorus present in cereal grains and other seeds occurs as phytates, Ca, Mg, and other salts of phytic acid, a phosphoric-acid derivative, composed of a six-carbon ring structure with a phosphate group bonded indigestibly to each carbon atom. The horse can utilize only about 30% of the phosphate phosphorus, partly through the activity of bacterial phytase present in the intestines, but probably also as a result of some phytase activity which has been recognized in the wall of the intestine of several species. Phytates reduce the availability of dietary zinc. Wheat bran contains 1% of P present as phytin.

piloerection Erection of the hair.

pinworm See ***Oxyuris equi***.

pituitary gland (hypophysis) A gland lying at the base of the brain and connected with the hypothalamus by the pituitary stalk. Physiologically it consists of two parts, the anterior and posterior pituitary. Six hormones are secreted by the anterior portion: growth hormone, adrenocorticotropin, thyroid-stimulating hormone, prolactin, follicle-stimulating hormone and luteinizing hormone. The posterior portion secretes antidiuretic hormone (vasopressin) and oxytocin.

pK The symbol used to express the dissociation constant of weak acids (or bases) in the form of a negative logarithm. The larger the value the weaker, or less dissociated, is the acid. When equal concentrations of the salt of an acid and the acid are mixed, the pK = **pH** and the buffering capacity of the mixture is maximal. Thus, for the primary ionization of carbonic acid to bicarbonate, as in blood, the pK = 6.36, and at pH 6.36 half the molecules of carbonic acid are dissociated forming bicarbonate. At the normal pH of venous blood (7.4) the mixture is an even more effective buffer to acid produced during anaerobic muscular activity with the formation of undissociated carbonic acid.

placenta An organ that develops within the uterus in early pregnancy and that establishes communication between the dam and the developing foetus. It is composed of a maternal portion and a foetal portion attached to the foetus by the umbilical cord. Following **parturition**, it is passed as the afterbirth.

progesterone A hormone liberated by the corpus luteum, adrenal cortex and placenta. It prepares the uterus for the reception, development and maintenance of the fertilized ovum.

prognathia Abnormal protrusion of the upper or lower jaw bone, creating difficulties, e.g., in grazing.

prolactin A hormone secreted by the anterior pituitary gland that stimulates and sustains lactation.

prostaglandins Grouped in six main series of cyclic compounds derived from unsaturated fatty acids such as arachidonic acid – itself a derivative

of the dietary indispensable (essential) linoleic acid – and from fatty acids with one less and one more double bond. Prostaglandins were first recognized in seminal fluid and the prostate gland. They show a variety of biological actions that influence smooth muscle contraction (as in contraction of uterine muscle and in blood-pressure control) and are mediators in the regulation of the dilatation and permeability of arterioles, capillaries and venules in the inflammatory response. They are involved in immune mechanisms and are used for oestrus synchronization and for abortions in cases of twin foetuses. Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) which terminates the life of the corpus luteum, is the one most commonly used.

protease An enzyme that digests proteins by hydrolytically splitting off amino acids. Several kinds are secreted into the alimentary canal (see Chapter 1).

protein True proteins are chains in which the links are amino acids. All amino acids possess at least one N-containing amino-group. The crude protein content of the diet is defined as the N content × 6.25 as it is assumed that protein contains 16% N. However, this method includes dietary nucleic acids, nitrogenous glycosides, amines, nitrates, etc. and so overestimates the true protein content.

proteolysis The enzymatic digestion of protein, which, if carried out by the horse's own secretions, yields proteoses, peptones and amino acids, but, if carried out by intestinal bacteria, includes deamination with a loss of protein value.

prothrombin time The synthesis of prothrombin occurs in the liver and requires vitamin K. It is essential for the clotting of blood, and any defect in prothrombin formation, or in the activity of other substances involved in clotting, extends the interval between the initiation of the process and the formation of fibrin from fibrinogen. Fibrin spontaneously coagulates. **Dicoumarol**, which arises from the activity of a mould on coumarin in spoiled sweet clover or melilots, interferes with the metabolism of vitamin K causing an extension of prothrombin time and consequential extensive haemorrhaging (see Chapter 11).

proximal Nearest to the centre or origin (cf **distal**. The duodenum is proximal to the jejunum and the 'knee' is proximal to the fetlock.

purgative Cathartic, a medicine that stimulates peristaltic action and evacuation of the intestines.

pylorus The distal or duodenal aperture of the stomach. It is controlled by a sphincter muscle and through it stomach contents enter the small intestine.

pyrexia Abnormal elevation of body temperature.

Quarter Horse (American Quarter Horse) A breed devised mainly from dams of Spanish origin, long bred by American Indians, and from Galloway sires, introduced by early settlers of North America.

quidding The expulsion of partially chewed feed from the mouth. The habit may arise from injuries to the tongue, or cheek, resulting from molar teeth which are too sharp, irregular in height or in alignment, or even from permanent teeth pushing the temporaries out from the gums. Causes also include infections of the mouth or teeth and paralysis of the throat and consequent inability to swallow.

radius One of the two long bones of the 'forearm', between the point of the 'elbow' and the 'knee'. The other long bone is the ulna.

ration The amount of daily feed rather than its composition. It should include all the constituents of the diet apart from water.

rectum The distal portion of the large intestine extending from the small colon to the anus and holding the faeces.

red blood cells (erythrocytes) The most numerous cells in the blood ($6.8–12.9 \times 10^{12}$/L), there being only 5.4–14.3×10^{9}/L white blood cells (leucocytes). The cellular portion of blood makes up 32–53% of the total, the remainder being the plasma. About 35% of each red cell is the protein haemoglobin, which transports oxygen from the lungs to the various body tissues.

renal clearance The ratio of the concentration of a substance in the urine to that in the blood times the rate of urine formation. As the latter is normally unknown, the **creatinine** clearance ratio is measured (see Chapter 12). To calculate the glomerular filtration rate, when the rate of urine formation is known, creatinine or insulin may be used; these substances are readily filtered by the glomerulus but not secreted or absorbed by the renal tubules.

renal tubules These run from the glomerulus through the cortex of the kidney, as convoluted tubules, through the medulla as collecting tubules, and then open into the pelvis of the kidney at the apices of the renal pyramids. Their main function is the reabsorption of water and various solutes (glucose, chloride, Ca, P, etc.) required by the animal.

renin A proteolytic enzyme synthesized by the juxtaglomerular cells of the kidney that plays a role in blood-pressure control by catalyzing the conversion of angiotensinogen to angiotensin when renal arterial pressure falls.

reproductive cycle The time from the conception of one foal to the conception of the next.

requirement (nutrient) The requirement for any given nutrient is the amount of that nutrient that must be supplied in the diet to meet the net requirement of a normal healthy animal given an adequate diet in an environment compatible with good health. The net requirement is the quantity of that nutrient that should be absorbed to meet the needs of **maintenance**, including the replacement of obligatory losses, and of any work, growth, production or reproduction taking place.

resistin A cysteine-rich protein secreted by adipose tissue of mice and rats. It is called 'resistin' because of the observed insulin resistance in mice injected with resistin, so it probably participates in obesity and type II diabetes mellitus. Among the proteins synthesized and released from adipose tissue (adiponectin, angiotensin, **oestradiol**, IL-6, **leptin**, plasminogen activator inhibitor, **TNF-α**), resistin also participates in the inflammatory response, causing increased expression of several pro-inflammatory cytokines.

respiratory minute volume The volume of air which can be inhaled (inhaled minute volume) or exhaled (exhaled minute volume) from a horse's lungs in one minute. Minute volume is calculated by taking the tidal volume and multiplying it by the respiratory rate (the number of breaths per minute). The higher the minute volume the more CO_2 the horse is releasing. Hyperventilation is the term for having an increased minute volume. The symbol used to represent minute volume is $\dot{V}$ Tidal Volume × Respiratory Rate = Minute Volume.

respiratory quotient (RQ) and respiratory exchange ratio (R) When one molecule of glucose is oxidised the number of molecules of CO_2 liberated equals the number of O_2 molecules used. The RQ is defined as the molecular ratio of CO_2 output to O_2 usage, or CO_2/O_2. When TAG is oxidised the ratio is much lower, the precise value depending on which fatty acids are present in the TAG. The situation is comparable to that of amino acid oxidation when the RQ falls between that of carbohydrates and that of fats:

$$C_6H_{12}O_6 + 6\,O_2 \rightarrow 6\,CO_2 + 6\,H_2O$$
RQ: $6/6 = 1.00$ Glucose

$$C_{57}H_{104}O_6 + 80\,O_2 \rightarrow 57\,CO_2 + 52\,H_2O$$
RQ: $57/80 = 0.71$ Triolein

$$2\,C_3H_7O_2N + 6\,O_2 \rightarrow (NH_2)_2CO + 5\,CO_2 + 5\,H_2O$$
RQ: $5/6 = 0.83$ Alanine

The overall total intake of O_2 and the total output of CO_2 by a horse measured continuously for a period of time reflects the relative quantities of carbohydrates, fats and proteins metabolized by the horse for energy production. This is termed the Respiratory Exchange Ratio (RER), i.e. rate of CO_2 output/rate of O_2 uptake).

rheology The science of the deformation and flow of matter. In this text it relates to the flow of blood through the vascular system, especially as affected by an elevated packed cell volume during intense exercise.

rhinopneumonitis (equine) A mild viral disease of the upper respiratory tract of horses, which also commonly causes abortion.

rickets Defective calcification of the epiphyseal cartilage of growing horses owing to inadequate dietary vitamin D, Ca and P or an incorrect ratio of Ca to P in the diet.

ringbones Any bony exostosis affecting the interphalangeal joints of the horse's foot, or any bony enlargement in the same region.

Ringer's solution An isotonic solution, devised by Sydney Ringer, containing sodium chloride, potassium chloride and calcium chloride. However, it is only a little more physiological than physiological saline as its chloride concentration is higher and it can therefore cause metabolic acidosis (see Chapter 9).

roughage There are several types of roughage, which fall broadly into the following categories: (1) long and dry, e.g. hay and straw; (2) ground and pelleted hay, straw and oat feed; (3) ensiled long grass and comparable succulent forages; and (4) chopped succulent ensiled material. Within each category only feeds containing more than 20% crude fibre on an air-dry basis should be included. Roughage tends to reduce the intake of dry matter in horses fed *ad libitum* and decreases net energy intake in these animals, compared with those also receiving **concentrates**. The fibre is useful in maintaining the microbial populations of the large intestine in a steady state.

ructus Eructation or belching of gas from the GI tract.

ruminant Herbivorous species that possess an enlarged forestomach (rumen) and that chew the cud by regurgitation of ingesta from the forestomach to the mouth.

sclerosis An induration or hardening, especially resulting from persistent inflammation.

scours Diarrhoea.

septicaemia A serious condition in which bacteria circulate in the bloodstream and become widely distributed throughout practically every organ. In its severe form, the horse becomes distressed, respiration and heart rates are accelerated, and body temperature is elevated.

serotonin (5-HT) 5-Hydroxytryptamine (5-HT). A vasoconstrictor produced enzymatically from tryptophan. It is released by blood platelets, is found in many tissues and has many physiological properties including inhibition of gastric secretion and stimulation of smooth muscle (e.g., of gut wall). It also serves as a central neurotransmitter.

serum The clear liquid that separates from the clot and the corpuscles in the clotting of blood.

set-fast See **tying-up**.

SGOT See **AST**.

SGPT See **ALT**.

shank (of long bone) See **diaphysis**.

silage (ensilage) Succulent feed preserved either by adding acid or allowing natural fermentation to occur under anaerobic conditions in compacted material. The pH achieved is approximately 4–4.2. Too low a pH limits intake and too high a pH provokes protein breakdown. Materials ensiled include fresh grass, forage crops, the above-ground growth of young cereal crops and a variety of by-products from beet pulp to fish waste. The dry matter content is 30–50%.

spavin Bone spavin is a disease of the hock, or tarsus, in which changes occur in the small bones on the inner aspect of the joint resulting in the deposition of new bone. Bog spavin is a puffy swelling of the same joint.

spleen A gland-like ductless organ in the anterior part of the abdominal cavity on the left side. Its functions are at least threefold: first, it disintegrates red cells, setting free the haemoglobin, which the liver converts to bilirubin, conserving the iron; second, it acts as a storehouse of red cells, which it releases into the blood during times of higher oxygen demand; third, evidence (mainly from other species) indicates a role for the spleen in immunological responses. The spleen is an antibody-forming tissue in which macrophages constitute a predominant cell form.

splints Bony enlargements that occur on the cannon bones or in connection with the small metacarpals or metatarsals (splint bones) as the result of localized inflammation of the bone or periosteum (periostitis or osteitis).

stereotypy (stable vices) The persistent repetition of functionless and senseless acts and movements. These include cribbing (crib-biting), weaving and stall walking; but not wood chewing, coprophagy or geophagia (soil eating).

stifle The joint corresponding to the human knee at the top of the hind limb.

stocking up Swelling, or **oedema**, of the legs, owing to the accumulation of fluid beneath the skin frequently caused by a period of inactivity on rich feed immediately after an extended period of activity. Exercise, purging and a reduction in energy intake normally bring rapid relief. The condition is also occasionally seen in horses with damaged livers, especially where the diet is of low quality and deficient in protein.

strangles An acute contagious fever of horses, donkeys and mules caused by the bacterium *Streptococcus equi*, characterized by catarrhal inflammation of the mucous membranes of the nasal passages and pharynx, and frequently accompanied by abscess formation in the submaxillary or pharyngeal lymphatic glands noticeable under the jaw.

stridor A harsh, high-pitched respiratory sound.

stroke volume See **cardiac output**.

strongyles A group of strongyloid nematodes or roundworms widely distributed in the intestinal contents of mammals. In horses, strongyles are commonly called redworm (see Chapter 11).

sucrase (invertase) A digestive enzyme secreted by the small intestine, which hydrolyses sucrose (cane and beet sugar), forming glucose and fructose, both of which are readily absorbed.

sweetfeed An American term for a concentrate mix containing molasses.

sweet itch Recurrent seasonal pruritis. A hypersensitive reaction occurs to antigens in the saliva of a biting insect of the genus *Culicoides* leading to a crusting dermatitis.

synchronous diaphragmatic flutter Contraction of the diaphragm in synchrony with the heart beat. It is observed in fatigued horses following severe exercise in hot weather when excessive sweating may precipitate a large decrease in the plasma concentrations of ionized Ca, Cl and K.

tachycardia Excessive rapidity of heart action and pulse.

tachyphylaxis Rapid decrease in response to a drug, toxin, or other physiologically active agent, after administration, or previous tissue receptor experience of a few small doses.

tachypnoea Excessively rapid and shallow breathing.

tapeworm A parasitic intestinal cestode composed of numerous flattened segments and attached to the gut wall by a head. Not infrequently occurring in horses, but generally causes no major problem. *Anoplocephala perfoliata*, the only species to have been observed infecting horses in the UK, may occasionally block the ileocaecal sphincter and cause severe colic.

tarsus Bones of the hock. There are usually six bones of the hock, which join the tibia above to the two metatarsal bones below in the hind leg.

tetany A condition in which there are localized spasmodic contractions, or twitching, of muscles (see Chapter 11 for stress tetany).

therapeutic index (therapeutic ratio) A comparison of the amount of a therapeutic agent that causes a therapeutic effect with the amount that causes toxic effects. Quantitatively, it is the ratio given by the toxic dose divided by the therapeutic dose. A commonly used measure of therapeutic index is the toxic dose of a drug for 50% of the population (TD_{50}) divided by the minimum effective dose for 50% of the population (ED_{50}). This ratio has also been applied to individual nutrients.

Thoroughbred (TB) A **hotblooded** breed of about 1.625 m (16 hands), which originated in the UK and has been used to improve many other breeds.

thoroughpin Distension of the sheath of the deep flexor tendon where it passes over the arch of the **tarsus** (hock) resulting from a sprain. It is not usually serious.

thrombophlebitis Inflammation of a vein associated with thrombus formation.

thrush A degenerative condition of the horn in the central cleft of the **frog** of the horse's foot caused by a bacterial infection and often resulting from the horse standing in dirty wet boxes with insufficient clean dry bedding.

thumps See **synchronous diaphragmatic flutter**.

thyroid gland Situated in the neck in connection with the upper extremity of the trachea. The gland secretes two hormones: iodine-containing thyroxin and **calcitonin** (thyrocalcitonin) (see Chapter 3).

thyrotropin-releasing hormone (TRH) Also called thyrotropin-releasing factor (TRF), thyroliberin or protirelin, TRH is a tripeptide hormone that stimulates the release of thyroid-stimulating hormone and **prolactin** by the anterior pituitary.

tibia The major long bone between the **stifle** and the **hock**.

tidal volume The volume of air inspired or expired with each normal breath.

tillering The process of forming side shoots from the base of the stem of graminaceous plants (cereals and grasses). Grazing, or cutting, in the vegetative phase of growth encourages this process, so thickening the 'bottom' or base of young pasture swards and increasing their suitability for grazing and exercising horses.

α-tocopherol The principal tocopherol with vitamin E potency (see Chapter 4).

torsion As described in the text, the twisting, or rotation, of part of the intestine, causing an obstruction.

toxin Any poisonous substance of microbial, vegetable or animal origin.

trace elements See **minerals**.

tricarboxylic acid (TCA) cycle The series of metabolic reactions occurring in the mitochondria of cells by which acetylcoenzyme A (acetyl CoA) is oxidized to carbon dioxide and water. The energy released is stored as **ATP** (see Chapter 9). Acetyl-CoA is generated by the catabolism of fatty acids, carbohydrates and amino acids.

triticale This is a hybrid of wheat (***Triticum***) and rye (***Secale***) first bred during the late 19th century in Scotland and Sweden. It combines the grain yield potential of wheat with the disease and environmental tolerance, e.g., soil conditions, of rye. Wheat is used as the female parent and rye as the male parent (pollen donor). The resulting hybrid is sterile and has to be treated with the alkaloid chemical colchicine to make it fertile and thus able to reproduce itself. Commercially available triticale is usually a 2nd generation hybrid, i.e. a cross between two kinds of triticale (primary triticales).

trypsin An enzyme which acts on peptide linkages that involve the carboxyl groups of lysine and arginine and ruptures protein chains at these points. It is one of the proteolytic enzymes secreted by the pancreas, but in the inactive

form of trypsinogen. This is activated by the enzyme enterokinase liberated from the duodenal mucosa.

tumour necrosis factor α (TNFα) A cytokine involved in systemic inflammation and a member of a group of cytokines that stimulate the acute phase reaction. The primary role of TNFα is in the regulation of immune cells. TNFα is able to induce apoptotic cell death, to induce inflammation, and to inhibit tumourigenesis and viral replication.

turgor The normal consistency of soft tissue, as opposed to the flaccid condition of dehydration.

tying-up (set-fast) A condition in racehorses in which stiffness, blowing and sweating occur after a period of hard extended exercise, said to be caused by a depletion of muscle glycogen (see Chapters 9 and 11).

tympany Filling of the stomach or intestines with gas, usually caused by rapid microbial fermentation of ingesta, leading to a drum-like distension of the abdomen and colic.

ulna A bone behind the radius in the foreleg that forms the point of the elbow. The shaft of the ulna is vestigial and the tapered end is fused to the radius.

umbilical cord Nourishment of the foetus mainly passes through the umbilical cord from the placenta. After birth, the cord should not be interfered with, as early severance deprives the newborn foal of placental foetal blood. It can be broken by twisting close to the foal's abdomen after it has stopped pulsing.

uraemia The presence of urinary constituents in the blood and the toxic condition produced thereby. Normal blood urea level is greatly exceeded, indicating a failure of normal renal function.

urea The chief nitrogenous waste-product synthesized in the liver, discharged from the body in the urine and also secreted into the small intestine. It is highly soluble in water and the amount produced by the healthy horse receiving regular meals is proportional to the crude protein content of the diet.

urticaria See **hives**.

uterus A hollow muscular organ lying in the female abdominal cavity below the rectum. In the mare it has a large body and small horns. It carries the foetal foal and nourishes it during pregnancy through the placenta attached to its wall.

vagus (pneumogastric) nerve A major parasympathetic nerve (tenth cranial) of the **autonomic nervous system** possessing both efferent and afferent fibres distributed to the larynx, lungs, heart, oesophagus, stomach, liver, intestines and, in fact, most of the abdominal viscera. It therefore plays a considerable part in digestion and physical exercise.

vein Thinner-walled vessels than **arteries**, in which deoxygenated blood, under lower pressure, is carried back to the heart. Veins possess a system of valves that control the direction of blood flow. Muscular contraction and relaxation of the limbs provides the main force by which blood is lifted back up the legs against the force of gravity.

vertebrae A chain of bones running from the base of the skull to the tip of the tail which carries, in the spinal canal, the spinal cord – the posterior part of the central nervous system.

VFAs See **Volatile fatty acids**.

villus (pl. villi) Small vascular processes covering the mucous epithelium of the small intestine. They greatly enlarge the surface area for the absorption of nutrients into branches of the portal blood system and into the lymphatic system.

viscera In this text the term refers to the abdominal viscera – the organs of the abdominal cavity.

vitamer Any of a number of compounds that possess a given vitamin activity.

vitamins A group of unrelated organic substances which occur in many foods in small amounts and which are necessary for normal metabolism of the body. They have been arbitrarily divided into a group of four major fat-soluble vitamins and at least ten water-soluble ones (see Chapter 4).

volatile fatty acids (VFAs) Short-chain steam-volatile acids, principally acetic, propionic, butyric and smaller quantities of higher acids, which are the microbial waste-products of fermentation of dietary polysaccharides and protein within the alimentary canal. They are absorbed into the bloodstream and constitute a major energy source for the horse.

volvulus Intestinal obstruction due to a knotting and twisting of the intestines.

warmblooded See **hotblooded**.

wasting disease A state of chronic emaciation.

wind-sucking The habitual swallowing of air (aerophagia) is similar to crib-biting. The strap muscles are flexed and the horse emits an audible grunt,

but does not seize an object with the incisors. The term 'wind-sucking' is also unfortunately used to describe mares that aspirate air, and frequently faecal material, into the vagina. This is rectified by Caslick's operation.

withers The ridge on the back of the horse over the dorsal processes of the thoracic vertebrae and the shoulder blades and directly in front of the saddle.

wobbler The name given to a horse showing a slight swaying action of the hindquarters, or stumbling, occurring mainly between one and three years of age. The signs can become progressively worse over six to nine months when the horse is unable to trot without rolling from side to side and falling. The condition apparently results from damage to the spinal cord in the neck through injury and/or nutritionally induced abnormalities of the vertebrae caused by imbalances or inadequacies of Ca and P.

wood chewing A habit developed by many horses probably as a result of boredom. The horse normally does not swallow the wood and the habit is unlikely to have any dietary implications.

zone of thermal neutrality The range of environmental temperatures over which heat production by the animal is minimized. Below this range the animal must increase heat production by shivering and other means in order to maintain a normal body temperature. Above the range, normal cooling mechanisms prove inadequate, body temperature rises and with it metabolic rate.

zoonosis A disease of animals that may be transmitted to man under natural conditions, e.g. anthrax, salmonellosis, arboviral encephalitis, cryptosporidiosis, *Clostridium difficile*, giardiasis, leptospirosis, dermatophytosis (ringworm), hendravirus, brucellosis, rabies, *Burkholderia mallei* and methicillin-resistant *Staphylococcus aureus.*

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Conclusion

The principles and science of equine nutrition and feeding practice can still be accommodated within one text. As our understanding of the subject increases, husbandry must be continually modified to be appropriate to each horse's evolving environment. The continual evolution of this means that any final resolution of all aspects of the subject is unlikely. A greater understanding of the relationship between diet and endocrine system should assist guidance towards better control of reproductive physiology and obesity.

For many stables, access to adequate pasture will continue to decline. Climate change and perhaps an increasing scarcity, and price, of cereals will require adaptations to husbandry practices. Improved analytical procedures that more reliably describe forages and byproducts should allow a wider and better use of materials not previously incorporated in the diet. This should lead to a more economic use of land and should be an asset to better husbandry, leading indirectly to a reduction in the risk of diet-related diseases. Yet skill and experience in feeding practice must reign in the management of individual horses, and I hope that the present text will provide a useful summary of recently published scientific evidence to be accommodated by readers with these skills.

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