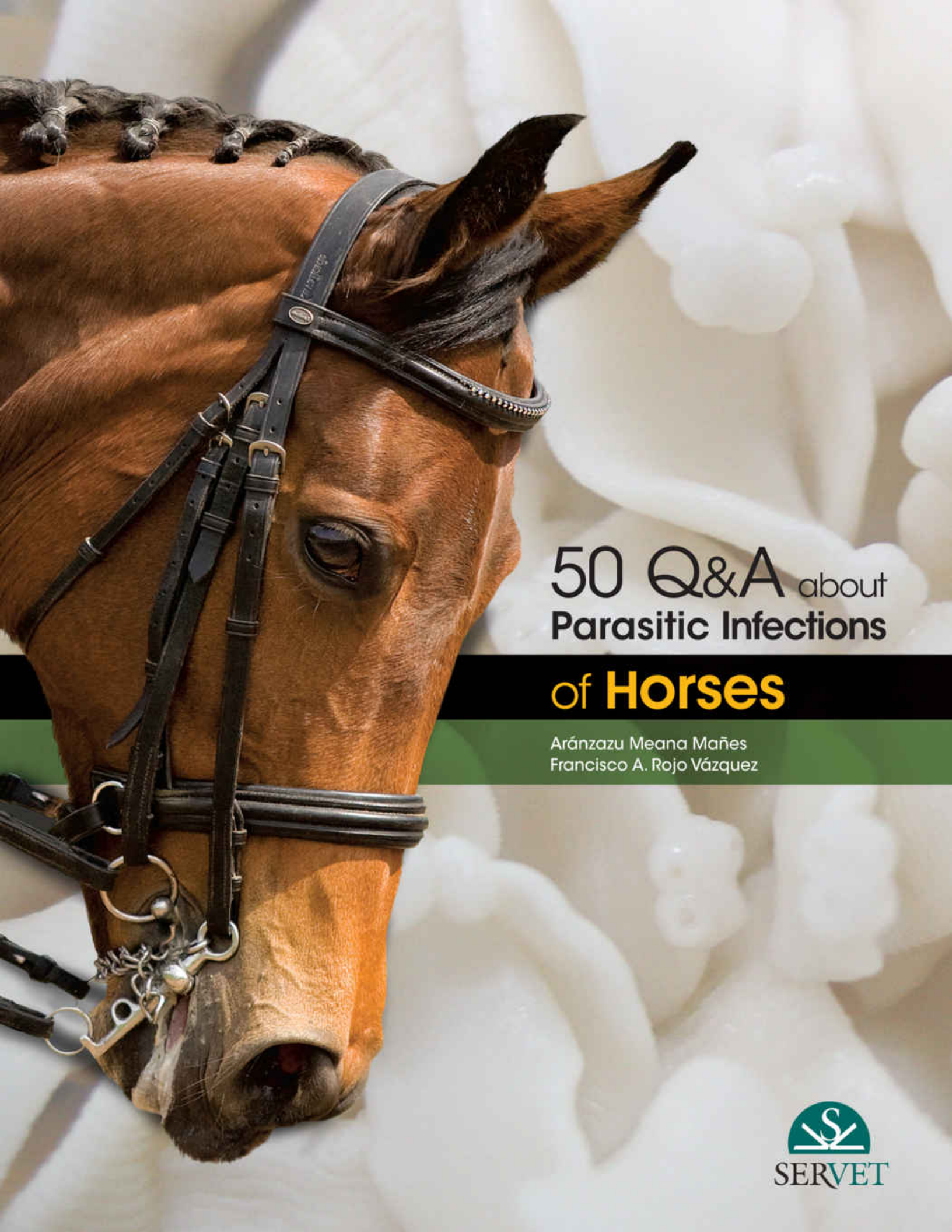


50 Q&A about Parasitic Infections of Horses

Aránzazu Meana Mañes
Francisco A. Rojo Vázquez

SERVET

50 Q&A about **Parasitic Infections** of **Horses**

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Plaza Antonio Beltrán Martínez, n° 1, planta 8 - letra I
(Centro Empresarial El Trovador)
50002 Zaragoza - Spain

First print: August 2018

Illustrator:
Jacob Gragera Artal

Translator:
Owen Howard

ISBN: 978-84-17225-82-7
eISBN: 978-84-17225-83-4
D.L.: Z 1135-2018

Design, prepress and printing:
Servet editorial - Grupo Asís Biomedia S.L.
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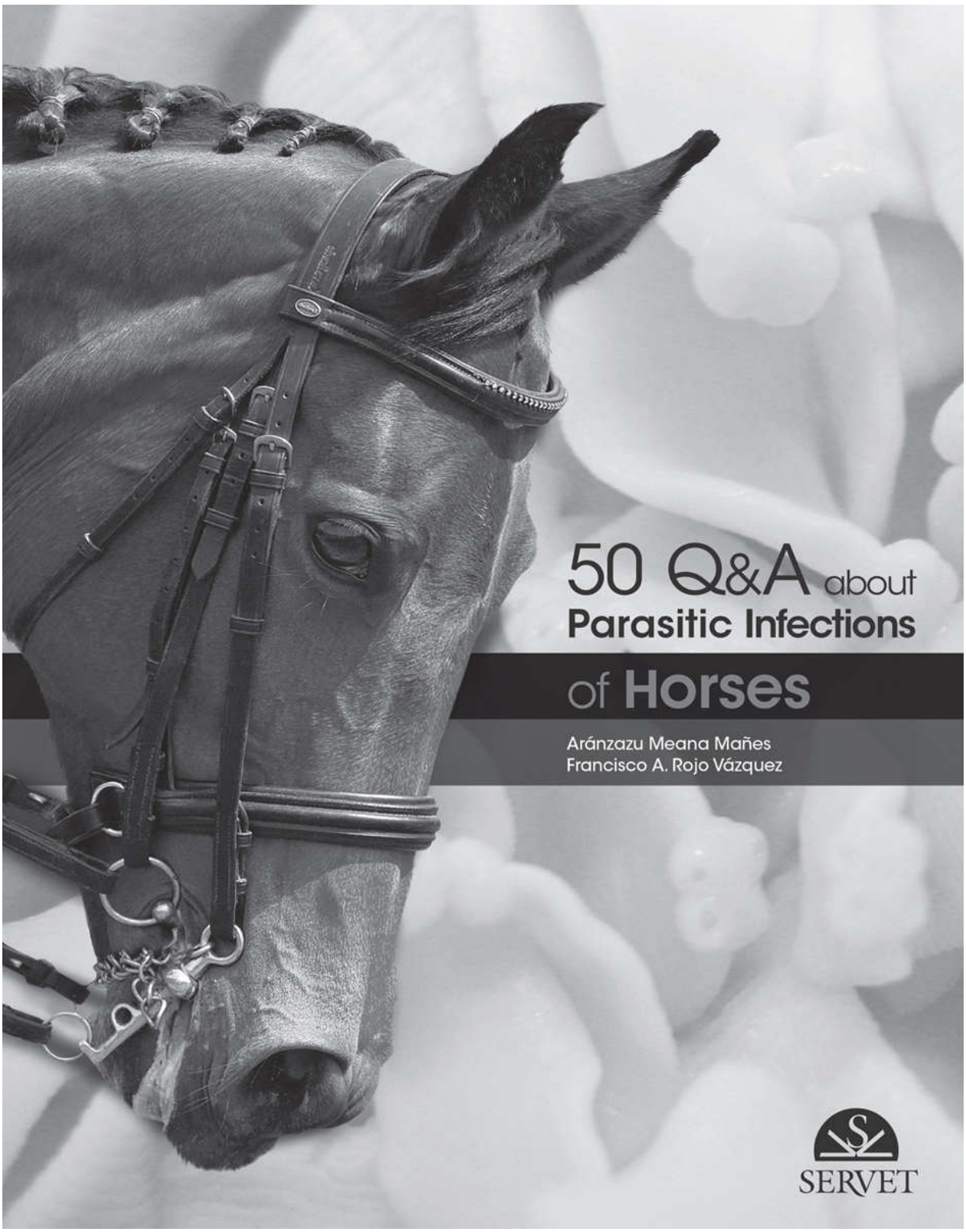
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This book is dedicated to our families, for their understanding of our dedication to our academic work, which at times has intruded upon our time with our loved ones, in particular our children.

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"In many situations, parasitic infections should be the first possibility to be eliminated before looking at the viral, bacterial and nutritional disorders as causes of disease"

Hugh McL. Gordon

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Acknowledgements

This work is the result of the authors' many years of study and experience. It could not have been written without the generosity of all those who shared their knowledge and concerns, stimulating our learning and facilitating the discovery of new knowledge and alternative teaching approaches. The book's original structure was the result of our desire to use new ways of teaching and sharing the findings of fruitful discussions, and should be of benefit to students who wish to learn about parasitic diseases of equids. We wish to thank all our fellow veterinary surgeons, both clinical and academic, who specialise in parasitic and equine pathology, for sharing their experiences. The strong practical emphasis of this book is a direct result of their participation.

In particular, we would like to thank the following (in alphabetical order): Sonia Almería, Alejandro Bohórquez, Cristina Botías, María Castaño, Carmen Castellá, Juan Carlos Castillejo, Pablo Crescente, Antonio Encinas, Rosario Famularo, Manolo Felipe, Nélida Fernández Pato, Jordi Figueres, Elkin Flores, Juana Mª Flores, Paloma Forés, Pilar García Palencia, Mónica Luzón, Guadalupe Miró, Ana Morcate, Javier Mota, Ángeles Sonia Olmeda, Belén IX del Olmo, Adolfo Paz, Bernardino Prieto, Francisco de Rivera, Manuel Rodríguez, Juan Rodríguez, Héctor San Martín, Jesús Sánchez, Ana Vicente, and ICTS Microscopia at the UCM.

While many of the images used are our own, others have been kindly provided by collaborators who are referenced in the text. We would like to express our gratitude to all those who provided us with data and images. We urge each and every one of the readers to contact the authors at the addresses provided below with comments, opinions, or experiences relating to parasites of equids and the pathologies they cause.

We hope you enjoy reading this book as much as we enjoyed writing it.

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Foreword

You have in your hands a clearly original work. Some time ago, when we discussed tackling this work, we thought about the best approach to share our experiences in the field of equine parasitology, in terms of both the information discussed and the manner in which it is presented to the reader.

From the outset, it was clear to us that this book would be primarily aimed at veterinary students, given our roles as educators of future graduates in veterinary medicine. We hope that the content and the manner in which it is presented will prove stimulating for those still studying, and that the many questions raised will provide the reader with an impetus to broaden their knowledge throughout their professional career, regardless of whether they work with equids or other animal species.

We also hope that practicing veterinary surgeons will find this book useful, and will find among its pages the answers to many of the questions that arise in daily practice.

Finally, this work, which is replete with fascinating images, should also be of help to horse owners, allowing them to identify the best approaches to manage specific clinical pictures, and to help orient the veterinary surgeon if and when veterinary assistance is requested.

This book on parasitic diseases of equids is in no way intended as a substitute for classical equine parasitology texts, but rather as a complement to the existing body of work in the field. It is presented in a somewhat atypical format, and focuses on the most important pathological processes of equids, avoiding unnecessary repetition as much as possible.

The question-and-answer format provides a unique means of discussing parasitic conditions of equids, although this approach is clearly far from new.

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One of the many innovations of the Ancient Greeks is a technique dating back to the fourth century BC that is usually attributed to the philosopher Socrates: maieutics. Maieutics is derived from the Greek word μαιευτικη, which is translated literally as obstetrics or "bringing to light", as in childbirth. It seems to be no coincidence that Socrates' mother, Phaenarete, was a midwife.

Maieutics is the method of research and teaching proposed by Socrates. In the field of philosophy, maieutics is an educational method based on dialectics (the dialogue between student and teacher). According to this approach, the knowledge and truth naturally inherent to each can be brought to light by posing the appropriate questions.

The use of this technique was not limited to the Greek academies. Throughout history there have been numerous examples of the application of maieutics to the field of education.

In the field of veterinary medicine, several treatises have been published on breeding and diseases of domestic animals using a question-and-answer structure. Using this method of teaching, the teacher does not seek to instil knowledge; the student is not viewed as an empty drawer that must be filled. Rather, the student attains this knowledge through dialogue with the teacher via a more individualised approach. This teaching technique appears to have returned to the university classrooms of the 21st century with an unprecedented force.

One of the treatises in which this approach is evident is Francisco García Cabero's *Institucion de Albeitería* (Institutions of Veterinary Medicine), published in 1740.

In Spain, a country with a strong equine tradition, it is difficult to find data in scientific or popular publications on parasites of equids or the pathologies they cause, and few researchers have focused their studies on parasites of equids. During the last century, official institutions showed no particular interest in this field, and adequate funding of research into the main problems caused by parasites was lacking, despite the enormous economic repercussions of bans on the export to some countries (e.g. USA) of horses with certain parasitic diseases.

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Consequently, there is a dearth of up-to-date information in the field of equine parasitology. However, as early as 1290, J.A. de Salamiella described skin lesions that may have been the result of dourine. He also reported cases of mange and described the use of different staining techniques. In 1430, Manuel Díaz recommended anthelminthic treatments for horses, and in 1560 Fray Miguel Agustí described haematuria compatible with piroplasmosis in horses. Baltasar Francisco Ramírez (1629) described infections by *Parascaris equorum* and *Oxyuris equi* and the anal itching caused by the latter, as well as verminous colic. In the 18th century, Domingo Royo (1734) and García Cabero (1764) wrote about "leeches" of equids, verminous colic, as well as infections caused by and the surgical removal of *Setaria equina.* These authors also described mange of horses.

In the second half of the 20th century, research into equine parasitology began in earnest, driven initially by the studies of Dr José María Tarazona Vilas and subsequently by those of various research groups, which included the authors of this book.

Equids can be parasitised by many species of protozoa, helminths, and arthropods, resulting in processes that can only be controlled and corrected based on a solid knowledge of the underlying epidemiological and clinical aspects covered throughout the four chapters of this book.

The authors
Madrid-León, January 2018

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Table of contents

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1 Q & A

Endoparasites of the **digestive system**

Introduction

1 What are gasterophilids?

2 How are gasterophilids transmitted?

3 What are the effects of *Gasterophilus* ?

4 How can we tell if a horse is parasitised by gasterophilids?

5 How is *Strongyloides* transmitted?

6 How do foals become infected with *Parascaris* if the adults are resistant and do not shed eggs?

7 What are the consequences of *Parascaris* infection?

8 Why is it so difficult to eliminate *Parascaris* from a farm?

9 How are *Parascaris* transmitted?

10 Are any *Parascaris* species resistant to anthelmintics?

11 Can cestodes cause the death of their host?

12 How is the risk of cestodiasis avoided?

13 How important a disease is strongyloidiasis in equids?

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14 What are the most pathogenic strongyles?

15 Can *Strongylus vulgaris* larvae be observed in the mesenteric arteries?

16 Which nematode migrates via the right parietal peritoneum?

17 How and when are equids infected by strongyle larvae?

18 How does thromboembolic colic occur?

19 What is exercise-related lameness of parasitic origin?

20 What pathology do cyathostomins cause?

21 Are any equine strongyles resistant to anthelmintics?

22 How is oxyuriasis diagnosed?

23 Can more than one pinworm species affect equids?

24 Is *Fasciola* commonly found in equids?

25 Which parasites are implicated in diarrhoea of newborn animals?

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2

Q & A

Endoparasites of the **respiratory system**

Introduction

26 How is dictyocaulosis diagnosed in a horse?

27 Why do donkeys transmit dictyocaulosis to horses?

28 Why are so many eosinophils produced in cases of parasitic bronchitis?

29 What fly larvae can cause catarrhal processes in horses?

30 What parasite can cause laryngeal stridor?

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3

Q & A
Systemic
endoparasites

Introduction

31 What is the best method to diagnose piroplasmosis?

32 Can piroplasms cause death?

33 Why is piroplasmosis not fatal in horses in endemic areas?

34 Why do animals infected by *Theileria equi* continue to carry the disease?

35 Are hydatid cysts of horses the same as those found in other species?

36 Can horses transmit trichinellosis?

37 What is dourine?

38 Can horses transmit toxoplasmosis?

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4 Q & A
Parasitic infections **of the skin**

Introduction

39 What types of mites parasitise equids?

40 Are mites easily visualised?

41 Can rabbits transmit mange to horses?

42 How is leg mange diagnosed?

43 Are males required in order for lice to multiply?

44 Why are ticks so abundant?

45 What diseases do ticks transmit?

46 Are ticks found during winter?

47 Can severe allergic reactions be caused by mosquitoes?

48 What diseases do flies and mosquitoes transmit?

49 Why does the forest fly not fly?

50 Which parasite impairs covering in stallions?

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1

Q & A
Endoparasites of the **digestive system**

Chapter 1

Q & A Endoparasites of the digestive system

Introduction

This chapter deals with problems caused by parasites that reside in the digestive tract. While few parasitic species reside in the acidic environment of the stomach, those that do are particularly striking, except for the inconspicuous nematodes of the genus *Habronema* . It is common to find large larvae of *Gasterophilus* flies. Less common are nematodes such as *Draschia* and *Trichostrongylus* , the latter of which is also found in ruminants. Many cysts, eggs, or larvae pass through the stomach after ingestion on their way to the intestine, where they continue their endogenous biological cycle.

Given that the function and structure of the small intestine varies throughout its length, it is unlikely that the parasites that reside in this structure will all be found in the same location. For example, the small *Strongyloides* shows a predilection for the duodenum, while *Anoplocephala* tends to accumulate in the final third of the intestine, next to the ileocecal

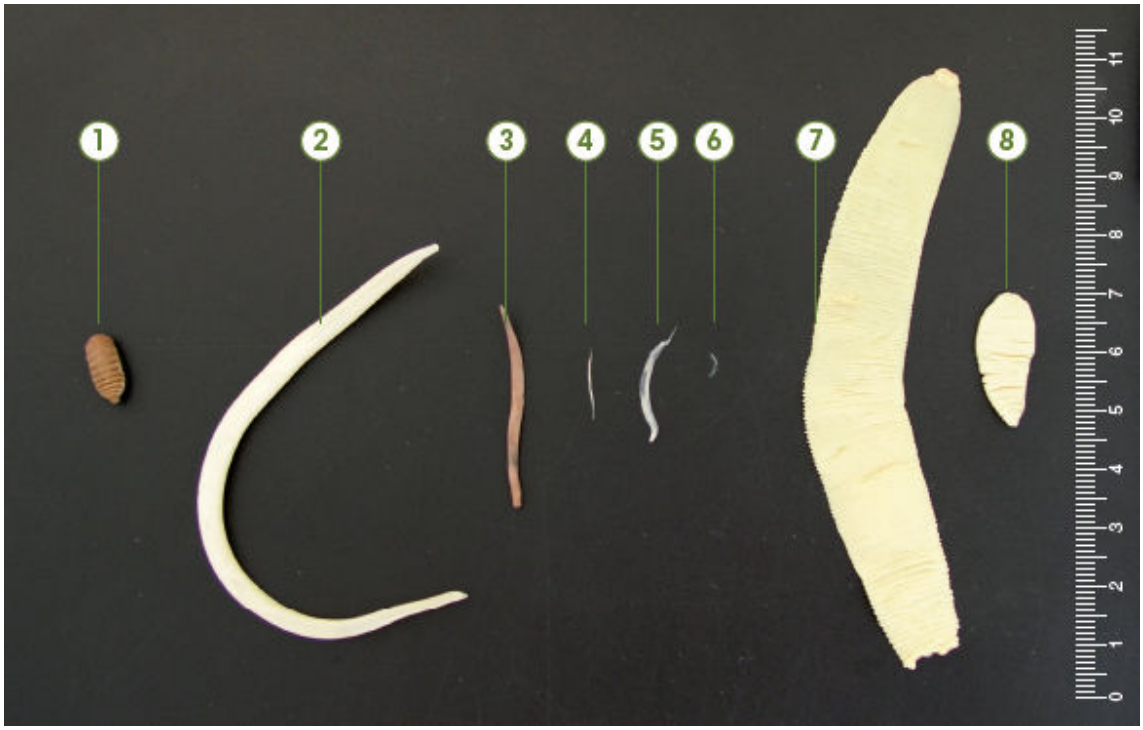
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valve. Other parasites typically found in this region include protozoa and the most common and cosmopolitan nematode, *Parascaris* . *Trichinella* , which can affect equids, is rarely found in horses.

The large intestine of equids is the digestive organ in which parasites are most abundant, both in terms of number and species diversity. This is the site of numerous fermentation reactions that mediate the decomposition of cellulose, and where large numbers of nematodes, some cestodes, and even fly larvae can coexist with thousands of microorganisms. Among the many nematode species described in this location are migratory species that must invade the organism before reaching sexual maturity in order to complete their endogenous cycle. The best known are the three *Strongylus* species, in particular *Strongylus vulgaris* . These species have significant pathological repercussions associated with the metabolic requirements of their larvae and their invasion of the digestive arteries.

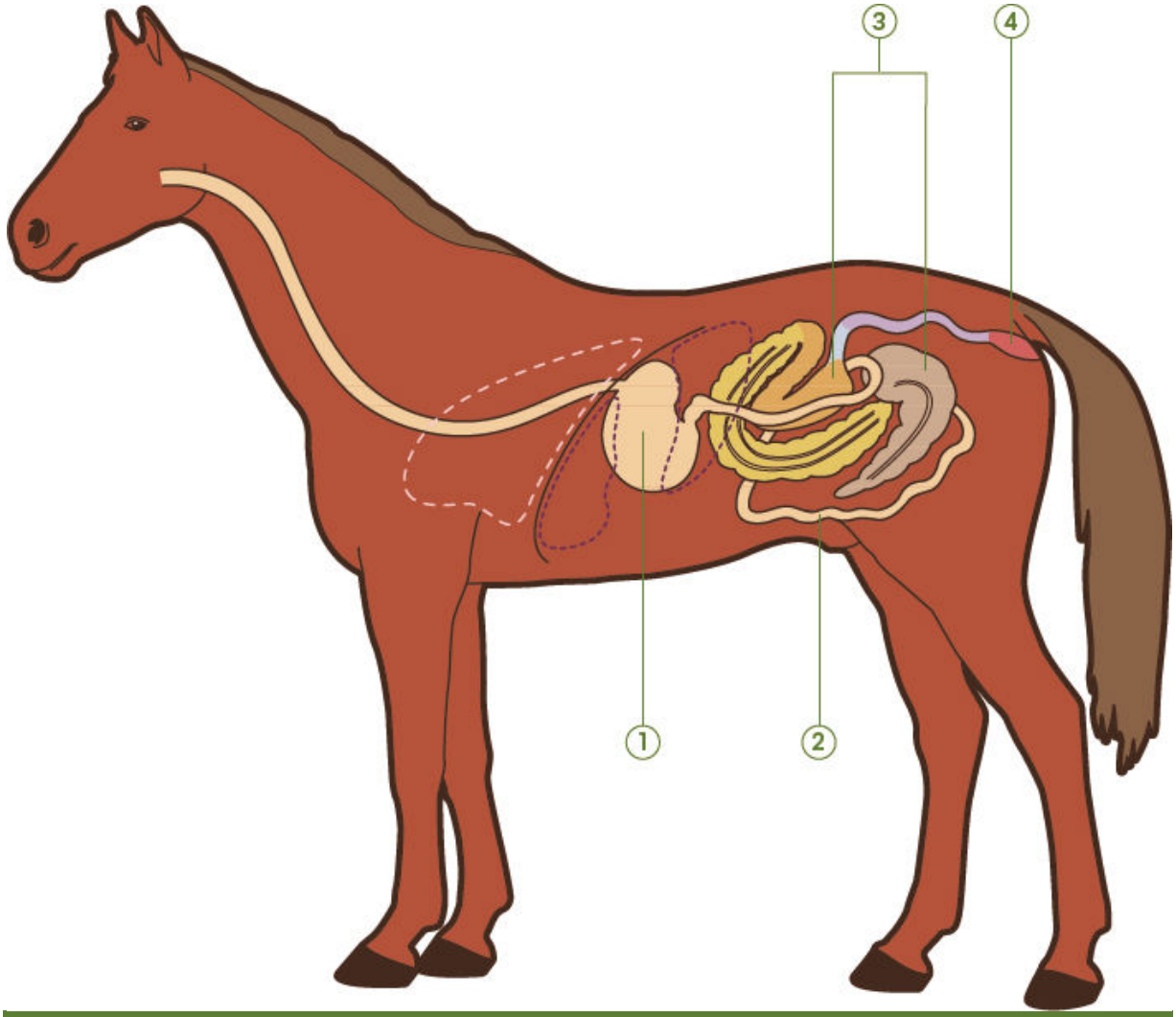
Comparison of the sizes of various parasites of the digestive system.
1.*Gasterophilus* larva.
2.Adult *Parascaris* .
3.Adult *Strongylus vulgaris* .
4.Adult cyathostomin.
5.Adult *Oxyuris* .
6.Adult *Strongyloides* .
7Adult *Anoplocephala magna.*
8Adult *Anoplocephala perfoliata* .

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Most important parasites of the equine digestive system (simplified diagram)

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1.Stomach
- –*Gasterophilus*
- –*Habronema /Draschia*
- –*Trichostrongylus*

2.Small intestine
- –*Gasterophilus*
- –*Parascaris*
- –*Strongyloides*
- –*Anoplocephala magna*

3.Caecum and colon
- –*Anoplocephala perfoliata*
- –Subf. Cyathostominae
- –Subf. Strongylinae
- –*Oxyuris*

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4.Rectum
–*Gasterophilus*

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1 What are gasterophilids?

During a routine necropsy, numerous red clumped formations are observed in the stomach. Other yellowish formations are detected in the duodenum and in the rectum. In the latter site the formations are smaller and are anchored deep in the rectal mucosa.
The morphological differences between the different locations are striking.

1.What could be the cause?
2.Is the presence of these parasites compatible with any clinical signs?
3.At what time of the year are they most often found?
4.What product is recommended to eliminate them?



1 What could be the cause?

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These are larvae of flies of the genus *Gasterophilus* . The larvae are found in the digestive system of equids and rarely cause serious disorders. The most significant aspects of these infections are the accidents that can occur when animals try to avoid the attacks of adult flies, and the lesions caused by the developing larvae in the digestive tract. These are specific forms of myiasis that are found worldwide, but are more frequent in warm climates where the flies, whose populations peak during the hotter months, are more prevalent.

2 Is the presence of these parasites compatible with any clinical signs?

These parasites cause a chronic process associated with signs of malnutrition and, occasionally, peritonitis, and particularly affect young animals at pasture. They are quite frequent, and have been detected in 80 % of animals slaughtered in central Spain. The most common species is *G. intestinalis* , which is found in the non-secretory mucosa of the stomach, often clustered in large numbers. Although they can cause crater-like lesions around the site of attachment, they do not usually produce obvious clinical signs.

The other species differ in terms of localisation, and are characterised by lower parasitic burdens and certain clinical signs, such as duodenal ulcers (*G. nasalis*) or discomfort during defecation (*G. haemorrhoidalis*). *G. inermis* , *G. pecorum* , and *G. nigricornis* are less common. *G. ternicinctus* is the only species not described in the Iberian Peninsula to date.

The developmental cycle of *Gasterophilus* consists of the egg phase, three larval stages, and the pupal and adult stages. The only parasitic phases are the larval stages, which occur in the digestive system. Eggs and first-stage larvae of some of these species are found in the skin.

The colour of the L3 stage can be intense red (*G. intestinalis, G. haemorrhoidalis* , *G. pecorum*), brown (*G. inermis* and *G. intestinalis*), or yellowish-grey (*G. nasalis*). They measure 15–22 mm in length, and have a pointed anterior end and a blunt posterior end. The body consists of 11 bulging, spine-covered rings. The first segment has 2 chitinous buccal hooks, which allow the larva to adhere firmly to the digestive mucosa and remain for months in the same location. The shape and distribution of the

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spines in the L3 stage is the most important feature by which species can be differentiated.



3 At what time of year are they most often found?

Animals are parasitised during the months in which conditions favour the development of flies (mainly summer). The larvae remain for about 10 months in the host until they complete their development and detach, subsequently exiting the host in the faeces and continuing their life cycle. After a short period in organs of the anterior digestive system (mouth, pharynx), the larvae moult and attach in areas of the posterior digestive system (stomach, small intestine, rectum) until maturation. During the months that they remain attached they grow to reach almost 2 cm.

4 What product is recommended to eliminate them?

Control of this parasite is based on the use of products that are effective against fly larvae: macrocyclic lactones, either injected or administered orally,

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are generally active against all larval stages of *Gasterophilus* . They can be administered in very young foals and pregnant mares. Organophosphates (e.g. trichlorfon, dichlorvos) should be used with caution. Trichlorfon (metrifonate) is contraindicated in foals of less than 4 months of age and in mares at the end of pregnancy, while dichlorvos should not be used in animals of less than 100 kg or in those with chronic respiratory disorders. At therapeutic doses they can produce some temporary side effects (e.g. salivation, incoordination, tremors). Benzimidazoles and pyrantel, which are strict anthelmintics, are not active against gasterophilid larvae.

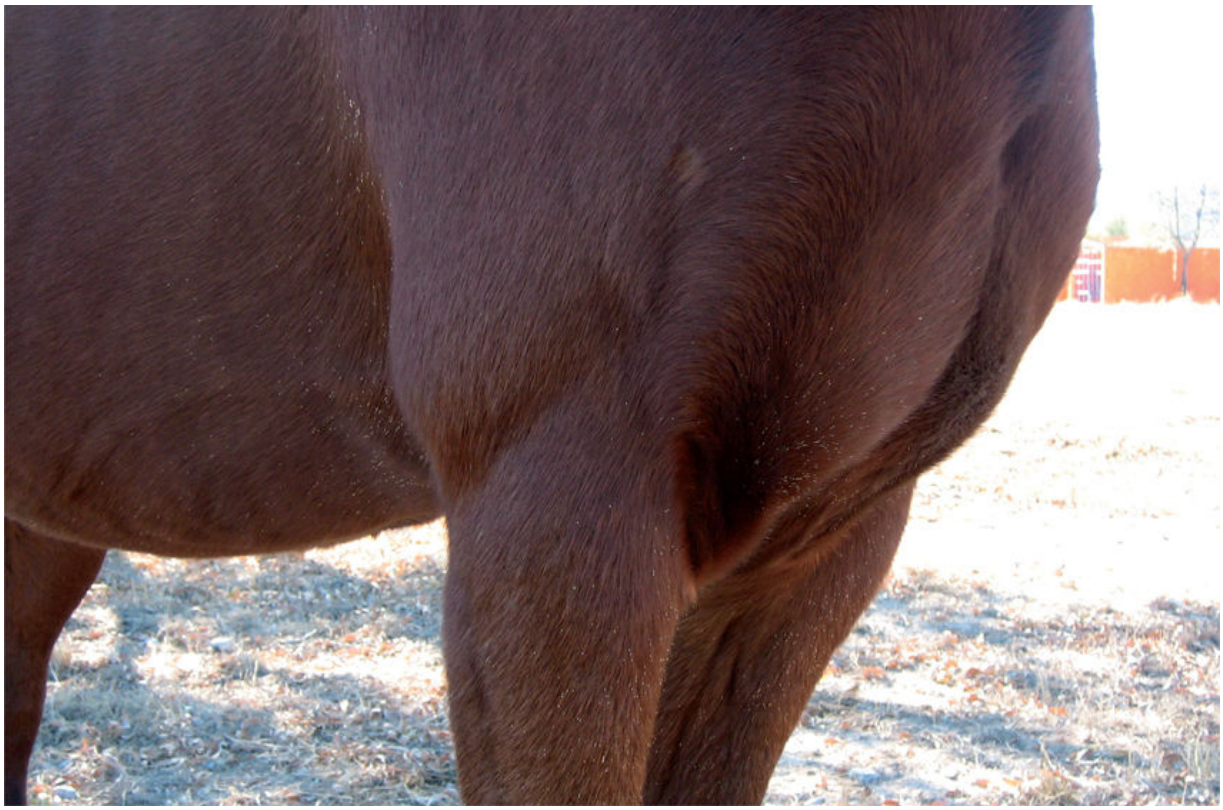
Regardless of the need for curative treatments, gasterophilids are very common parasites that should be included in annual strategic treatment programmes, based on the seasonal activity of the flies in each region, in order to act at the optimal moment to target established larvae (usually the beginning of the cold season). Therefore, treatments should be administered at the end of autumn (November–December) to prevent the development of third-stage larvae (L3), thus preventing the emergence of new generations of gasterophilids the following summer. If the infestation is frequent and strong, it is recommended to repeat the treatment after 3 months. Maintaining good hygiene of the skin and hair (showers, washes, and energetic brushing of areas invaded by eggs with hot water and an insecticide) is an effective means of stimulating the emergence of the larvae, which can be subsequently destroyed.

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The adult flies are brown, large (13–17 mm long), hairy, and somewhat similar to bees. Their oral apparatus is vestigial and nonfunctional. Consequently, they are unable to feed themselves and have a very short life span. They have a long and prominent ovipositor on the underside of the body that can be mistaken for a sting.

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The eggs are laid individually and adhere to the hair of the host by means of a sticky, water-insoluble glue. They are elongated and measure 1–2 mm and are easily visible attached to the hair of equids. The eggs of G. *pecorum* and G. *haemorrhoidalis* are dark in colour (brown or black), while those of the other species are pale yellow.

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2 How are gasterophilids transmitted?

During a visit to a riding school in autumn, large numbers of eggs are observed adhered to the mane and forelimbs of many animals. The owner asks if they are nits, like those found in the hair of children, and how they can be treated.

1. Is it necessary to send samples to a laboratory to confirm the diagnosis?
2. One of the species is much more frequent than the others, and always appears in large numbers. Which species is this, and why?
3. Would it be advisable for the owner to place insecticide strips inside the boxes to prevent egg laying?

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1 Is it necessary to send samples to a laboratory to confirm the diagnosis?

During the summer and the beginning of autumn it is common to find large amounts of eggs laid in the mane and forelimbs of animals kept outdoors, since flies of the genus *Gasterophilus* do not usually enter closed spaces. While they are especially visible in animals with darker coats, owing to the whitish-yellow colour of the eggs, this does not indicate a predilection for this coat colour in particular.

The eggs, unlike lice nits, are attached to the hair at a considerable distance from the skin surface and are not ovoid; the operculum or orifice through which the larvae emerge is positioned obliquely. Therefore, *Gasterophilus* eggs (which are striated) can easily be differentiated from lice nits (which are

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mottled and much whiter). Moreover, the presence of lice is more frequent in winter than in summer.

In temperate zones, flies appear mainly in the summer months. On sunny days they hover around free-roaming horses emitting an intense buzzing sound similar to that of bees. Gravid females lay their eggs in flight, without landing on the animals. Using a forward and upward pointing ovipositor, they fix one egg after another to the hair of the host. The flies lay eggs almost continuously on hot, sunny days, from mid-morning to late afternoon. They fly rapidly and produce a loud buzzing that can frighten horses. As with other Oestridae, males die after copulation. The females are also very short-lived; they are unable to feed with their atrophied mouth apparatus, and live only up to 1 week.

2 One of the species is much more frequent than the others, and always appears in large numbers. Which species is this, and why?

After copulation, the females hover around the animals and lay their eggs on the lower part of the forelimbs, the abdomen, the upper third of the hindlimbs, and in some cases on the shoulders. The number of eggs deposited by a female is quite high, ranging from 300 to 1000. Each species deposits their eggs in a different body region of the host. For example, the eggs of *G. intestinalis* are found attached to the hairs of the forelimbs, mainly on the inner aspects, and in severe infestations on the abdomen and hindlimbs. *G. haemorrhoidalis* deposits its eggs at the base of the hair follicle, around the mouth and maxillary region; *G. nasalis* on the hairs of the mandibular canal and the upper area of the neck or throat; *G. inermis* at the base of the hairs of the masseteric and mandibular regions; *G. pecorum* in feed, on objects on the ground, and on the hooves; and *G. nigricornis* around the mouth and nostrils.

In suitable conditions, a first-stage larva (L1) develops inside the egg and must reach the interior of the host after cutaneous migration to the mouth. These larvae are extremely mobile. In the most common species, the eggs do not hatch and the larvae remain inside in the absence of a stimulus in the form of an increase in temperature and humidity. When the animal licks or nibbles, the larva, stimulated by the tongue, emerges from the egg and adheres to the lingual mucosa, subsequently reaching the interdental spaces, the base of the

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tongue, or the soft palate. In other cases, eggs fall to the ground or into water, and infestation occurs following ingestion of these contaminated substances.

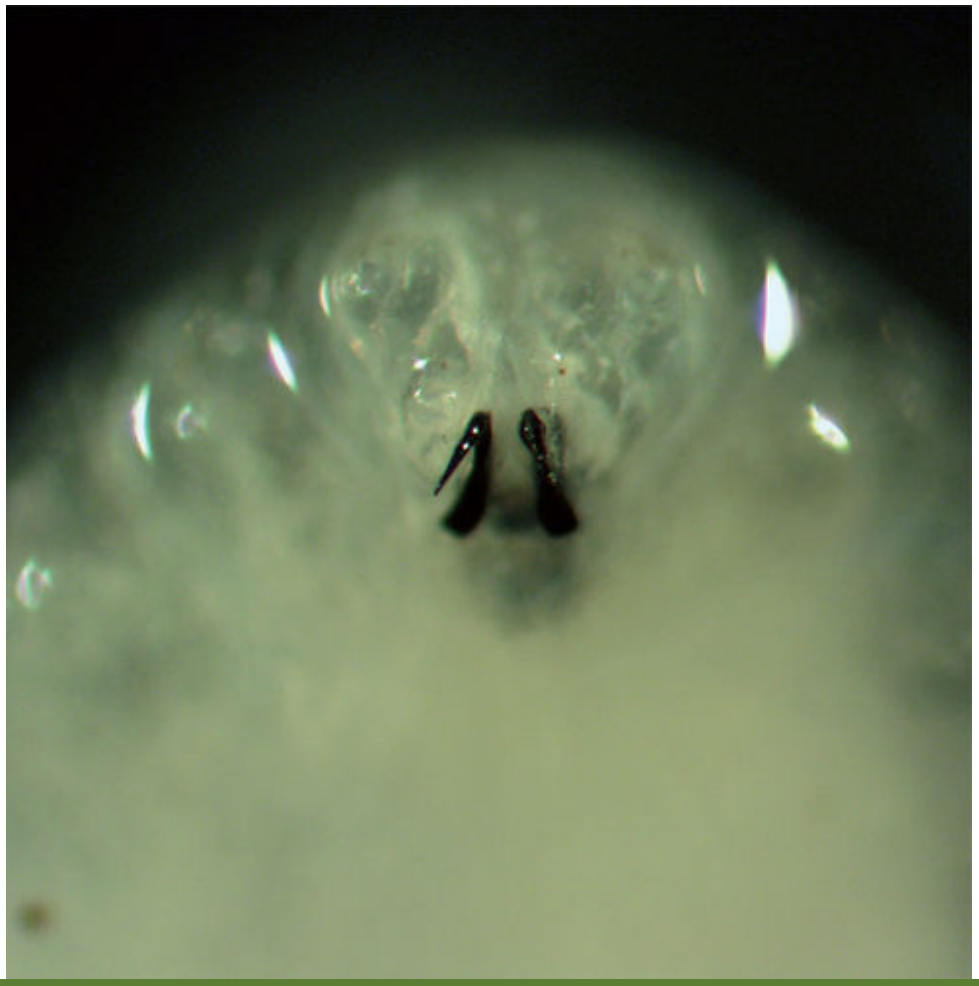
After a few days, the L1 stage leaves the buccal, lingual, or gingival mucosa and moults to the second-stage larva (L2), which reaches the stomach approximately 30 days after entering the body. Depending on the species, they remain in a given location for about 10 months. They grow to about 2 cm and, once mature, detach and are expelled in the faeces.

In dry areas they bury to a shallow depth and transform into pupae. In favourable conditions, the pupal phase lasts about 20 days, after which adult individuals are formed. However, when conditions are less than favourable, this period can be prolonged to more than 2 months. The pupae are very sensitive to frost and excessive humidity (e.g. flooding of grasslands). They cannot develop in stables or boxes in the absence of soil; the life cycle can only occur outdoors, on pasture.



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The L1 stage develops inside the egg, from which it emerges either spontaneously or in response to a stimulus. It is light in colour, transparent, fusiform, about 1 mm in length, and has 13 spine-covered segments.



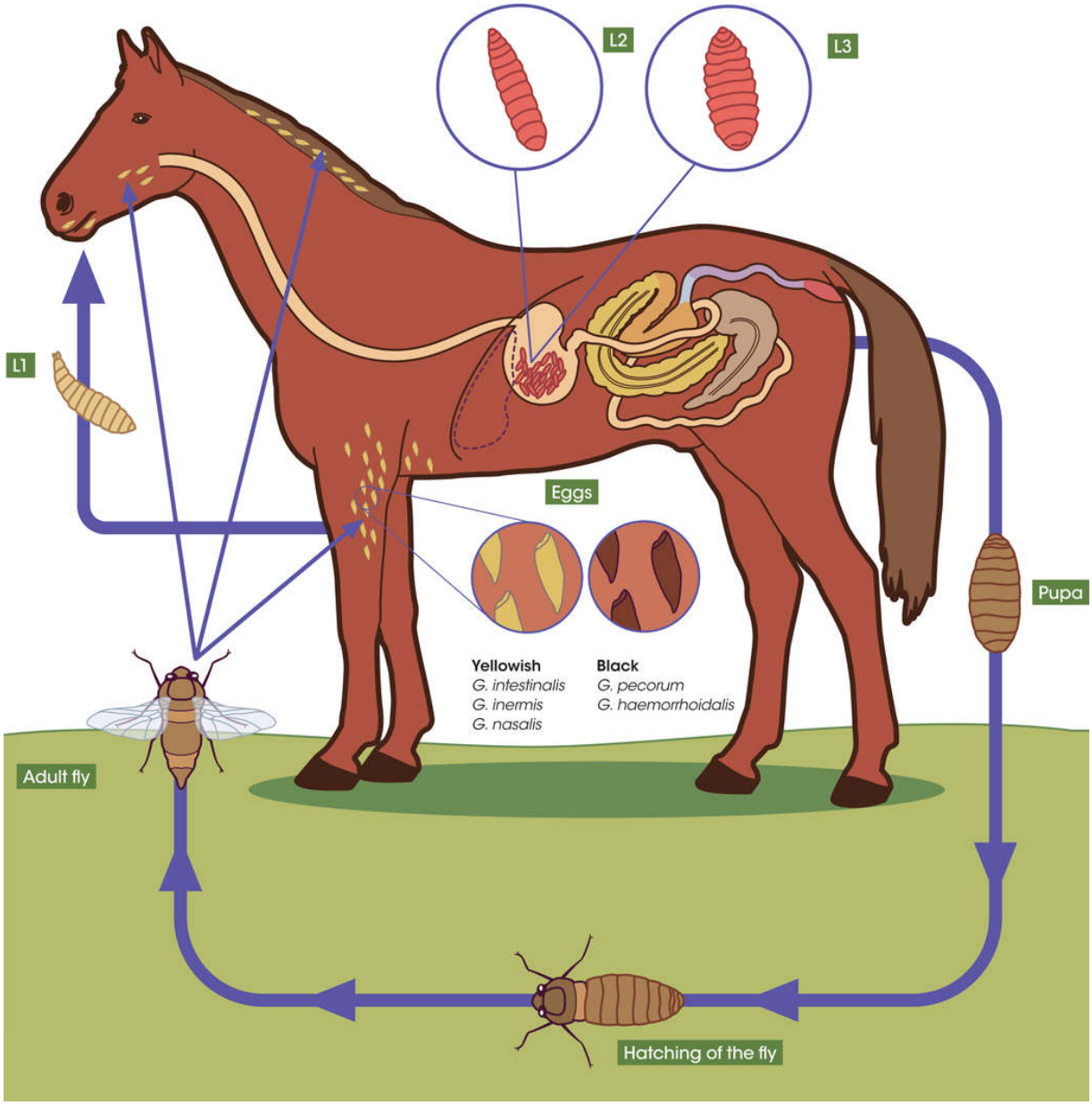
The L2 stage measures 6–12 mm in length, has 12 segments, a bulging posterior end with 2 stigmatic plates, and a blood red colour caused by the ingestion of blood from the tissues. The larvae attach to the gastric mucosa by means of buccal hooks, moulting to the L3 stage, which remains in the same location for several months until its development is complete.

3 Would it be advisable for the owner to place insecticide strips inside the boxes to prevent egg laying?

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Since the flies avoid closed areas, the use of insecticide strips inside boxes is ineffective.

Schematic depicting the biological cycle of *Gasterophilus*



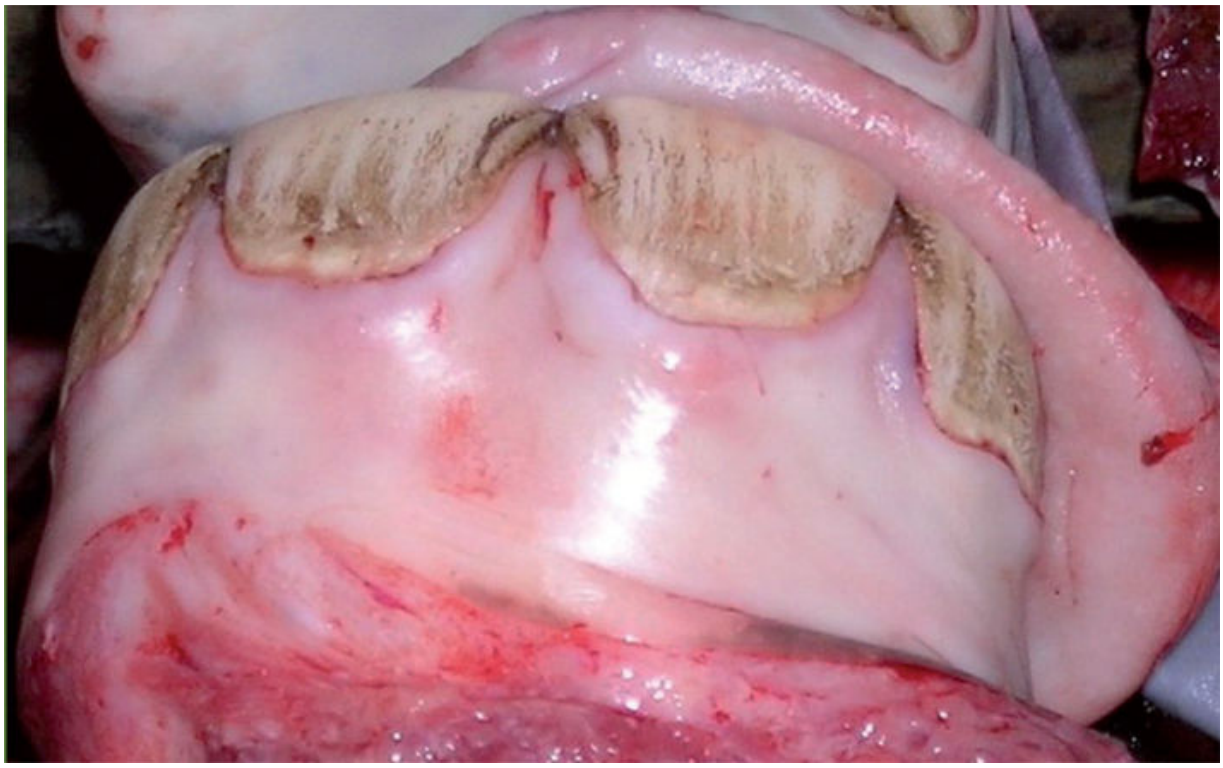
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3 What are the effects of *Gasterophilus* ?

In the month of October a veterinary surgeon is asked to evaluate the injuries of a donkey that has fallen down a slope. Examination reveals a possible pelvic fracture, as suggested by evident pelvic asymmetry, pain, and crepitus. In the absence of a confirmatory radiological diagnosis, the anamnesis reveals that this is not the first case of this type of accident on the farm. The owner says that the animals, for no apparent reason, suddenly run through the farm seeking shaded areas. During one such incident, this donkey fell and was injured. He says that these incidents usually coincide with a period of food rejection that then ends abruptly. A thorough examination of the animal reveals mild stomatitis with ulcers on the tongue and pharynx.

1. Using a scalpel a small larva is removed from one of these lesions. What is it?
2. How can the behaviour of the animals be explained?

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1 Using a scalpel a small larva is removed from one of these lesions. What is it?

It is an L1 stage of *Gasterophilus* .

2 How can the behaviour of the animals be explained?

This parasite has a negative effect on the host for two very different reasons. During oviposition adult flies harass and distress equids in the middle of the day. The response of the animals, which includes violent shaking of the head and limbs, or even fleeing when plagued by large numbers of flies, can result in accidents.

However, in general, the most obvious negative effect of gasterophilosis is caused by the invasion, development, and settlement of larvae in both the skin and the mucous membranes of the digestive system. The earliest stages develop in the buccal, lingual, or gingival mucous membranes, giving rise to variable local inflammation that causes eating difficulties and pain when chewing or swallowing. The recently hatched L1 stage of *G. nasalis* crawls

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towards the labial commissures, enters the oral cavity and migrates through the mucous membranes of the gums and palate, while *G. inermis* larvae produce characteristic clinical signs following their direct migration to the mouth through the skin and connective tissue, resulting in subcutaneous myiasis, also known as “summer dermatitis” or “serpiginous dermatitis”. This particular condition is very common in countries close to Spain (e.g. eastern France).

Gastric injury caused by *G. intestinalis*. Larvae in the gastric mucosa cause inflammation in the locations in which the settle, and can induce gastric dilatation and occlusion or stenosis, particularly in the pyloric zone. In some cases gastric perforation may be observed. The areas in which the larvae adhere are characterised by crateriform depressions, surrounded by chronic inflammation with local eosinophilia.





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The most common and abundant species, *G. intestinalis* , invades the host when the larva, stimulated by the temperature of the tongue, emerges from the egg, adheres to the lingual mucosa, and subsequently reaches the base of the tongue or the soft palate. When the larvae of this species leave the tongue they pass to the stomach, where they form compact groups in the non-secreting squamous mucosa, in front of the *margo plicatus* , where ecdysis occurs to give rise to the L3 stage. By settling in this location the parasite can remain in the host for long periods of time without provoking an acute clinical presentation of gastritis, even when the they reach their maximum size.

The main pathogenic effect of *Gasterophilus* species is therefore attributed to the L3 stage when it attaches and grow in its final location, be it the stomach, duodenum, or rectum. The attachment and feeding of the larvae induces a local peripheral inflammatory response at the site of attachment caused by penetration of the mucosa by the oral hooks, the continuous extraction of nutrients, and the mechanical effects of their mere presence and the volume they occupy, resulting in gastric dilatation and occlusion or stenosis, predominantly affecting the pyloric and duodenal areas. Furthermore, the erosion of the mucosa allows the entry of agents that can provoke secondary infections and even abscesses. Gastric perforations are uncommon, although the chronic inflammatory reaction usually leads to thickening of the gastric wall. In the stomach, the areas to which the larvae adhere display crater-like depressions surrounded by chronic inflammation with local eosinophilia. These areas are separated by 1–2 cm.

In mild infestations in which the larvae are located in the cardia of the stomach, evident clinical signs are absent, even in animals harbouring up to 200 larvae. However, in animals in which the larvae settle in pyloric regions, digestive disorders are common, especially in young, malnourished, or weak animals.

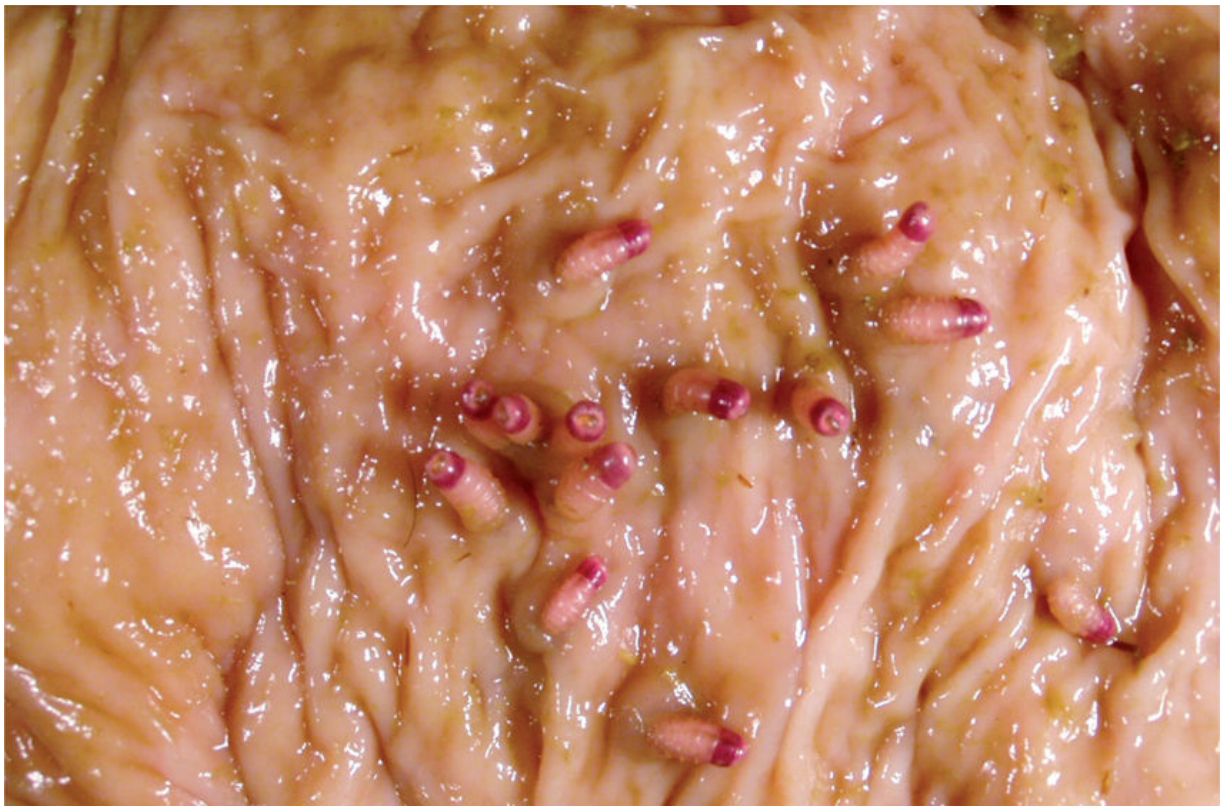
Severe infestations are uncommon and tend to occur in animals that spend long periods of time outdoors and do not receive any type of antiparasitic treatment. These severe infestations result in disorders of the motor and secretory activity of the stomach, and consequent digestive alterations and malnutrition. Stenosis of the pylorus or duodenum may also be observed.

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Duodenal lesion caused by *G. nasalis*. The L3 stage in the intestinal mucosa provokes an intense local reaction that progresses to chronic inflammation, with the formation of circular ulcers with raised borders that are particularly visible when the larvae are released. The sites of attachment are surrounded by a reddened halo, swelling, and in some cases small haemorrhages. Perforations and peritonitis may be observed.

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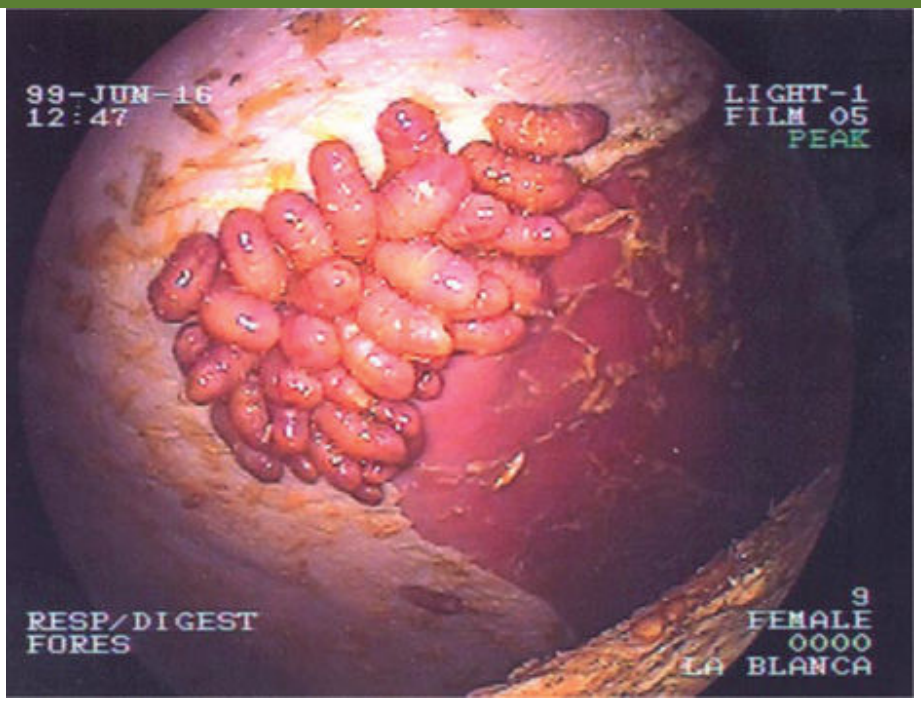
Lesion of the rectal mucosa caused by *G. haemorrhoidalis* . The larvae attach to the distal end of the digestive tract, mainly the rectum, until reaching maturation, at which point they measure almost 2 cm in length. They are firmly anchored to the wall of the large intestine, and are lighter in colour at the anterior end, through which they attach to the rectal mucosa. Despite the presence of relatively few larvae, they can cause defecation disorders. They are commonly detected during rectal palpation.

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4 How can we tell if a horse is parasitised by gasterophilids?

The patient is a 5-year-old mare with acute colic. After undergoing an examination, gastric dilatation is suspected. During gastroscopy, performed to relieve intragastric pressure, numerous red larvae over 1 cm in length are observed adhering to the stomach wall. The animal had spent long periods on an open-air farm and had been dewormed with fenbendazole 1 month earlier.

1.How is this clinical picture explained?
2.What lesions has the parasite caused in the digestive mucosa?



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1 How is this clinical picture explained?

Fenbendazole is an anthelmintic with no activity against fly larvae. As the clinical manifestations are highly variable, it is very difficult to diagnose gasterophilosis based only on clinical signs. However, epidemiological data, such as the time of presentation and whether the patient has grazed continuously, are of great value. The observation of eggs attached to the coat during the summer months or at the beginning of autumn (depending on the geographical area) and the possibility of performing a necropsy will allow establishment of a reliable diagnosis.

There is no possibility of a coprological diagnosis, although sometimes the L3 stage can be observed in faeces. To date no serological test been commercialised. Indirect methods include allergic tests using extracts of larvae taken from the conjunctival sac and skin, although these have not proven particularly successful. Several serological tests have also been used to detect circulating antibodies with nonpurified L3 antigen. These include double immunodiffusion (DD), counterimmunoelectrophoresis (CIE), indirect haemagglutination (IH), thin-layer immunoassay (TIA-Western blot), and gel diffusion-ELISA (DIG-ELISA), the last two of which are particularly sensitive.

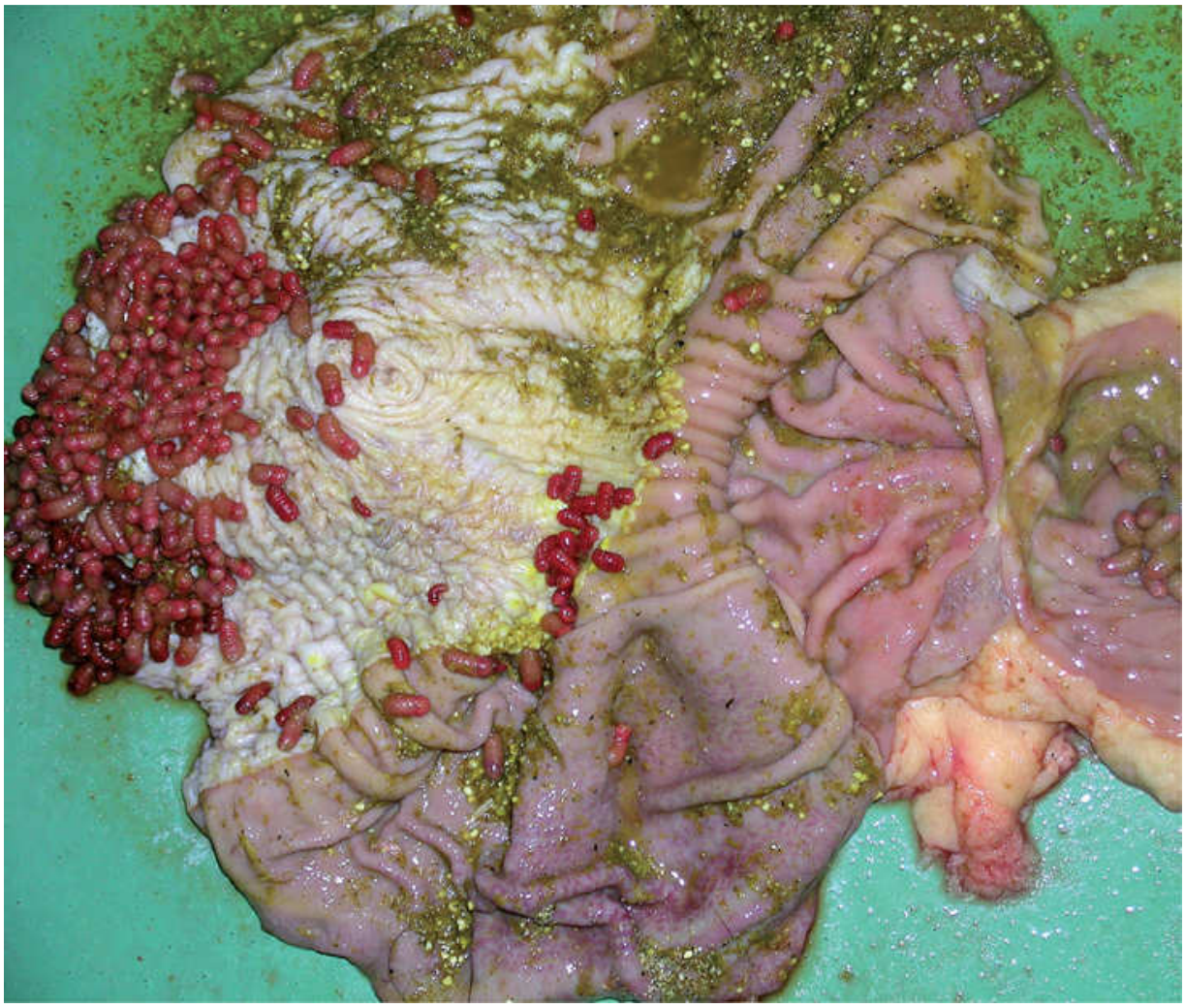
Digestive endoscopy can allow visualisation of larvae attached to the gastric wall. Similarly, rectal examination allows the detection of larvae of certain *Gasterophilus* species. The species most commonly detected in the rectum is *G. haemorrhoidalis* .

2 What lesions has the parasite caused in the digestive mucosa?

The lesions caused by the L1 stage in the anterior digestive system are limited to mild stomatitis and, in rare cases, small ulcers on the tongue, pharynx, and oesophagus.

Attachment of the L3 stage to the gastric and intestinal mucosa provokes an intense local reaction, with proliferation of the epithelial layer and abundant cellular infiltration. Over time this evolves to chronic inflammation, with the formation of circular, crateriform, definitive ulcers with raised borders. In advanced stages these ulcers surround almost one third of the body of the larva. This effect is more striking in the rectal mucosa.

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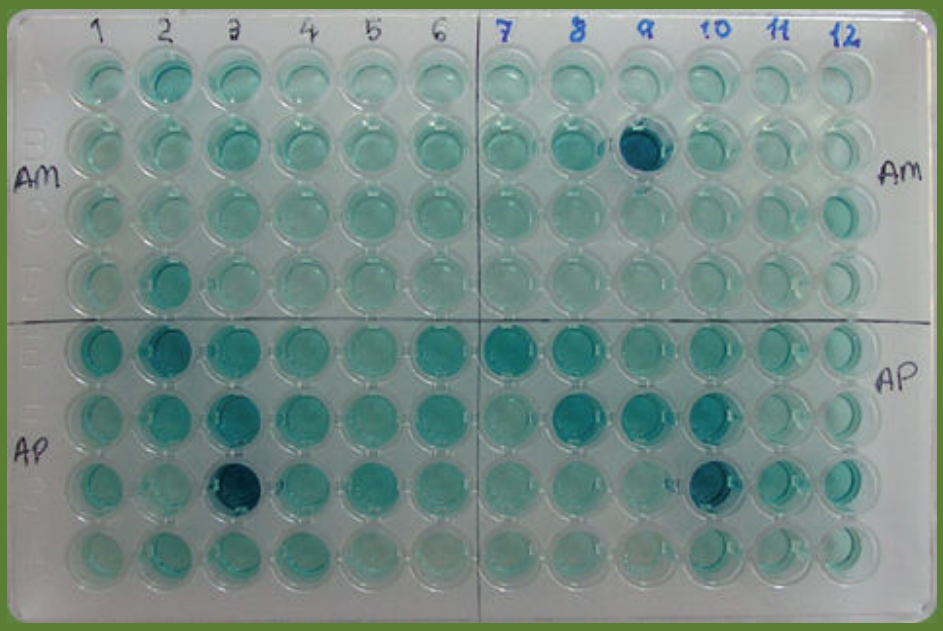
Post-mortem diagnosis of *Gasterophilus* in the stomach: clusters of numerous larvae, which produce crateriform lesions at the sites of attachment, are often observed in the area of the non-secretory mucosa.

Chronic inflammation and hyperplasia cause an increase in the thickness of the gastric wall, especially in the area near the *margo plicatus* , that is evident externally. In these cases, large numbers of larvae can be observed upon making an incision along the greater curvature of the stomach. The larvae are generally attached to the squamous mucosa. In the spring or summer months when the larvae emerge, these lesions are filled with scar tissue, and within a few weeks only a remnant of the lesion remains. The gastric lesion has a honeycomb-like appearance.

If the larvae are attached in the pyloric region or are in the duodenum (*G. nasalis*), ulcers of 3–4 mm in diameter, with a reddened background and thickened border (resembling smallpox lesions) are evident following their

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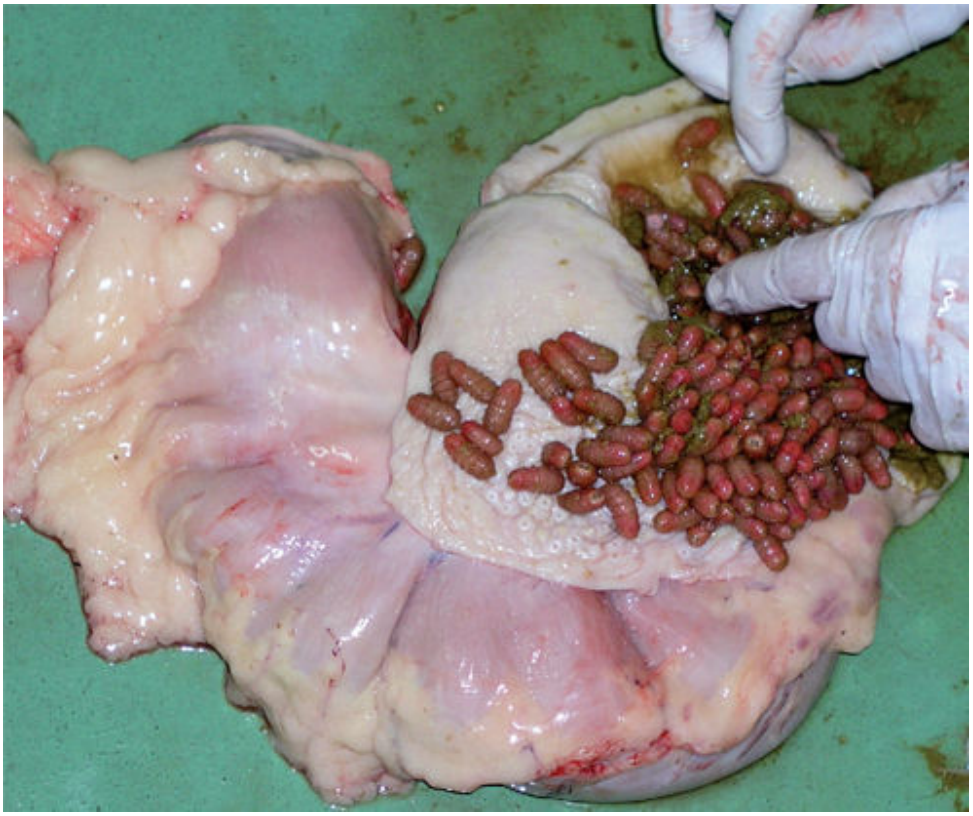
detachment. Sites of attachment are surrounded by a reddened halo, swelling, and in some cases, small haemorrhages. In some cases well-defined abscesses on the digestive wall and even perforations with peritonitis may be observed.



In vivo diagnosis (ELISA). In vivo diagnosis of equine gasterophilosis can be confirmed by laboratory techniques. Various techniques can be used to detect circulating antibodies with L3 antigen.
The most sensitive are thin-layer immunoassay (TIA-Western blot) and diffusion-in-gel ELISA (DIG-ELISA).

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It is important to rule out other parasitic diseases, such as parascariosis and intestinal strongyloidosis, and infectious diseases, such as infectious anaemia, which affects horses of all ages and can be associated with febrile episodes with little or no change in appetite.

The clinical prognosis is benign in most cases. However, the possibility of complications of a traumatic nature (e.g. perforations, occlusions, colic) should be anticipated. In economic terms, the prognosis is particularly unfavourable in young equids (foals) and very old animals.

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In vivo diagnosis: in hotter months, *Gasterophilus* females hover around equids and lay their eggs during flight, without landing on the host. It is common to observe the whitish eggs of *Gasterophilus* attached to the hair of the mane and forelimbs of animals on pasture. Detection of the eggs is easier in animals with darker coats.

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5 How is *Strongyloides* transmitted?

A 2-week-old foal has mild diarrhoea that has not improved despite treatment with an antidiarrhoeal agent. Although the animal has not lost its appetite, it is somewhat apathetic and spends a lot of time lying on the ground, even in its own faeces. The results of the laboratory analysis are negative, but the laboratory has requested a new sample, and has emphasised that it must arrive at the laboratory in good condition. The previous sample was lost and remained at room temperature for 3 days before analysis. The second analysis reveals the presence of small, ovoid, embryonated eggs, despite the fact that the faecal sample was analysed less than 2 hours after collection.

1.How can nematode eggs be shed by such a young animal?
2.Why are they embryonated?

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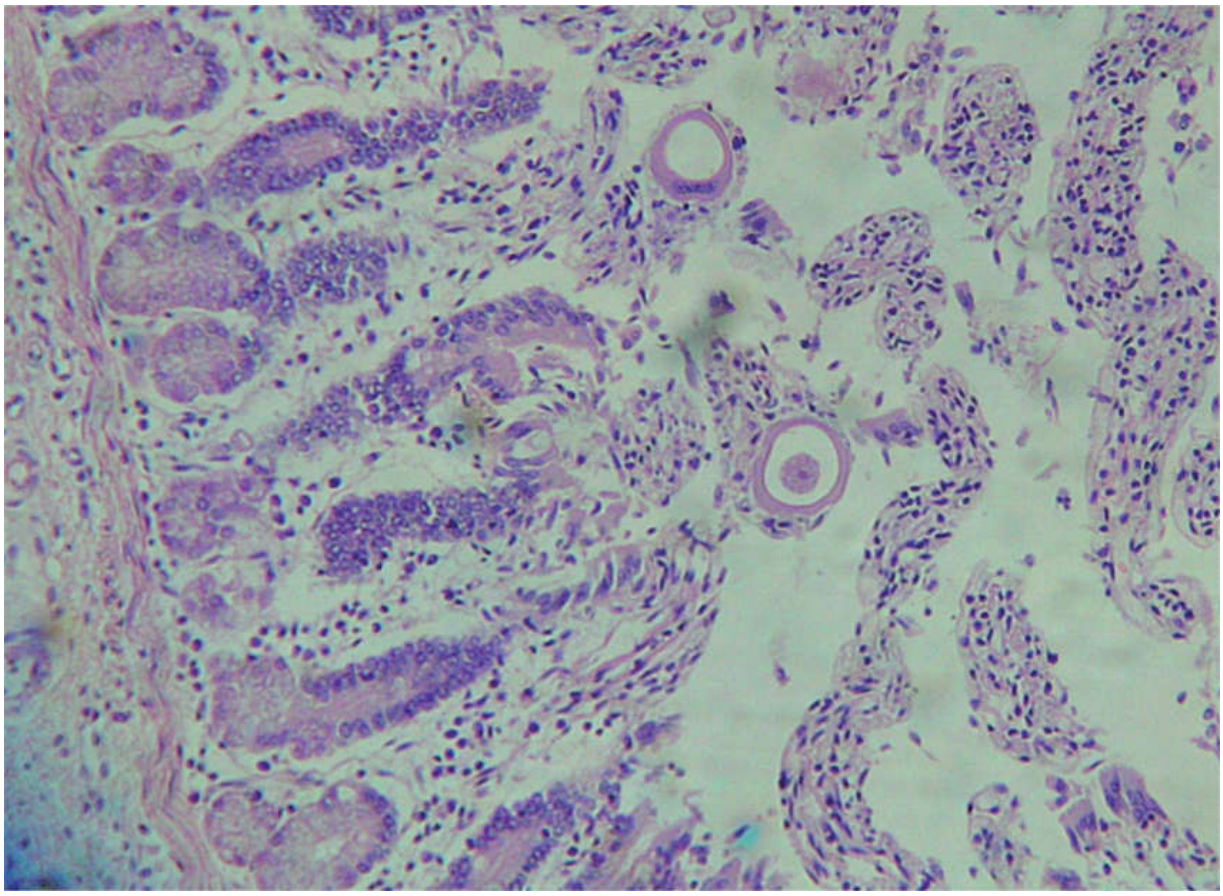


1 How can nematode eggs be shed by such a young animal?

These are *Strongyloides westeri* eggs. Foals are generally infected by this species following penetration of the skin or entry via the oral route following ingestion of contaminated food. However, these routes of infection appear to be secondary; it has been shown that transmission predominantly occurs via the mother's milk, in which larvae are present between 4 and 47 days after foaling. In the case of cutaneous infection, as described for other strongyles, the infective larvae penetrate the capillaries and reach the bloodstream, and are then carried to the pulmonary alveoli and ascend via the bronchi and trachea. They are then swallowed and reach the small intestine, where they complete their development, reaching adulthood within 5 to 7 days. In cases of oral infection following consumption of contaminated milk or food, the

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larvae cross the oral and oesophageal mucosa, and follow the aforementioned migratory path to reach the intestinal lumen. The shedding of larvae in milk is not continuous; numbers are generally higher in the morning, probably due to the accumulation of larvae in the mammary cistern while the animal rests.



Histological sections show marked epithelial desquamation and congestion of the mucosa, a consequence of the presence of the worms, which are surrounded by an exudate consisting mainly of desquamated epithelial cells, lymphocytes, plasma cells, and neutrophils, as well as some macrophages and eosinophils.

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The main clinical manifestation is diarrhoea, with very liquid stools in severe cases, occasionally accompanied by mild pain and moderate fever. Anorexia and polydipsia may be observed. Congestion of the mucous membranes, an accelerated and weak pulse, and a tendency to decubitus are observed in severe infections. Affected foals become weak, stop feeding, and can die within 2–3 days of onset of the process. In milder cases, spontaneous cure occurs within a few days.

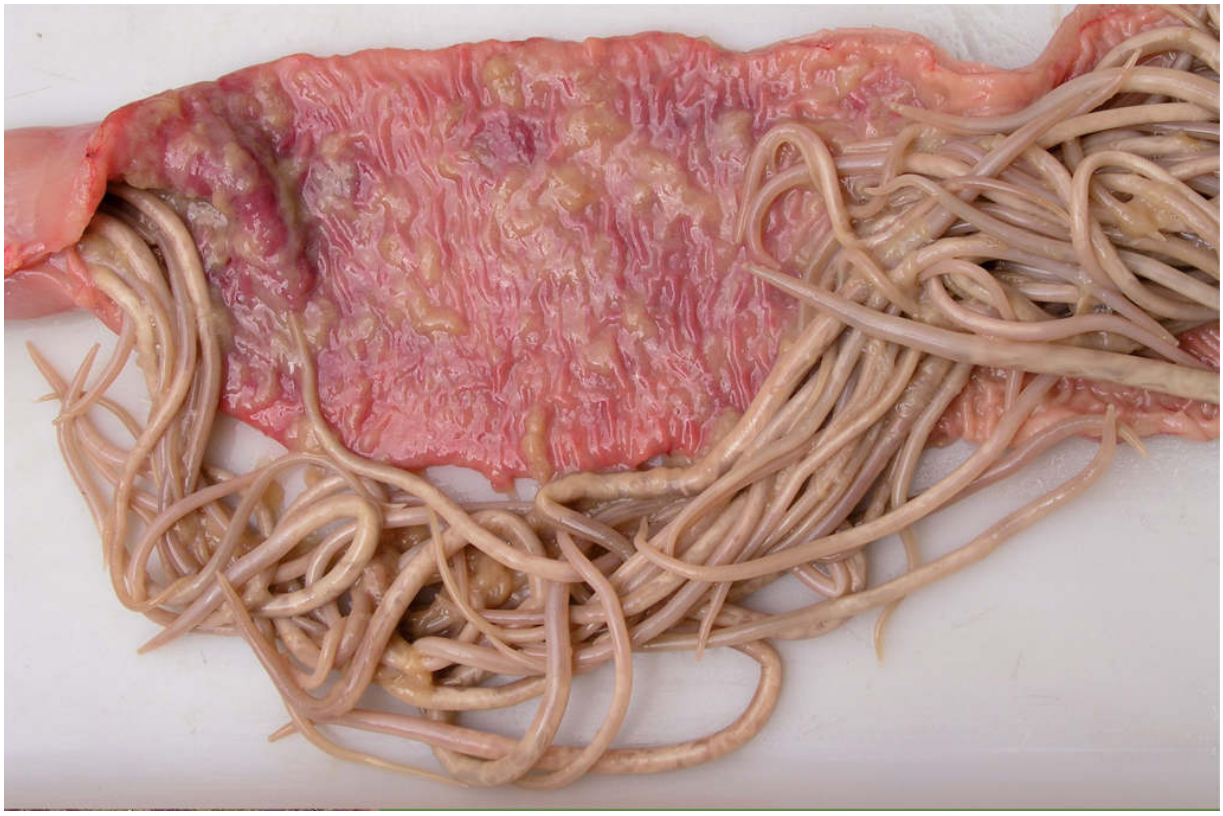
In experimental conditions, infections with a very high number of larvae are required in order to elicit clinical signs of disease. The administration of more than 4 million larvae causes diarrhoea and death in foals, while 100,000

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larvae can cause enteritis and macroscopic and microscopic lesions, but without clinical manifestations.

Penetration of the intestinal mucosa by the larvae and the production of adult females in that same location result in inflammation of the intestinal mucosa with atrophy of the villi and a decrease in the mucosa's capacity to absorb water and nutrients. Consequently, the amount of water in the contents of the intestine increases and the faeces become diarrhoeic. Dehydration can be fatal in very young animals. Affected foals lose weight and exhibit a deterioration in their general condition.

In infected foals, strong immunity is established at 15–23 weeks of age, after which the larvae become immobilised in different tissues, but are not eliminated by the immune reaction.



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In equids, strongyloidosis is caused by *Strongyloides westeri,* which preferentially affects foals of a few weeks or months of age and manifests with mild or moderate intestinal alterations. Except in very severe infections and in animals weakened by other pathologies, progression is usually favourable and resolution is achieved. In foals, strong immunity is established at 15–23 weeks of age and appears to persist for life. Mixed infections with *Parascaris* spp. are common.

The main clinical manifestation is diarrhoea, with liquid faeces in severe cases, which may be accompanied by mild pain and moderate colic. Anorexia and polydipsia may be observed. Severe infections are characterised by congestion of the mucous membranes, an accelerated and weak pulse, and a tendency to decubitus. The foal becomes weak, stops feeding, and can die within 2–3 days of the onset of the process. In milder cases, spontaneous resolution occurs within in a few days. The entire length or the anterior third of the small intestine can be inflamed, congested, and even haemorrhagic. The mucosa becomes thickened and reddened, and increases mucus production. The transparency of the mucosa allows visualisation of the worms within.

Due to their biological characteristics, the eggs develop very quickly once excreted by female worms. In faeces not freshly obtained, the eggs may have hatched. In these cases the analysis should focus on the presence of larvae, which are unsheathed and have an oesophagus almost half of the length of the body.

Diagnosis can only be established upon detection of the characteristic eggs in the faeces by coprological analysis using any of the appropriate (preferably quantitative) methods. It is estimated that values of over 5,000 epg (eggs per gram of faeces) in foals, coinciding with diarrhoea of variable severity, are

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compatible with the presence of this parasite. However, higher counts can be obtained in animals without clinical signs.

The differential diagnosis of strongyloidosis should take into account the possibility of other concomitant parasitic processes, especially cryptosporidiosis and coccidiosis, as well as colibacillosis and other bacterial and viral infections that can cause diarrhoea and other nutritional alterations.

The progression of affected animals is usually favourable, resulting in resolution, except in very severe infections.

2 Why are they embryonated?

Within *Strongyloides* eggs, which are located in the uterus of the female, embryonation occurs progressively; the L1 stage can form so rapidly that in some species it hatches inside the host, and free larvae are found in the faeces. In equids, embryonated eggs are excreted and hatch within a few hours within the faecal mass. This feature should be taken into account when performing coprological analyses. If analyses are carried out 6 hours after collection, without refrigeration of the sample, in addition to using a flotation technique, the presence of larvae should be ruled out using a larval migration technique (e.g. Baermann–Wetzel technique).

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6 How do foals become infected with *Parascaris* if the adults are resistant and do not shed eggs?

The patient is a 3-month-old foal with colic and respiratory problems. He comes from a riding school where several females are being reared. The animal was stabled for some time, alternating with periods of grazing on common pasture frequented by both young and adult animals. No information on recent deworming is available. The patient shows signs of acute pain, is restless, and rolls frequently. During nasogastric catheterisation, several adult specimens of *Parascaris* spp. are obtained. The patient's condition worsens and a laparotomy is performed. This reveals perforation of the proximal duodenum and the presence of numerous adult forms of *Parascaris* spp.

1. Are such high worm burdens common in foals so young?
2. What is the relationship between *Parascaris* and the respiratory clinical signs?
3. In addition to exploratory laparotomy, what other approaches can be used to confirm the aetiology of this process?

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1 Are such high worm burdens common in foals so young?

Young equids are much more susceptible than adults, especially those between 3 months and 9 months to 1 year of age, and exhibit clinical signs of disease. Adult equids can be parasitised, although in these cases the parasitic loads tend to be very low.

The female worms are very prolific and lay hundreds of thousands of eggs, which are shed in the faeces. Like the eggs of the other ascarid species, they are highly resistant to unfavourable environmental conditions and the larvae develop protected within the egg until reaching the sheathed L3 stage, which is infective and remains inside the egg.

The egg cover protects the larva from chemical and environmental agents. Eggs that do not develop when temperatures are low remain viable for many months.

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Eggs containing infective larvae retain their infective capacity for years. Infection occurs upon ingestion of the L3 form in food or drinking water. Once ingested, the egg hatches in the host's intestine. As described for other nematodes, this process appears to require a host stimulus.

2 What is the relationship between *Parascaris* and the respiratory clinical signs?

The larvae of *Parascaris* spp. embark on an entero-pneumo-enteric migratory pathway until they reach the small intestine. After 48 hours the larvae reach the liver, where they remain for several days. Seven to 14 days after infection they reach the lungs, where they moult twice, passing through the L3 stage to reach L4. These larvae migrate through the pulmonary alveoli to the bronchioles, ascend via the bronchial tree, and are expectorated. From the trachea they reach the pharynx, at which point they are swallowed and settle in the intestine. The migratory phase ends at 4 weeks and the larvae, after moulting again, transform to the fifth or immature adult stage and begin a period of rapid growth that lasts about 10 weeks, during which time they complete their development and become sexually mature adults.

The preferred location of adults is the duodenum and the anterior part of the jejunum, although in very severe infections they can be found throughout the entire small intestine. If the worms are very numerous and are forced to compete for space and nutrients, they tend to be smaller in size and many can die, likely in part due to immune factors. Dead, often partially digested adults are expelled with the faeces.

Foals can shed parasite eggs on reaching 10–13 weeks of age, indicating that infection can occur immediately after birth, probably caused by contamination of the teats of the mother with eggs present in the environment. However, this does not appear to be a major route of infection. Although no larvae have been found in colostrum or milk, lactogenic transmission cannot be ruled out completely.

Male and female *Parascaris* . The males are smaller and have a rounded or blunt conical posterior end, with 2 small lateral caudal wings and numerous preanal papillae arranged in 3 longitudinal subventral rows. The spicules are 2–2.5 mm long and are of equal size. The females have a small conical

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protuberance at the caudal end. The vulva is located about one quarter of the total body length from the anterior end.

Anterior end of *Parascaris* . *P. equorum* is a large nematode. The males measure 15–28 cm × 3–6 mm, and the females 18–50 × 2–2.5 cm. They are yellowish-white with a flexible body. At the anterior end are 3 quadrangular lips, which are divided into 2 parts by a transparent membrane that forms horizontal grooves on their inner surface. The dorsal lip has 2 large double papillae, while each of the subventral lips has a large, double papilla. The main lips are separated by small interlabia. Cervical wings are absent.





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3 In addition to exploratory laparotomy, what other approaches can be used to confirm the aetiology of this process?

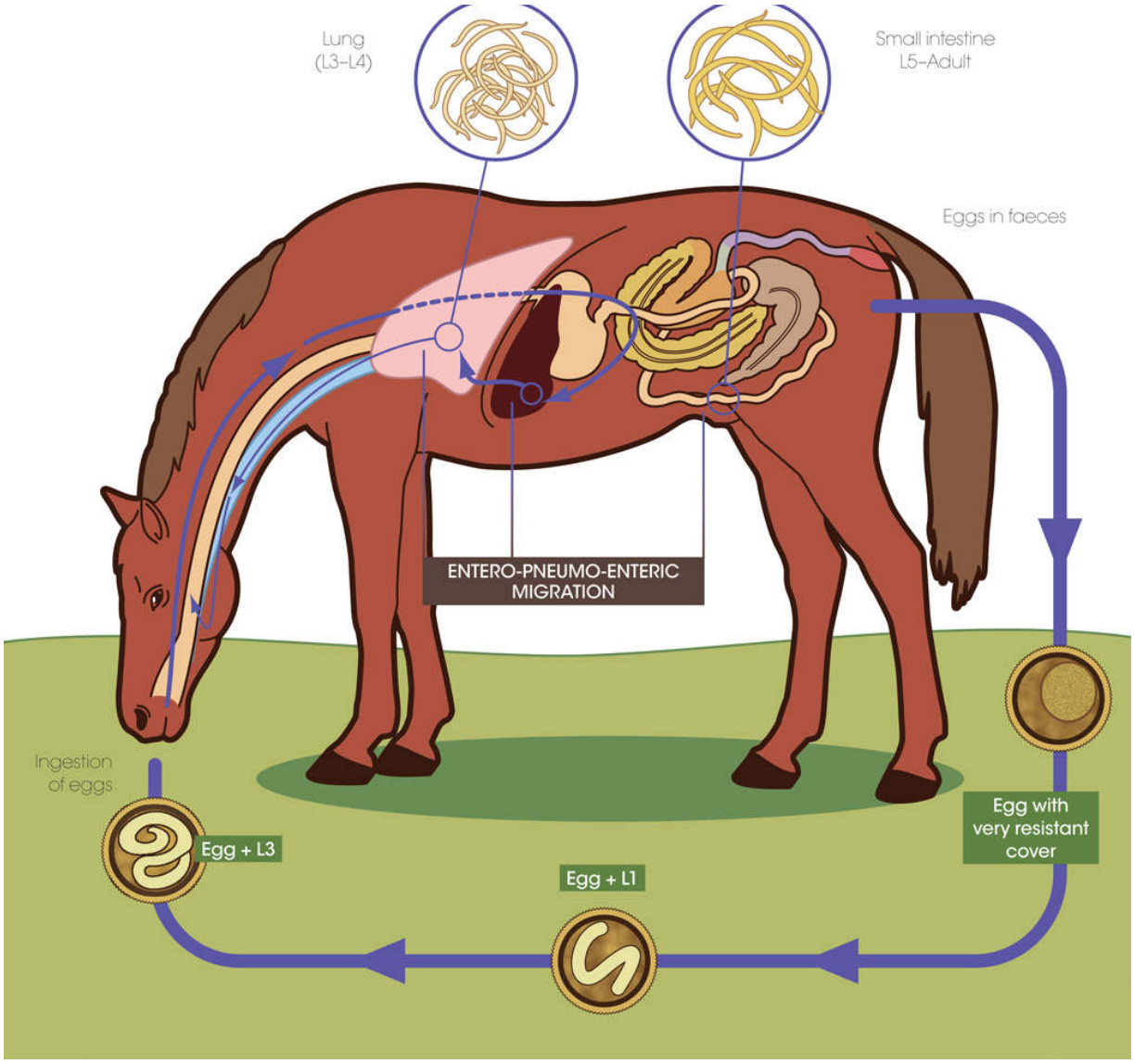
The most frequently identified species in equids throughout the 20th century has been *Parascaris univalens* (a sister/twin/cryptic species of *P. equorum* that is morphologically indistinguishable but can be identified using karyotyping or molecular techniques). Therefore, *P. univalens* should be considered the main species in equids.

Once the eggs are isolated, molecular techniques can be used to determine whether the species is *P. univalens* or *P. equorum* .

Egg counts are initially very high during this phase, but decrease between 6 and 12 months of age, after which eggs are no longer detected in the faeces. Experiments have shown that age-dependent immunity develops in foals, and is established around 6 months, even in animals that have not had previous contact with the parasite.

Life cycle of *Parascaris*

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7 What are the consequences of *Parascaris* infection?

On a stud farm, all foals exhibit deterioration of their physical condition and have developed foul-smelling diarrhoea. Compared with previous years, weight gain has decreased to half of normal values. In addition, the foals have bristly, dull hair and the visible mucous membranes are pale. Some have stopped eating while others exhibit pica or malacia. The loss of weight is so significant that bony prominences are highly evident, and the abdomen (belly) is markedly distended. The most severely affected animals are weak and show a tendency towards decubitus, and their general condition deteriorates resulting in cachexia. In some cases white parasites of over 10 cm in length are expelled with the faeces.

1. What has caused this clinical picture?
2. Are there any other clinical signs associated with this parasite?

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1 What has caused this clinical picture?

The consequences of parascariosis are linked to the endogenous biological cycle (the migrating larvae, the immature stages, and the adult forms in the intestine).

In the prepatent phase, parascariosis should be suspected in foals of 2–4 months of age with mild respiratory clinical signs, especially those that come from studs with a history of parascariosis and that display growth retardation and weight loss. Eosinophilia is elevated in this phase.

The penetration and movement of the larvae in the liver during the invasion phase result in the rupture of numerous blood capillaries, causing haemorrhages beneath the hepatic capsule and parenchyma, but do not give rise to clinical manifestations.

Between 7 and 14 days after infection, the larvae invade the lungs and produce petechiae and small haemorrhages on the surface and in the lung tissue caused by rupture of capillaries. At the same time, they stimulate the secretion of mucus that surrounds the larvae in the alveoli and bronchioles.

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However, the most important reactions are bronchitis and eosinophilic bronchiolitis. The cuticle expresses eosinophil chemotactic factor of anaphylaxis, which attracts eosinophils to the area. The histamine released by mast cell degranulation in the areas invaded by the larvae is destroyed by the antihistamines of the eosinophils. This period of eosinophilia is temporary and subsequently gives way to lymphocyte invasion, which results in the formation of subpleural nodules.

All these phenomena affect respiratory function. The increase in mucosity and the formation of inflammatory lesions in the alveoli, bronchioles, and bronchi lead to difficulties in pulmonary ventilation and the appearance of respiratory signs that last from 2 to 4 weeks post-infection and can be exacerbated by the presence of secondary infections.

After this pulmonary phase, the larvae migrate to the small intestine where they complete their development. Their rapid initial growth and the high levels of egg production by fertile females require large amounts of nutrients, which are provided at the expense of the nutrients needed for growth of the animal. Affected animals exhibit hypoalbuminaemia, an increase in the body water to body mass ratio, and a decreased ability to incorporate methionine from the diet into plasma proteins. The consequence is a deterioration in the animal's general condition, resulting in delayed or arrested growth.

Complications requiring surgery (intestinal invaginations, occlusions, and perforations) associated with parascariosis appear to result from alterations in intestinal motility and the mechanical action of the groups of worms characteristic of severe infections.

2 Are there any other clinical signs associated with this parasite?

The first clinical signs observed in foals with parascariosis are respiratory disorders: tachypnoea and, occasionally, dyspnoea, cough, and white or greyish nasal discharge. These signs are observed during weeks 3 and 4 post-infection and, although other clinical signs are usually not observed, they may be accompanied by mild fever, mild anorexia, loss of sensorium, and growth arrest. These manifestations disappear during the following 1 or 2 weeks, but the foals do not recover, and the infection progresses to the next phase: the intestinal phase.

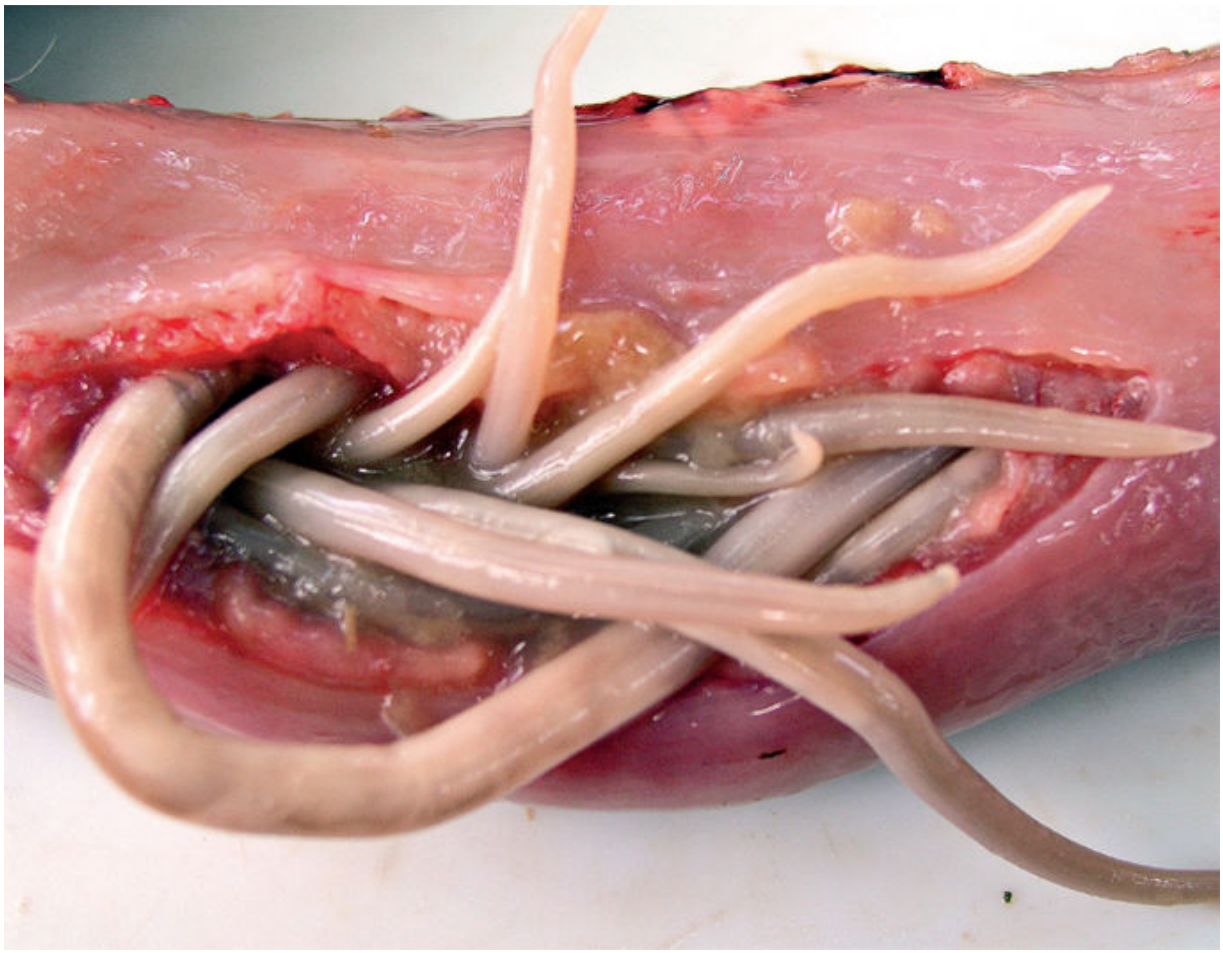
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Foals in the first months of life are most vulnerable to the pathogenic action of *Parascaris* spp. Possibly due to prior infections and the acquisition of immunity, foals of over 1 year of age do not develop such a marked clinical picture, and their general condition and growth are generally unaffected.

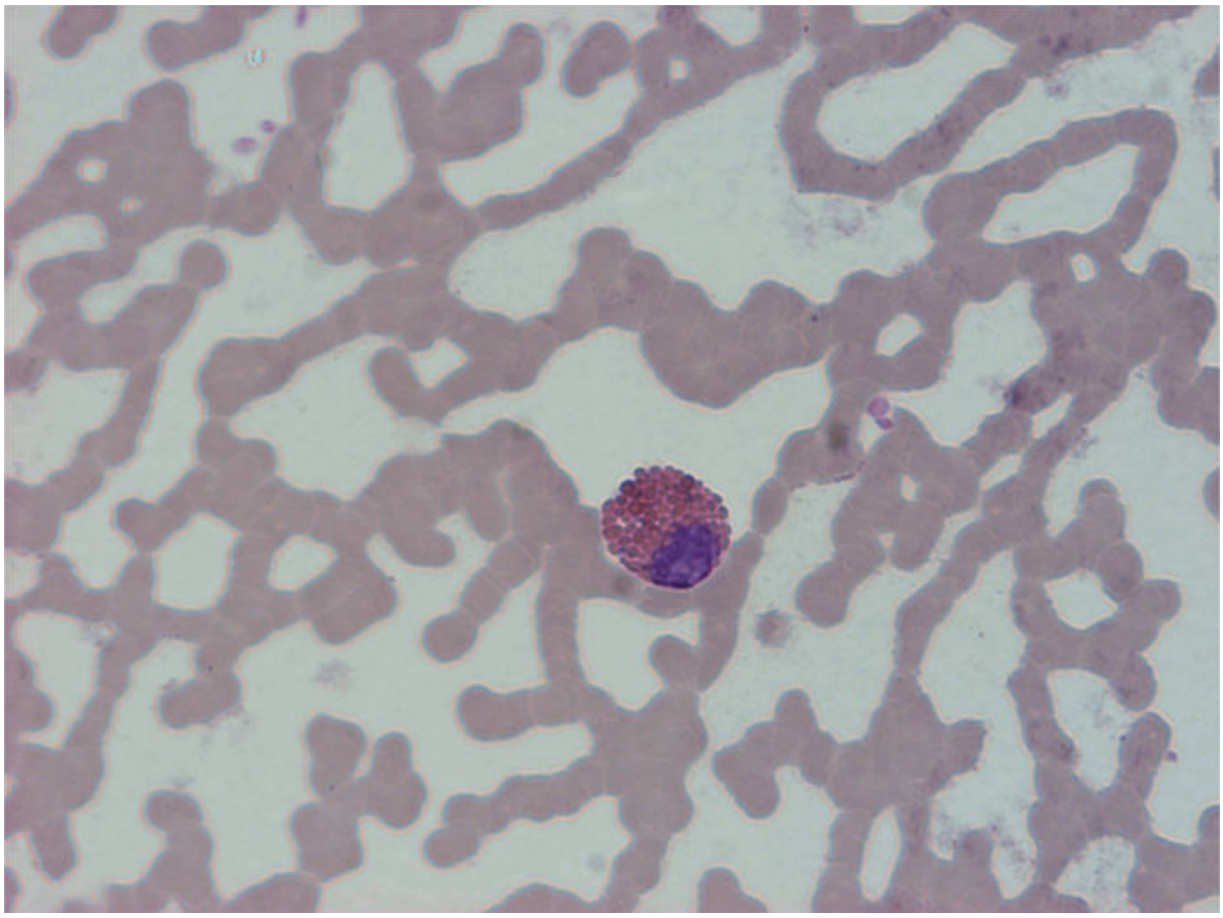
Complications that may be observed include colic due to intestinal occlusion or intussusception and acute abdominal signs caused by perforation of the intestine with consequent peritonitis. These complications usually appear in autumn or early winter, at the end of the lactation period, or in weaned foals. In these cases, death is the most common outcome.

The muscle layer of the mucosa of the small intestine becomes thickened and oedematous. Small lesions caused by the worms may be evident in the mucosa. The parasites are found either individually or in tangles, predominantly in the anterior half of the small intestine. If the intestinal wall is ruptured, the content is released into the peritoneum, in which corresponding inflammatory lesions develop. Perforations occur along the mesenteric fixation, at sites in the intestinal wall weakened by lesions caused by penetration by the larvae of the blood vessels.

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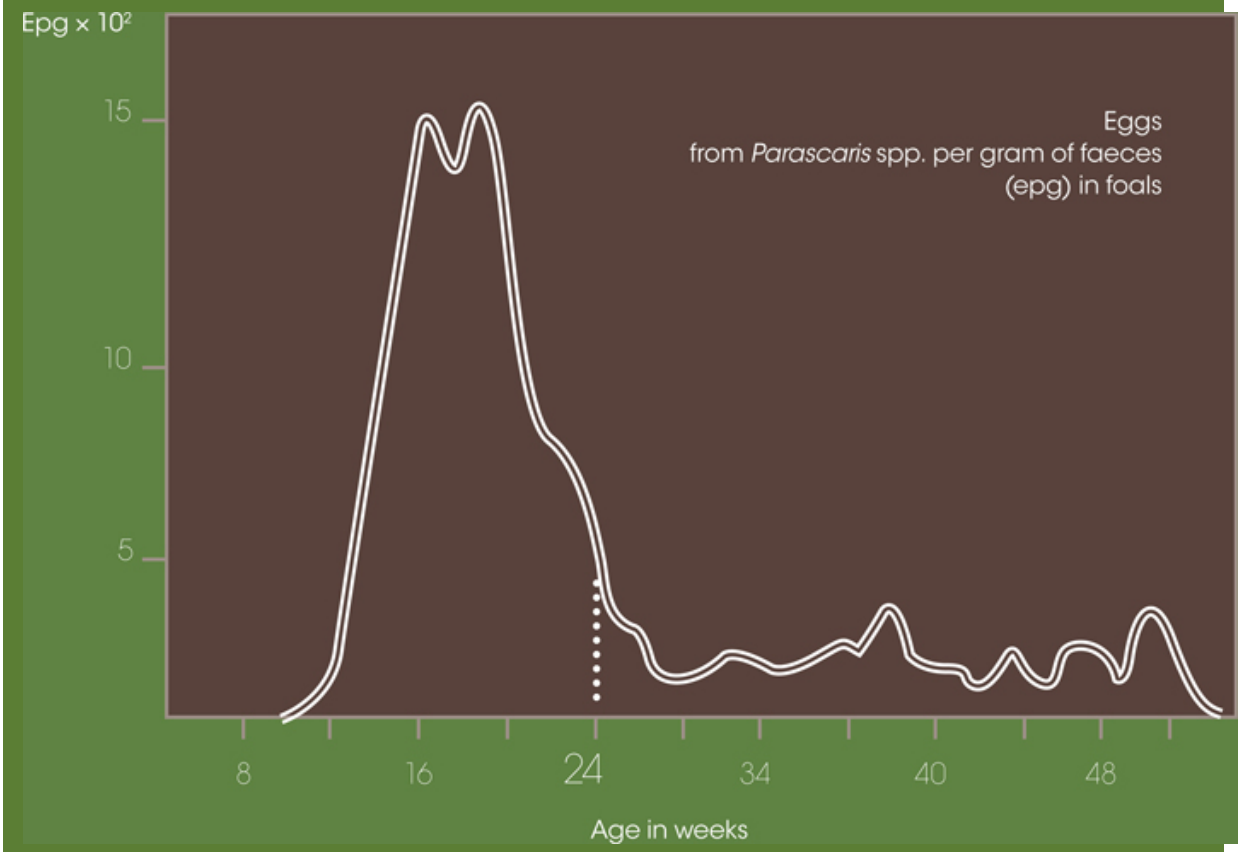


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During the larval migration phase blood tests reveal marked eosinophilia, which becomes more severe with increasing numbers of invasive larvae, and peaks between 10 and 40 days post-infection. Subsequently, eosinophilia diminishes and even disappears, but in cases of reinfection the number of eosinophils increases again, even surpassing the values observed in the first infection. At the beginning of the phase of worm development in the intestine, about 30 days post-infection, haematocrit levels decrease slightly, but are restored in 3–4 weeks. Comparable variations in the levels of haemoglobin, red blood cells, and leukocytes have been observed.

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There is a fairly close relationship between the excretion of *P. equorum* eggs and the age of the infected animals. In natural conditions, shedding peaks at around 4 months of age, rapidly decreasing to very low levels from 6 months of age. In immunocompetent animals of about 12 months of age, shedding is practically nil. During the period of maximum shedding, the environment is contaminated with millions of eggs.

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8 Why is it so difficult to eliminate *Parascaris* from a farm?

In a stud located in Aranjuez (Madrid), a 3-month-old foal with respiratory signs has died. Necropsy reveals a moderate amount of nematodes, identified as *Parascaris* spp., in the small intestine. The parasite control programme was apparently appropriate, consisting of bimonthly administration of anthelmintics with rotation of active ingredients during the first year of life. Coprological analysis of other animals reveals the presence of eggs. Embryonated eggs are also detected in enclosed areas and on the pasture.

1. What role do mares play in the transmission of parascariosis?
2. How long do embryonated eggs survive in the environment?
3. What measures can be taken to reduce environmental contamination and control this parasitosis?

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1 What role do mares play in the transmission of parascariosis?

The infection of foals is horizontal, and is primarily mediated by parasitised young and adult horses that can harbour a certain number of worms and play a key role in disseminating the parasite eggs. Lactogenic transmission has not been demonstrated. It has been calculated that an infected foal can shed up to 13 million eggs daily and that an adult horse (which defecates 10–15 kg/day) with a mild infection that sheds 50 epg contaminates the soil with 500,000–750,000 *Parascaris* eggs per day. Given the marked resistance of the eggs to unfavourable environmental conditions and to disinfectants, rapid elimination of parascariosis from a farm should not be anticipated.

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All stages of the parasite respond well to current therapeutic approaches. Therefore, once diagnosis has been established, it should not be difficult to eliminate. However, affected animals are slow to recover after deworming, and patients that have been severely weakened are unlikely to develop fully upon reaching adulthood.

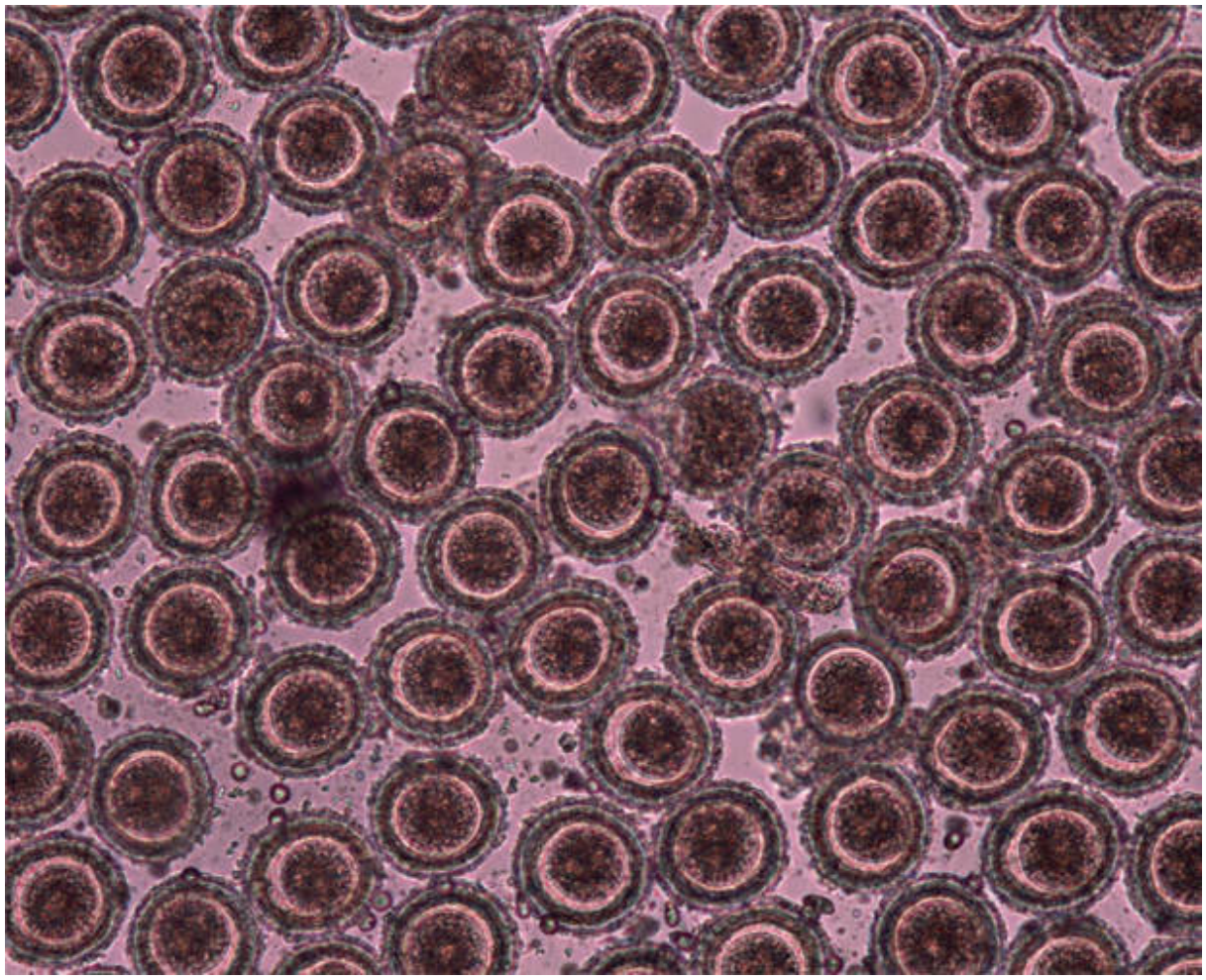
Most anthelmintics are effective against adult forms of *Parascaris* spp., but not against larval stages. Therefore, a dewormed animal can continue to harbour large numbers of parasites. Of the benzimidazoles, the most effective are oxybendazole (90–100 % against adults and 74.2 % against 28-day-old larvae), mebendazole, cambendazole, and fenbendazole. Levamisole and febantel also show good activity. Pyrantel pamoate is widely used given its activity against adult worms and immature forms (ranging between 86 % and 100 % at the recommended dose). Some organophosphates (e.g. dichlorvos and trichlorfon) provide excellent results, but can be quite toxic. The use of macrocyclic lactones guarantees the elimination of both adults and larval stages in the host.

2 How long do embryonated eggs survive in the environment?

The eggs of ascarids are characterised by great resistance to chemical agents, thermal changes, and even mechanical action. This is due to the peculiar structure of their cover, which consists of three layers. The two outermost layers are formed at the expense of the uterine wall. The most superficial layer is albuminous and contains glucosamide, which is stained brown by the bile substances present in the faeces. It can be removed by mechanical action, and its stickiness allows the egg to adhere to any surface and protects against desiccation. The middle layer is composed of three layers that protect the embryo from mechanical forces but not from chemical inhibitors of development. The deepest, vitelline layer contains a lipoid substance that is permeable only to oxygen and fat solvents, and protects the larva from chemical substances such as formaldehyde and copper sulfate. Only substances such as hydrochloric acid, phenol, lysol, acetone, benzene, and carbon tetrachloride can eliminate this layer and destroy the larva. Eggs that are mixed with soil, mud, or slurry are better protected. At low temperatures nonembryonated eggs can retain their developmental capacity for almost 2 months, whereas embryonated eggs are much more resistant. Eggs maintain

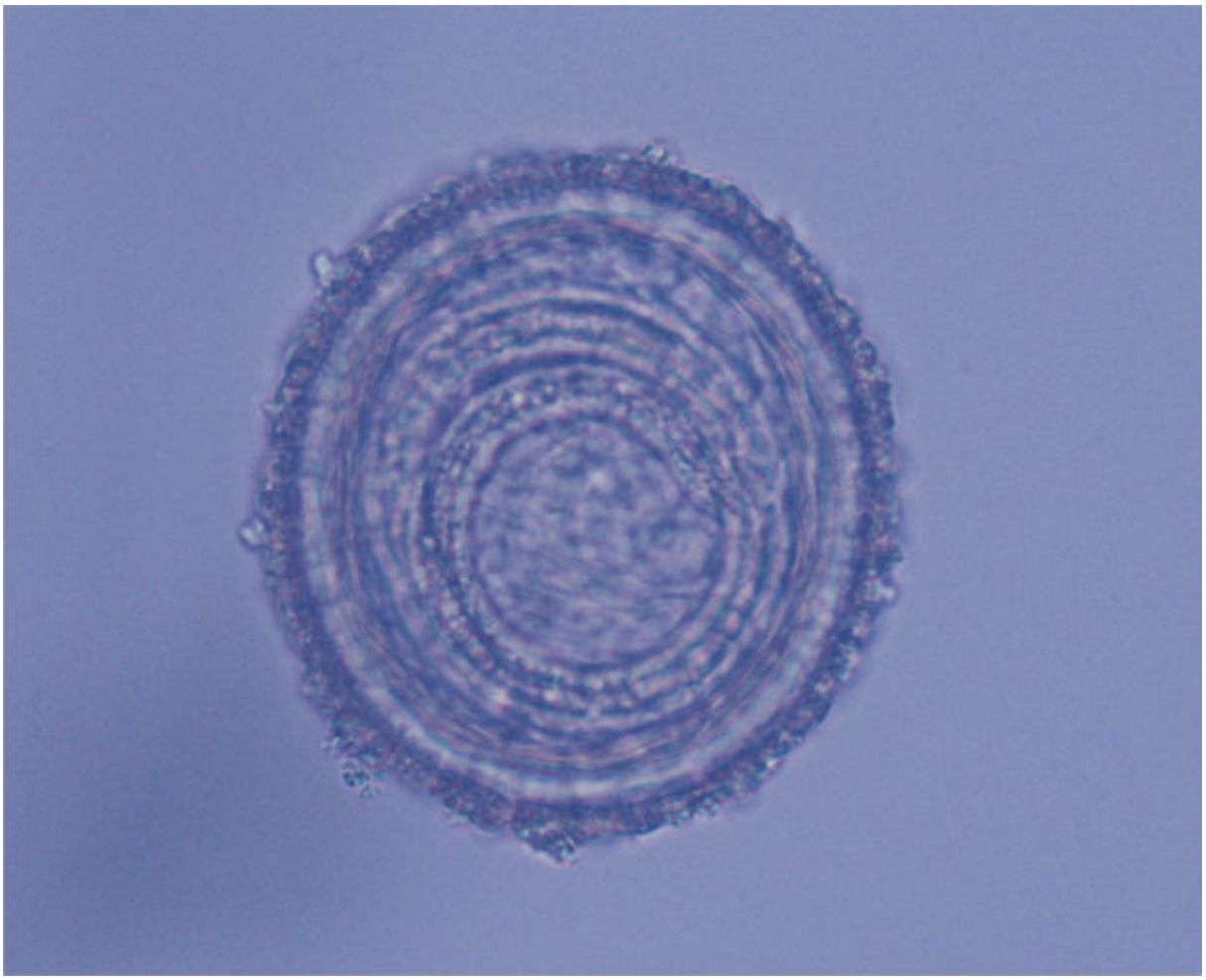
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their infective capacity for more than 3 months in silage, but die after 5 days in fermented manure due to the effects of heat. Some studies have described the presence of infective eggs for more than 2 years in moist earth or in the furrows of irrigated meadows. They are sensitive to both desiccation and direct sunlight.



Nonembryonated eggs. The eggs are irregularly spherical and measure 90–100 μm in diameter. Inside, they contain a single cell surrounded by three covers: a thin inner vitelline membrane; a thick middle cover formed by protein sheets with intercalated layers of chitin; and a third, highly adherent, yellowish-brown external layer with scattered small concavities and protuberances.

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Embryonated egg. The embryo develops at a minimum temperature of 10 °C and a maximum temperature of 38–40 °C (optimum range, 15–35 °C). At 35 °C the infective stage is reached in 9 days, although at lower temperatures this takes 14 days or more. They are highly resistant to environmental conditions, and capable of surviving several years on pastures and in stables.

3 What measures can be taken to reduce environmental contamination and control this parasitosis?

Deworming of affected foals, even if it is adequate and covers all larval stages, is not sufficient and should be complemented with other therapeutic measures. To control parasitic diseases it is important to provide mothers and newborn foals with access to uncontaminated pastures and to minimise contamination of pastures and stables using adequate hygiene measures or treatments. On horse farms it is rare to find pastures that have not been grazed

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over the previous 3–4 years. The only way to provide new pastures is to sow grass in plots allocated for other crops.

Treatment of foals at 8 weeks of age and subsequently every 8 weeks during the first 8–12 months of life, where possible using an anthelmintic with activity against the larval stages, can prevent clinical parascariosis and, in part, its consequences on development. Pregnant mares should be treated 1 month before foaling, and throughout the year it is advisable to perform routine coprological sampling of animals of all ages. Newly acquired horses and foals should undergo faecal analyses and be treated if necessary or, better still, should be dewormed upon entry to the farm even if faecal analyses prove negative, since many worms will have not reached sexual maturity by this stage.

Removal of faeces from stables and pastures at least weekly can prevent eggs from developing and producing infective larvae in these locations, and subsequently spreading via the floor, soil, or vegetation. The temperature of fermenting manure destroys the majority of eggs, but it is important to ensure that this manure is not spread as fertiliser on pastures allocated to horses.

It is very difficult to eliminate *Parascaris* spp. eggs from concrete or cobbled floors, and even more so from earthen floors. The only effective approach is to use high pressure steam to destroy the eggs on the floors of stables where foals and their mothers will be housed.





Collection of faeces from stables and pastures at least weekly can prevent eggs from developing and producing infective larvae in these locations. It is very difficult to eliminate *P. equorum* eggs from cement or cobbled floors, and even more so from earthen floors. Very few disinfectants (except for phenol cresol at 55 °C, or caustic soda) are lethal to larval eggs. High

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pressure steam can be used to destroy the eggs on the floors of stables where foals and their mothers will be housed.

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9 How are *Parascaris* transmitted?

After the introduction of a group of foals into a feedlot for the final fattening phase, the animals become very restless when enclosed with limited freedom of movement, in contrast to their prior situation in large open areas in León, northern Spain. The farmer observes abundant large, white worms on the ground during the first week.

1.How is their presence explained if the animals have not been dewormed?
2.How is the infection transmitted between generations?
3.Can humans be infected?



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1 How is their presence explained if the animals have not been dewormed?

In parascariosis, the immune response of the host to the presence of migratory larvae has important pathological and epidemiological repercussions. The number of eggs in the patent phase is initially very high. However, when the host reaches 6–12 months of age this number decreases and eggs are no longer observed in the faeces, even in the absence of anthelmintic treatment. Clinical signs are observed in young animals (3–9 months of age), but after 6 months marked resistance is established, as evidenced by the spontaneous expulsion of worms and a reduction in the number of migratory larvae, which are unable to reach or settle in the intestine. This immunity appears to be exclusively linked to age and does not depend on previous contact with the parasite.

It has been demonstrated experimentally that the prepatent period in a primary infection ranges from 72 to 115 days, and is prolonged until a competent response is established after 6 months of age, even if there has been prior contact with the parasite. In horses and older foals that are experimentally infected with *Parascaris* spp., intense hepatic and pulmonary lesions develop, but very few worms complete their development in the intestine. This suggests that the liver and lungs are the sites in which the immune response is established.

The stress induced in the animals as a result of their capture, transportation, and transfer to the feedlot for fattening may be the trigger that provokes spontaneous expulsion of these parasites.

2 How is the infection transmitted between generations?

There are no data available on the prevalence of prenatal infections. It is thought that foals are infected by ingestion of embryonated eggs in the environment. Since it is known that foals can shed parasite eggs when they are 10–13 weeks of age, it is evident that infection can occur immediately after birth. It is possible that the teats of mare can become contaminated with eggs from the environment, although this is not likely to be the most

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important route of infection. Although no larvae have been found in colostrum or milk, lactogenic transmission cannot be completely ruled out.

The marked resistance of the eggs and the large numbers (several million) shed in the faeces of young animals result in significant contamination of pastures, which remain infective to subsequent generations for years. The role of adult animals in the transmission of this disease is limited, since they tend not to harbour eggs. In some cases shedding of small numbers of eggs is detected in mares that have recently foaled, probably due to the decrease in immune defences that accompanies the peripartum period. This can result in contamination of pastures and subsequent infection of newborn foals.



Stabled meat horses. The change of habitat and stress associated with the capture of animals destined for human consumption, and their transfer to facilities where they will remain for the duration of the final fattening period, frequently results in the spontaneous expulsion of the parasites they harbour.

3 Can humans be infected?

The biological cycles of most ascarids involve migrations, and the larvae can invade and migrate in a variety of hosts, although the presence of *Parascaris* spp. in humans has not been reported. However, in these cases, larvae are generally destroyed in the lungs and never reach adulthood, except in equids.

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Due to the marked resistance of the eggs, young animals are the main source of infection. However, in primary infections it is common for mares to shed some eggs during the peripartum period. This is due to the evolution of quiescent somatic larvae and their maturation in the intestine, a consequence of the diminished immune system activity associated with foaling and lactation.

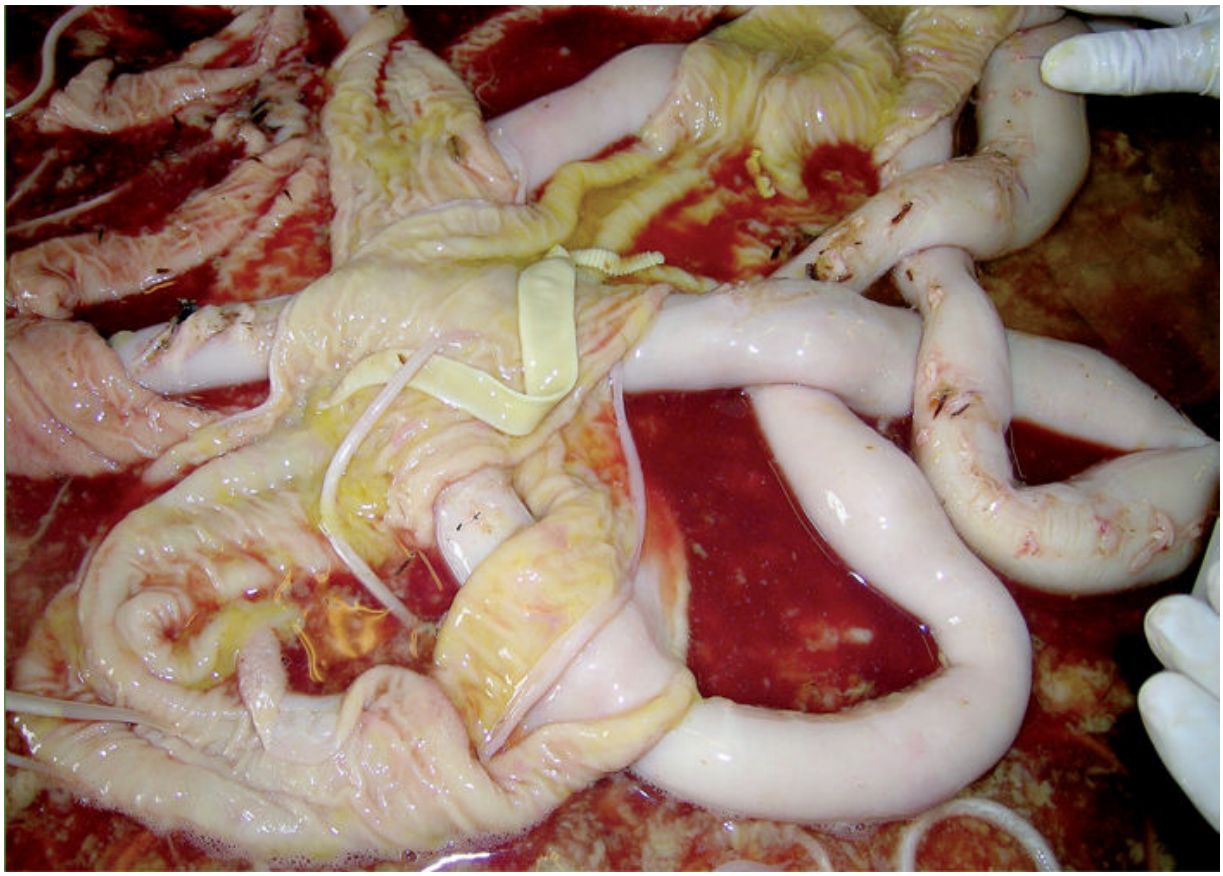
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10 Are any *Parascaris* species resistant to anthelmintics?

On a stud farm, 1-year-old animals are underweight, many have coarse, bristly, and dull hair, and some exhibit pica or malacia. Due to the loss of weight bony protrusions are prominent and the affected animals have a bloated appearance. Workers report that foul-smelling diarrhoea and flatulence are common. Faeces from 10 % of the animals are sent for coprological analysis, which confirms the excretion of large numbers of *Parascaris* eggs. Based on this finding the veterinary surgeon suspects the presence of a large amount of adult worms, and, to avoid the risk of colic caused by the death and lysis of these parasites, administers half of the recommended dose, and the remainder of the dose the following week, in line with the regimen administered over the previous few years. Surprisingly, in contrast to previous occasions, no parasites are observed in the faeces and the clinical signs persist.

1. Is the anthelmintic regimen administered adequate?
2. Can colic be caused by the death and lysis of hundreds of *Parascaris* in the small intestine?
3. Could the dosage used result in selection for resistant populations?
4. Do resistant populations of these parasites exist?

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1 Is the anthelmintic regimen administered adequate?

The administration of drugs at non-recommended doses is inappropriate. The recommended dosage of a drug is based on numerous pharmacological and clinical studies and is designed to achieve the maximum result with the safest amount of product both to eliminate the parasites and maintain the greatest therapeutic margin. The administration of lower than effective doses is one of the main factors that favour the development of populations of parasites resistant to the active ingredients used.

2 Can colic be caused by the death and lysis of hundreds of *Parascaris* in the small intestine?

Massive *Parascaris* infections can lead to intraluminal obstruction in suckling, weaned, and 1-year-old foals. Affected foals tend to have a history of treatment using suboptimal antiparasitic programmes, which give rise to

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high loads of these parasites. Impaction is often preceded by the administration of fast-acting, highly effective anthelmintics, such as piperazine, pyrantel, or organophosphates, or occurs after administration of general anaesthetics or tranquillisers.

The mechanism of action of some anthelmintics causes flaccid paralysis of the parasites, which are carried by the content of the intestinal lumen, and usually emerge in the faeces the following day.
In other cases, the active ingredients cause spastic paralysis, but the final result is the same.



Although certain aspects of the mechanisms of action of these drugs remain unclear, their direct effects on parasites are well known. Most do not cause immediate lysis and death of parasites. Some active ingredients, although equally efficacious, have a slower mechanism of action than others and are therefore preferable in cases of very severe infections.

Some anthelmintic drugs cause flaccid paralysis of parasites, facilitating their expulsion as passive objects as part of normal intestinal transit. In these cases obstruction is uncommon, since the parasites are semi-digested in the faeces.

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Nematodes have only two longitudinal nerve cords that run the entire length of the body within the hypodermis: one dorsal and one ventral, to which the corresponding dorsal and ventral muscle cells are connected. In certain areas, both cords are connected by interneurons. Consequently, activation of dorsal nerve cells induces contraction of the dorsal musculature and relaxation of the ventral musculature, while activation of the ventral nerve cells has the opposite effect. Nematode movement is therefore serpentine, in a vertical plane. Provided that they have sufficient energy to sustain this movement, these constant undulations prevent the nematode from being carried away by the content of the intestine.

Macrocyclic lactones are the group of antiparasitic drugs most commonly used in the treatment/prophylaxis of parasites of equids, and cause pronounced flaccid paralysis of the oesophageal musculature of parasites, preventing feeding. These drugs act slowly, acting on nerve receptors and modifying the activity of glutamate-mediated chlorine channels in the membranes of nematode nerve cells. This results in an increase in chloride ion conduction, altering the membrane potential and inhibiting the electrical activity of the nerve cell. Interactions with the metabolism of gamma-aminobutyric acid (GABA) have also been described, mediated by drug binding to GABA receptors, as occurs following piperazine administration. Consequently, neuronal transmission is interrupted and the parasites die of starvation and progressive paralysis, which facilitates their expulsion.

In other cases, the cause of death is the inhibition of energy metabolism, as induced, for example, by benzimidazoles (which inhibit the activity of the mitochondrial enzyme fumarate reductase), preventing the formation of ATP and leading to the death of worms, which have insufficient energy to sustain metabolism. This metabolic inhibition results in death by starvation. Parasites do not possess energy reserves; since they are surrounded by all the nutrients they require, they possess very small amounts of stored glycogen and lipids. Nutrients are used almost immediately to fuel vital functions; these include migration, the search for other nematodes or for conditions suitable for development, egg laying, and the production of eggs or sperm.

This absence of energy reserves results in death of the worm upon inhibition of the main metabolic pathway (anaerobic glycolysis via dismutation of malic acid in the mitochondria). Mebendazole, a drug belonging to the same group, appears to act by directly inhibiting glucose transport, but results in a similar outcome. Benzimidazoles also directly affect the production of proteins such as tubulin. This protein polymerises in a

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reversible manner, producing microtubules that form part of morphological structures (cytoskeleton) and are implicated in cellular physiology (e.g. mitosis, membrane formation, nutrient transport). The binding of benzimidazoles to tubulin prevents the formation of these microtubules, causing irreversible alterations and ultimately death of the parasites.

Pyrimidines such as pyrantel have a different mechanism of action, mediated by their cholinergic effects on the neuronal ganglia. These drugs induce rapid, spastic contraction of the parasite. In this case it is possible that large numbers of rigid parasites in the intestinal lumen can give rise to a transient disorder. Similar effects are observed after administration of organophosphates, owing to their rapid inhibitory effect on cholinesterase.

In cases in which large numbers of *Parascaris* are suspected, the choice of drug is crucial; administration of parasympatholytic agents that inhibit natural intestinal motility (which facilitates the expulsion of parasites) is never indicated.

3 Could the dosage used result in selection for resistant populations?

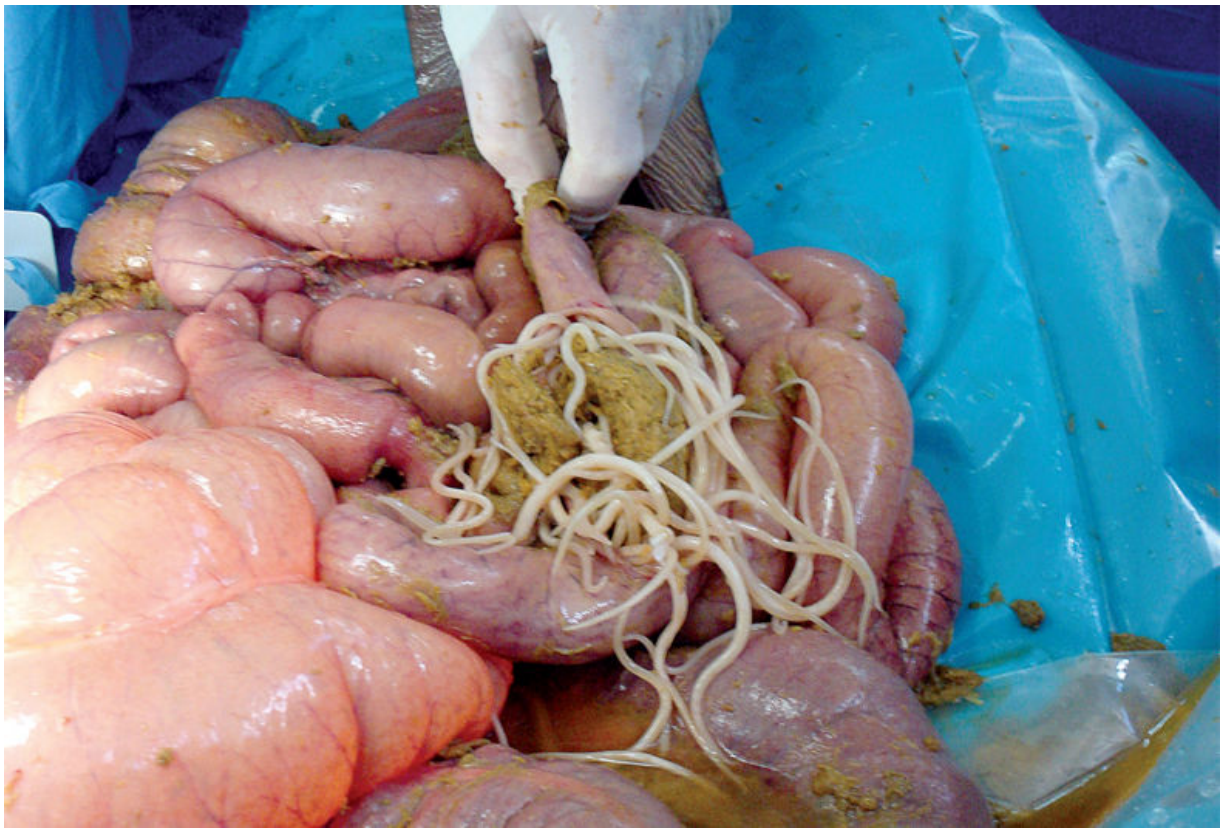
Dominant or recessive alleles that encode metabolic pathways other than those on which drugs act can occur naturally in any parasitic population. The elimination of part of the population by underdosing allows the survival of individuals that carry non-dominant alleles and that serve as progenitors of subsequent generations, resulting in a gradual and progressive increase in the presence of such alleles.

4 Do resistant populations of these parasites exist?

Owing to the ubiquity of *Parascaris* spp. and the possibility of infection in young animals, infections of foals are very frequent. For this reason, control strategies have traditionally been based on the administration of anthelmintics, either rotationally or monthly. As a result of the intensive use of anthelmintics, cases of ivermectin resistance have been reported in many places, and several recent reports indicate the emergence of resistance to pyrantel and benzimidazole salts. To date, *Parascaris* spp. is the only ascarid that has developed anthelminthic resistance. However, little is known about

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the genetic basis of resistance in equid parasites and almost none of the genes involved have been identified. It appears that the eggs of *Parascaris* spp. cannot tolerate the external environment for as long as previously thought, meaning that this phase is less important as a form of refuge than once believed. In any case, these species appear to be capable of developing resistance to all currently available anthelmintics.



Parascaris in an impacted intestine. Massive *Parascaris* infections can lead to intraluminal obstruction in suckling, weaned, and 1-year-old foals. Affected foals tend to have a history of treatment using suboptimal antiparasitic programmes, which give rise to high loads of these parasites. Impaction is often preceded by the administration of highly effective fast-acting anthelmintics, such as piperazine, pyrantel, or organophosphates, or after the administration of general anaesthetics or tranquillisers.

Equine antiparasitic drugs: class, dose, mechanism of action, and effect on the parasite (Hutchens et al., 1999).

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Equine antiparasitic drugs: class, dose, mechanism of action, and effect on the parasite (Hutchens et al., 1999).			
Antiparasitic	Class	Mechanism of action	Effect on parasite
Piperazine	Heterocyclic	Neuromuscular hyperpolarisation	Flaccid paralysis
Mebendazole	Benzimidazole	Inhibition of energy production	Starvation of the parasite and ovicidal action
Fenbendazole	Benzimidazole	Inhibition of energy production	Starvation of the parasite and ovicidal action
Oxfendazole	Benzimidazole	Inhibition of energy production	Starvation of the parasite and ovicidal action
Oxibendazole	Benzimidazole	Inhibition of energy production	Starvation of the parasite and ovicidal action
Levamisole	Imidazothiazole	Neuromuscular hyperpolarisation	Flaccid paralysis
Pyrantel	Pyrimidine	Ganglionic cholinergic effect	Spastic paralysis
Trichlorfon	Organophosphorus	Cholinesterase inhibitor	Spastic paralysis
Dichlorvos	Organophosphorus	Cholinesterase inhibitor	Spastic paralysis
Ivermectin	Macrocyclic lactone	Binds to glutamate chlorine channels and interferes with gamma-aminobutyric acid receptors	Flaccid paralysis and inhibition of feeding
Moxidectin	Macrocyclic lactone	Binds to glutamate chlorine channels and interferes with gamma-aminobutyric acid receptors	Flaccid paralysis and inhibition of feeding

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11 Can cestodes cause the death of their host?

A necropsy is performed on a 12-year-old mare used for riding lessons with a history of spasmodic colic, particularly in colder months. The colic episodes were brief, since the animal responded well to analgesic and anti-inflammatory therapy. The animal died due to impaction of the ileum. Upon opening the intestinal tract, large numbers of anoplocephalids are observed at the end of the ileum and around the ileocaecal valve.

1. What types of lesions do these parasites cause?
2. What is the relationship between their presence and the animal's clinical features?
3. Given the mare was in a riding school and spent most of her time in a box, how did she become infected?

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1 What types of lesions do these parasites cause?

For years, the presence of cestodes in horses' intestines went unnoticed, and was considered of little importance. In rare cases they were associated with digestive processes, but were not suspected as the underlying cause. In recent years things have changed: these parasites are no longer considered mere intestinal commensals, but a direct cause of a series of severe lesions that can give rise to colic.

The only lesions associated with the presence of cestodes are attributed to the tendency of *Anoplocephala perfoliata* to accumulate in compact groups, causing highly localised enteritis that, due to this species' predilection for the ileocaecal valve, can hinder the passage of food. Tissue inflammation at the sites at which the parasites attach, as well as the mechanical obstruction, are directly related to the spasmodic colic and ileal impaction seen in animals parasitised with *A. perfoliata.*

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2 What is the relationship between their presence and the animal's clinical features?

The pathogenic capacity of *A. perfoliata* is linked to its affinity for the ileocaecal valve, although the reason for this affinity is unknown. Considerable irritation occurs at the site of attachment, with inflammation of the deep tissue layers, resulting in focal pseudomembranous enteritis and necrosis in the most severe cases. In these cases, neuronal degeneration observed in Meissner's and Auerbach's plexuses may explain the spasmodic colic pain.

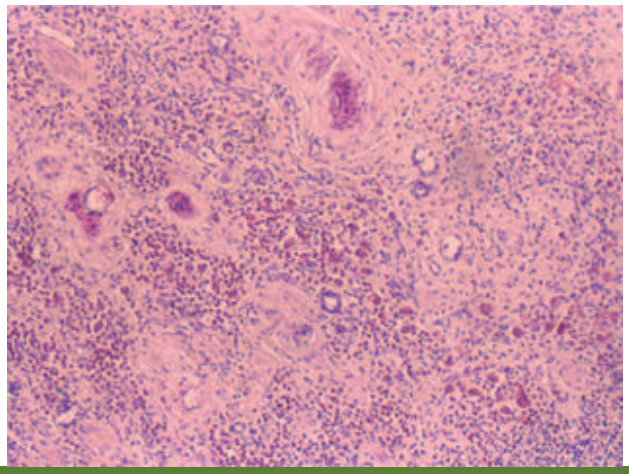
The most severe lesions are not always related to large numbers of parasites, although lesions tend to be more severe in animals harbouring over 100 cestodes. Perhaps the most striking finding is that these lesions are also observed in animals with very low parasitic loads, always when cestodes are grouped in a very small area, often near the ileocaecal valve.

The only report pertaining to other cestode species concerns the presence of a large number of cestodes, identified as *Anoplocephala magna* , in an animal with a ruptured small intestine, although no clear causative relationship was demonstrated. There are no data on the possible pathological repercussions of *Anoplocephala mamillana.*

3 Given the mare was in a riding school and spent most of her time in a box, how did she become infected?

The biological cycle of cestodes is indirect and requires the participation of oribatid mites. These are very common on pastures and act as intermediate hosts. Eggs shed in the faeces of parasitised equids are ingested by these mites, inside which the larval stage of the cestode (cysticercoid) develops after 2 to 4 months, depending on the temperature. The transmission of cestodes to equids occurs via the ingestion of grass contaminated with parasitised mites. It is therefore a common infection in grazing animals, but can also affect any animal that routinely uses the same paths or spends even limited time daily in the same paddock. These animals may be at a similar risk to those that graze permanently if conditions favour the development of cysticercoids in oribatid mites.

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Histologically, the mucosa, submucosa, and lamina propria exhibit infiltration of mononuclear cells and eosinophilic leukocytes, in some cases accompanied by macrophages, neutrophils, and plasma cells.

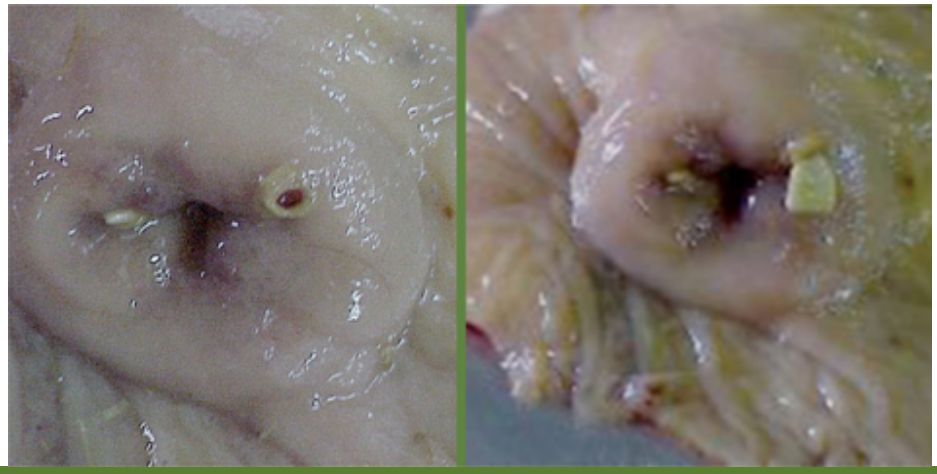
Although the presence of cestodes in horses is related to the ingestion of fresh grass, they can also be transmitted via dry grass, provided the conditions required for the survival of mites are maintained within the packed hay.

Damp, muddy, and acidic soils are the most favourable for the development of the mite and hence for the transmission of cestodes. Temperature does not appear to influence the population of these mites, since they reside under the foliage, 1 or 2 cm from the ground, where thermal variations are minimal. The greatest threat to these mites, which are very sensitive to desiccation, is a lack of moisture. For this reason, cestodosis is very common in northern areas of Spain with high levels of humidity, as well as in other humid habitats (e.g. fluvial plains, marshes) and in more southern areas, where higher temperatures can shorten the larval development phase within the mites.

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In *Anoplocephala perfoliata* infections, in addition to the presence of parasites adhered to the ileocaecal valve, the surrounding areas contain lesions of the mucosa that range from superficial congestion to slightly raised foci of ulceration. At the sites of parasite attachment the mucosa is engrossed, thickened, and ulcerated, and is covered with diphtheritic membranes that can form a granular mass that projects into the intestinal lumen.



Ileocaecal valve with a lesion caused by a single cestode. Of the three species that can be found in equids, *A. perfoliata* is the most pathogenic

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and the only one that has been linked to colic. Currently, the presence of this cestode is considered a risk factor in 81 % of ileal impaction cases and in 22 % of spasmodic colic cases. The risk increases with the intensity of the infection. Thus, the more cestodes harboured by a horse, the more likely it is to develop clinical signs, although similar lesions have also been described in some animals with low parasitic loads.

Owing to the characteristics of its biological cycle, cestodosis in equids has always been closely related to cold and humid periods and northern countries or regions. In temperate climates with dry summers, the risk period for transmission of cestodiasis is cyclical and appears to be regulated by humidity; the mites become infected in spring and by the following autumn the cysticercoids have already developed inside. The largest numbers of parasitised animals are detected during autumn and winter. These animals shed eggs again at the end of winter and beginning of spring, when mite populations increase again. Cestodes appear to have a short average life span; the number of animals parasitised in spring and summer is much lower than during the rest of the year, probably owing to the disappearance of the parasites due to natural death.



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The only way in which horses become infected is by ingesting mites present in grass (never by directly ingesting eggs). This does not imply that stabled animals are free of risk; they can be infected when they consume grass when taken for walks or upon ingestion of mites in stored food.

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12 How is the risk of cestodosis avoided?

In March a veterinary surgeon is asked by an equestrian club to attend to a 4-year-old female pony with signs of colic. The animal had begun to show signs of pain the previous night, upon returning to her stable after spending the day on pasture at the club. All horses at the club had been appropriately vaccinated and dewormed every 2 months with ivermectin. Upon examination the pony shows acute abdominal pain; the pulse rate is 70 ppm and temperature, mucous membranes, capillary refill time, and hydration status are clinically normal. Tympanism is not observed. The animal is too small for an adequate rectal examination to be performed. Anti-inflammatories and analgesics are administered. Over the following 6 days, the colic persists in a latent state. Some intestinal transit is observed, resulting in the expulsion of small amounts of faeces. However, once the animal is forced to eat the colic worsens drastically. A laparotomy reveals the presence of flat parasites 3–5 cm long attached to the wall of the caecum.

1. What information can blood tests or coprology provide?
2. How can the presence of cestodes in dewormed animals be explained? What products are used to eliminate cestodes?
3. When is the right time to administer cestocides?
4. Are there other measures that allow adequate control of this parasitosis?

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1 What information can blood tests or coprology provide?

Detection by laparotomy or postmortem of flat parasites, located mainly in the ileocaecal valve, with four visible anterior suckers, each located above a small flap, allows confirmation of parasitosis by *Anoplocephala perfoliata* . The tendency of this parasite to form groups in small areas of the intestine, almost always around the valve or in the caecum, could explain the clinical picture described (i.e. obstruction of the intestinal transit). Coprology cannot reliably confirm the presence of cestodes, given the poor sensitivity of this method for this purpose. Blood tests can only reveal eosinophilia.

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One active ingredient widely used in the treatment of human and canine cestodosis is praziquantel. Recent studies in horses have shown that this compound has a high efficacy and a wide margin of safety, even when administered at doses 3 to 5 times higher than recommended. No adverse effects have been observed in stallions, pregnant mares, or foals as young as 2 months of age. There is therefore no risk whatsoever associated with its use on any type of farm or in animals of any age.

Diagnosis in vivo is difficult owing to the poor sensitivity of coprological analyses. The low prolificacy of the adults and the low parasitic load, together with the large amount of faecal material excreted by equids, may help explain why standard coprology detects eggs in only 10 % of parasitised animals. Better results have been obtained using certain techniques specifically designed to concentrate the eggs of cestodes, as well as methods for the detection of antibodies in blood, although none of these tests are widely available.

Studies conducted in England have described a direct relationship between parasitic load (and hence the risk of colic) and optical density as determined by ELISA using *A. perfoliata* excretion-secretion antigens. However, this correlation has not been replicated in other countries, such as Canada.

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2 How can the presence of cestodes in dewormed animals be explained? What products are used to eliminate cestodes?

Ivermectin is not effective against cestodes. The treatment of choice is a cestocide. Currently available treatments include pyrantel pamoate (which should be administered at twice the dose recommended for nematodes) and praziquantel, both of which are highly efficacious. If these active ingredients are not included in the deworming programme, the picture may recur if the animals are in an endemic area. Other active ingredients with cestocidal effects that are also used in dogs and cats, such as mebendazole, niclosamide, and dichlorophene, are effective against cestodes of equids at doses higher than those necessary to eliminate roundworms, but are not currently available for equids.

The most effective control measures are applied to the definitive host. Control of the intermediate host mite is totally unviable, owing to its abundance and ubiquity. Treatment is thus based on the periodic elimination of adult worms.

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Sedimentation. Routine methods of detecting eggs in faeces are very unreliable for the diagnosis of cestodosis. Attempts have been made to improve these methods using greater amounts of faeces and by combining sedimentation with flotation using very dense solutions that often deform and contract the eggs, making identification difficult.

The moment of cestocide administration depends on the geographical area and the type of farm: administration a minimum of several times a year is recommended, or at least once in autumn (October) and again at the end of winter (March).

3 When is the right time to administer cestocides?

The period of risk of transmission of cestodosis to horses in temperate climates with dry summer periods lasts from the end of summer to the beginning of spring, with risk peaking in autumn.

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Experimentally, infection has been achieved with *A. perfoliata* in several species of *Galumna* , *Achiperia* , and *Ceratozetes* ; *A. magna* in *Scheloribates* species, *S. laevigatus* , *S. latipes* ; and with *A. mamillana* in *Galumna* and *Allogalumna* species. Experimental infection of the aforementioned mite species does not mean that these species play an epidemiological role in the transmission of equid cestodes, but has demonstrated that the developmental phase in controlled and suitable conditions lasts longer than 2 months.

The timing of cestodicide administration should be chosen based on the environmental conditions that influence the presence of parasitised mites in the grass. In the northernmost areas of the Iberian Peninsula, where incidence is high, it is advisable to deworm animals every 3 months, bearing in mind that during the summer mite populations in the grass decrease, as does the likelihood of transmission. It is advisable to administer a cestocide at the beginning of autumn. This helps to minimise the parasitic load in animals at a time at which the risk of transmission peaks owing to the presence in the grass of large numbers of mites harbouring already developed larvae.

The second treatment should be administered at the end of winter or at the beginning of spring, depending on the geographical area, to avoid contamination of the pastures with cestode eggs. Based on recent data, it appears that spring is the period during which mites are most vulnerable to

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infection. It should be borne in mind that it takes 2 to 4 months for larvae (cysticercoids) to develop inside mites. Mites harbouring cysticercoids are therefore abundant at the end of summer, the beginning of the risk period for equids.

4 Are there other measures that allow adequate control of this parasitosis?

Although not the only useful measure, the removal of faeces from pastures is an effective prophylactic measure to prevent continuation of the biological cycles of most parasites, not only cestodes. Removing faeces ensures the removal of eggs, thereby reducing the number of eggs available for consumption by mites and, in the long term, minimising the risk of ingestion of parasitised mites by horses. Although very effective, this measure is not feasible in communal pastures or in studs that have large numbers of animals at pasture.

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13 How important a disease is strongylosis in equids?

At a congress of specialists in parasitic diseases of equids, the absence of presentations relating to *Strongylus vulgaris* is notable. The organisers note that currently no research group is working on this issue, which explains the absence of presentations, and that this parasite has been eradicated from most countries.

1. What are the pathological characteristics of strongylosis?
2. Do large and small strongyles have similar pathological consequences?
3. What do the data tell us about the eradication of *S. vulgaris* in Spain?

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1 What are the pathological characteristics of strongylosis?

The term strongylosis is usually applied, incorrectly, to infections caused by Strongyloidea. Strictly speaking, the term describes pathologies caused exclusively by species of the genus *Strongylus* , which indisputably includes the most pathogenic species of equids, *S. vulgaris* .

There are about 50 species in the family Strongylidae, all of which have a direct biological cycle. The eggs excreted into the environment in the faeces evolve to infective L3 larvae, which are then ingested by grazing equids. In three species, all of the genus *Strongylus* , these larvae must undertake prolonged intraorgan migrations (6–12 months) to develop and mature; larvae of the remaining species only invade the intestinal mucosa, and therefore have a shorter developmental phase. Given that the pathological consequences are caused by these migrations, and are unrelated to the size of the adult forms, it is preferable to categorise these species not as large or small, but rather as

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migratory *(Strongylus* spp.) or nonmigratory (all other species of the superfamily, which includes large and small species).

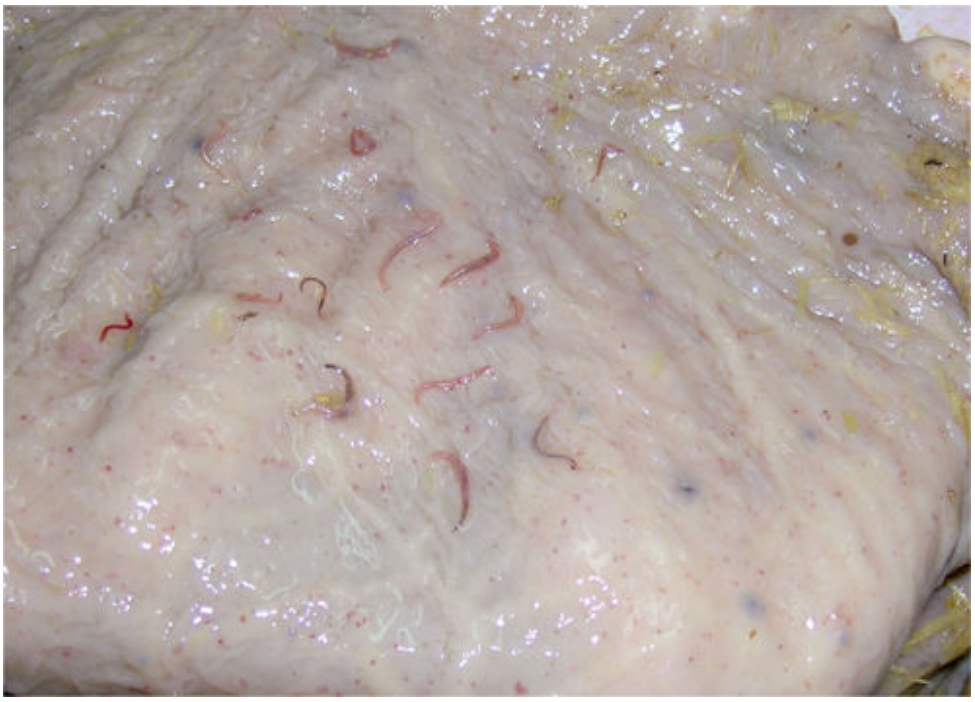
These species cause a parasitic disease of global distribution that is common in temperate climates that favour the exogenous cycle of the nematodes. The disease is characterised by intestinal disorders with colic caused by the presence of larval stages in the mucosa of the large intestine, as well as extraintestinal alterations such as endoarteritis and thromboembolism, caused by the intraorgan migration of *S. vulgaris* larvae. The lesions they cause in the main mesenteric arteries are so prominent that they have been erroneously called "vermiform aneurysms" of horses (weakening of the arterial wall does not occur).

The majority of nonmigratory strongyle species are of less than 1 cm in length, and are known as small strongyles. Most belong to the subfamily Cyathostominae, and are commonly called "cyathostomes". The correct denomination is cyathostomins. The presence of large numbers of larvae of these parasites in the intestinal mucosa can cause a clinical picture known as "larval cyathostomosis". This denomination also includes nonmigratory species of the subfamily Strongylinae (known as large strongyles), such as *Triodontophorus, Craterostomum, Oesophagodontus,* and *Bidentostomum* . These species are omitted from many texts as a consequence of the classification of strongyles as large or small, since only *Strongylus* species are considered "large".

2 Do large and small strongyles have similar pathological consequences?

In the majority of textbooks, "large strongyles" refer to species of the genus *Strongylus* , while "small" species refer to those of the subfamily Cyathostominae.

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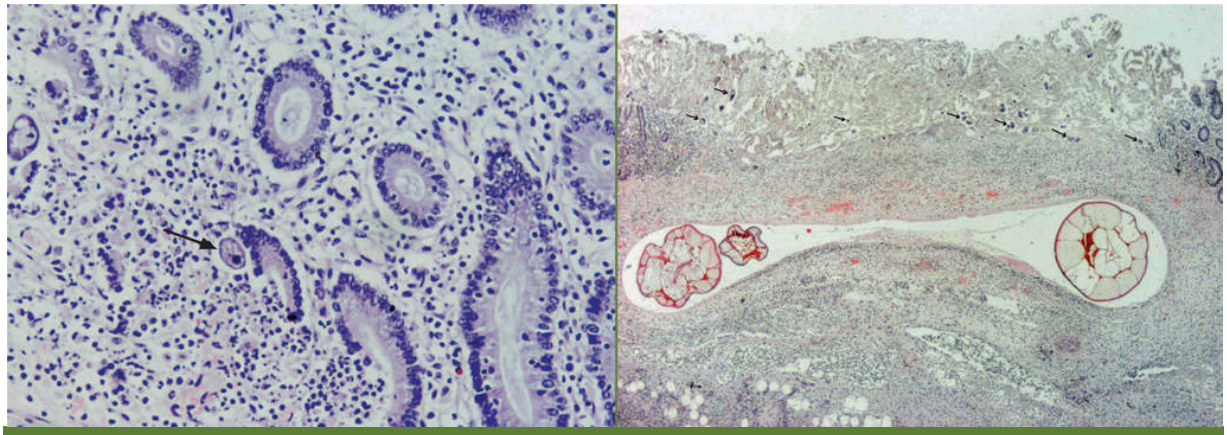
Adult strongyles on the mucosa of the large intestine (caecum). *Strongylus* species (over 2 cm in length) can be clearly differentiated from adult cyathostomins (less than 1 cm in length). In the latter, the recent ingestion of blood is evidenced by their red colour (redworms).



Encysted larvae. The population of developing larvae in the mucosa or submucosa of the caecum and colon is usually larger than the adult population, and numerous reddish-coloured nodules or coiled

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larvae can be observed. These range in size from 1 to 5 mm and can be easily removed by making a small incision.



Inhibited larvae (arrows). Freshly ingested third-stage cyathostomin larvae measure less than 1 mm and invade the mucosa of the large intestine (left image), where they can slow down their development and remain in hypobiosis. They are not visible to the human eye. They should be differentiated from encysted larvae, which are larvae in the fourth stage of development that become enveloped in a cyst as they grow. The image on the right shows a sagittal section of an encysted larva, of which separate portions are evident, and numerous inhibited larvae.

The development of larvicidal active ingredients and the implementation of control programmes specifically designed to control *S. vulgaris* has led in many countries to prevalences so low that this species is no longer considered the most important pathogen of horses.

Given the unchanged prevalence of colic in animals despite the apparent elimination of this pathogen, attention has turned to nonmigratory strongyles, which until now were considered much less pathogenic.

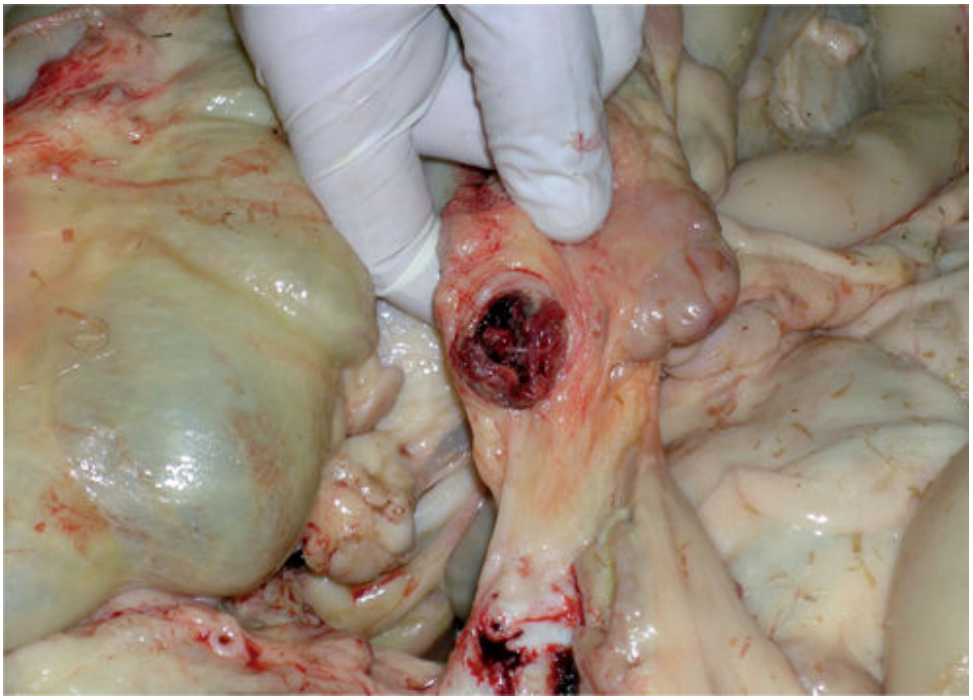
Numerous studies investigating the pathogenic effects of cyathostomins have revealed their potential pathogenic and adaptive capacities. For example, these species can inhibit larval development to their benefit, and include populations resistant to most drugs in use since the late twentieth century. The developmental phase of nonmigratory larvae ranges from 5 to 10 weeks, but they are capable of remaining quiescent in the mucosa for several months, and even years. In certain circumstances the synchronous activation of the development of hundreds or thousands of hypobiotic larvae is possible. This can result in very high mortality when it coincides with the simultaneous emergence of larvae from the mucosa. Horses do not acquire protective

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immunity against these agents, and thus become infected for life upon ingesting contaminated pasture.

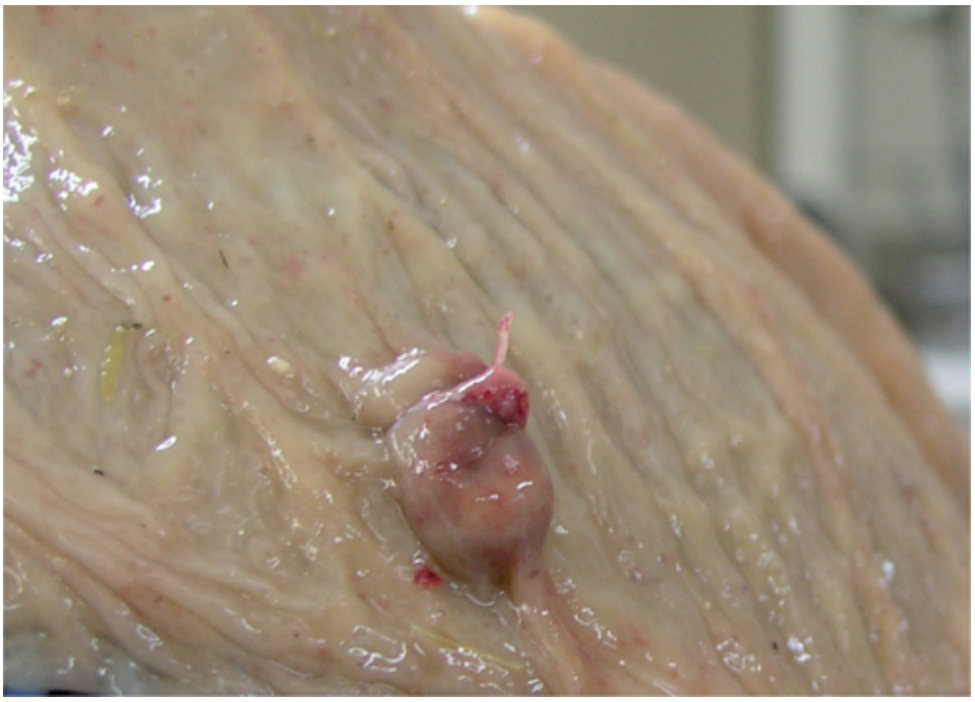
3 What do the data tell us about the eradication of *S. vulgaris* in Spain?

The presence of *S. vulgaris* larvae and associated arterial lesions are detected relatively often in Spain in animals destined for human consumption that are slaughtered in slaughterhouses. These animals rarely receive anthelmintics during the entire fattening period. Of animals harbouring strongyles (60 % of those from the mountainous area of Castile and León), lesions and larvae were detected in the mesenteric arteries, especially larger diameter arteries, in 1 out of 4 animals. Therefore, Spain cannot be considered a country with low risk of *S. vulgaris* infection.



Thrombosis containing larvae. The maturation of *Strongylus vulgaris* larvae occurs over 4 months in the arterial lumen, within thrombi, during which they moult and grow to up to 10 times their original size (over 2 cm).

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Nodule in the caecum caused by *Strongylus vulgaris* . The fifth-stage larvae are carried by the blood flow to the branches of the intestinal arteries, and show a certain predilection for the appendix of the colon, where they remain for 3 to 4 weeks within a nodule. Following gradual degeneration of the submucosa, the larvae migrate towards the intestinal lumen and complete their sexual development.

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14 What are the most pathogenic strongyles?

During collection of material in a slaughterhouse to obtain viscera for practical studies by students at a veterinary faculty, the veterinary surgeon shows the professor images of larger-than-normal nematode clusters in the mucosa of the large intestine, predominantly in the colon. The profile is highly characteristic; 40 or 50 parasites are grouped in a very limited space, and a large ulcer is revealed when they are removed. The veterinary surgeon comments that they are not frequent, since almost all the nematodes found in the viscera are small and red in colour, and are never grouped or associated with lesions of this type.

1. What are these "herds" of *Triodontophorus* ?
2. What type of biological cycle do they have?
3. How do they differ from other large strongyles?

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1 What are these “herds” of *Triodontophorus* ?

These groups of nematodes are the result of the gregarious nature of individuals of the genus *Triodontophorus* . In general, adult strongyles attach themselves to the mucosa of the large intestine and, as a consequence of their diet, become firmly fixed to the intestinal mucosa. Mild traction is usually sufficient to detach these parasites. To avoid coagulation of the vessels of the submucosa while feeding, anticoagulant substances are secreted from the paraoesophageal gland. These substances reach the edge of the capsule via an oesophageal channel or gutter that runs longitudinally from the bottom to the edge of the buccal capsule. The extent of the mucosal lesions depends on the size and structure of the capsule and the amount of blood that can be ingested. Thus, larger parasites can digest pieces of the intestinal mucosa, in some cases reaching the muscle layer and inducing the formation of ulcers. These ulcers can become confluent, resulting in the detachment of large pieces of the mucosa. Smaller ulcers do not extend deeper than the glandular epithelium. The large strongyles *Strongylus vulgaris* and *Strongylus equinus* have teeth at

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the bottom of their well developed buccal capsule and can ingest plugs of mucosa.

Parasites of the genus *Triodontophorus* are large, non-migratory strongyles with a well-developed capsule, and frequently gather in groups (“herds”) and cause large ulcers.

2 What type of biological cycle do they have?

The development of species of the genus *Triodontophorus* and of the other genera of the Strongyloidea superfamily of equine parasites has not been well studied. It is possible that their migrations are limited to the wall of the large intestine, from where they return to the intestinal lumen, as observed for Cyathostominae species and other nonmigratory strongyles.

Moreover, no detailed studies of the parasitic phases of the biological cycle of species of the genera *Cyathostomum, Cylicocyclus, Cylicodontophorus, Cylcostephanus, Gyalocephalus* , or *Poteriostomum* have been conducted. It is accepted that the infective larvae, once released from their sheath, penetrate the intestinal mucosa of the caecum and colon, in some cases reaching the subserosa via the muscular layer of the mucosa (*muscularis mucosae*), leading to the formation of cysts inside which the larvae moult to the L4 stage. After a period of development (1–2 months) in the intestinal wall, they return to the lumen of the colon and the caecum, moulting to the L5 stage and reaching adulthood. The prepatent period is short (about 2 months, although in some cases it may last only 5–6 weeks).

As described for some Trichostrongylidae of ruminants, the larvae of some of these species can inhibit their development for a period of time in the mucosa, thus prolonging the duration of their cycle. This phenomenon has significant repercussions for the course of infections, in terms of epidemiological consequences, clinical manifestations, and susceptibility to anthelmintics.

3 How do they differ from other large strongyles?

The term strongyle comes from the Greek “strongylos”, which means cylindrical/round/compact, and refers to the morphology of these species. Sagittal sectioning reveals a thin body wall and an interior space or

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pseudocoelom containing the tubular digestive system, as well as the tubular reproductive organs, which are usually wound around the digestive tract.

All have an anterior end with a well-developed buccal capsule containing the structures necessary for feeding. Given the central role of these structures in the pathogenic action of these species, we will mention the most important: the buccal orifice, which is surrounded by leaf crowns or papillary structures, with which they attach themselves to the intestinal mucosa; the gland located at the level of the oesophagus that secretes anticoagulant substances, which reach the exterior via a dorsal channel or gutter; and the teeth or cutting plates exhibited by many species, with which they section the mucosa before ingesting it.

Among the large migratory strongyles, three species of the genus *Strongylus* are of particular interest:

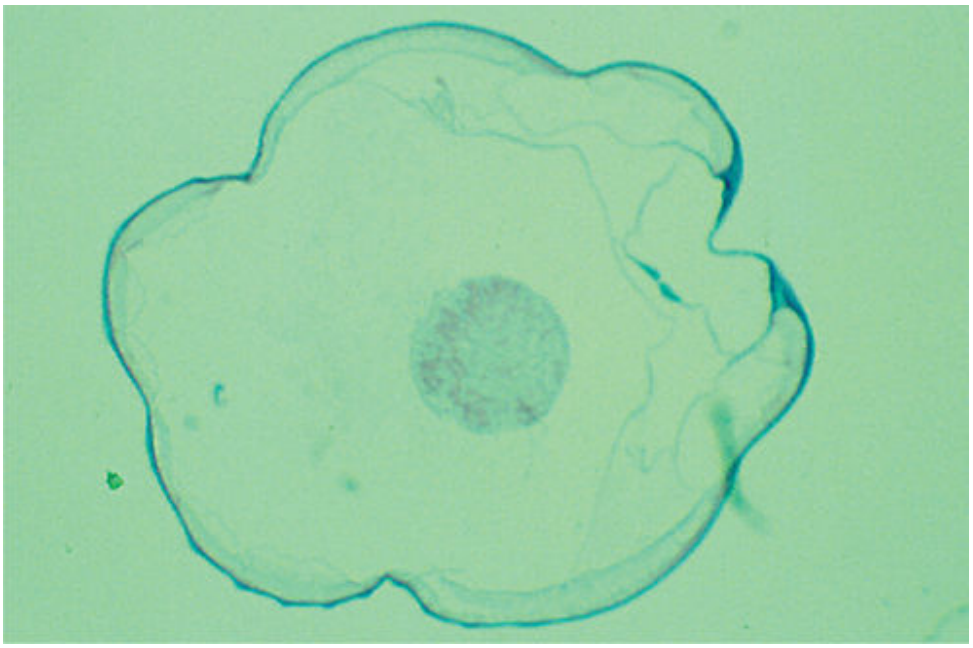
- ***S. vulgaris*** measures 10–20 mm in length and 1.5 mm in diameter and has an irregular, more or less elliptical buccal capsule, at the base of which are two teeth.
- ***S. equinus*** is the largest, measuring 35–40 mm, with a well developed globular buccal capsule containing four teeth.
- ***S. edentatus*** measures 25–30 mm in length. Its asymmetrical buccal capsule lacks teeth.

The powerful oesophagus is club-shaped, and is essential for sucking both mucosa and blood into the buccal capsule.

Nonmigratory strongyles include some large species, but in general they are referred to using the colloquial and erroneous terms “small strongyles” or cyathostomes. These measure less than 1.5 cm, and have a developed buccal capsule, which differs to that of large strongyles by being somewhat shorter and cylindrical. They lack teeth and have radial crowns. The oesophageal channel, if appreciable, does not reach the edge of the capsule. The specific identification of nonmigratory species is of little pathological relevance.

The posterior end exhibits the general characteristics of nematodes: in males the copulatory bursa is highly developed, particularly the dorsal system (double dorsal radius with bifurcated branches); in females the vulva opens at the caudal end, near the anus.

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Sagittal section of a nematode. Nematodes are rounded worms with a stratified body wall and tubular organs located within the internal space or pseudocoelom.

The biological cycle of all species is direct. The exogenous cycle begins when the ellipsoid, segmented eggs (which measure 80 x 40 µm, have a smooth and very thin external cover, and contain 16 blastomeres) are shed in the faeces of parasitised animals. In the environment, if the humidity and temperature are adequate, hatching occurs with the emergence of the first-stage larva. This stage moults twice to produce the infective third-stage larva. In optimal conditions this occurs within about 7 days.

These third-stage larvae are ingested by a new host, and begin the endogenous cycle, which varies between species, as does pathogenicity. Larvae of the genus *Strongylus* have specific requirements to complete their cycle. *S. vulgaris* matures in the cranial mesenteric artery or in the various branches thereof (iliac, middle, lateral, and ventral caecal); *S. edentatus* matures in the liver, which it reaches via the portal system; and *S. equinus* , after an initial moult in the wall of the large intestine, locates itself first in the liver and later in the pancreas.

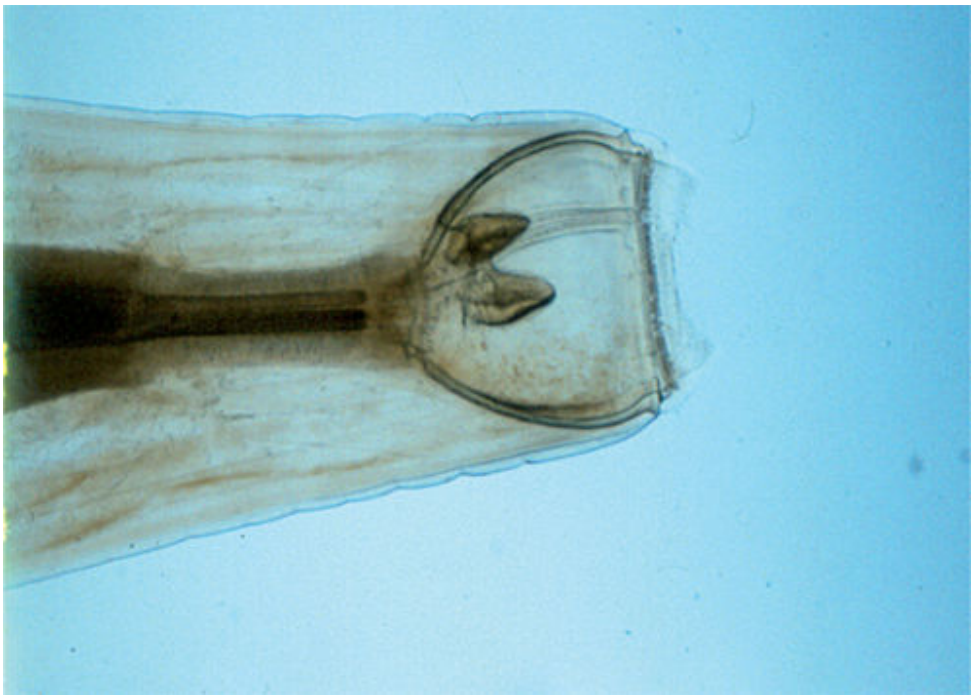
Non-migratory species only invade the wall of the large intestine and can give rise to the appearance of nodules. In this location they can remain in hypobiosis for months, as described for Trichostrongilidae of ruminants.

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Buccal capsule of *Strongylus edentatus.* This is the largest strongyle, and has no teeth in its buccal capsule.

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Buccal capsule of *Strongylus vulgaris* . Compared with the smaller sized nematodes, the buccal capsule of strongyles is highly developed, with anatomical structures that allow the ingestion of a plug of mucosa. This process is mediated by the generation of a vacuum upon contraction of the powerful oesophagus. The presence of a channel that carries substances secreted by a gland near the oesophagus prevents coagulation during sectioning of the mucosa by the two rounded teeth.



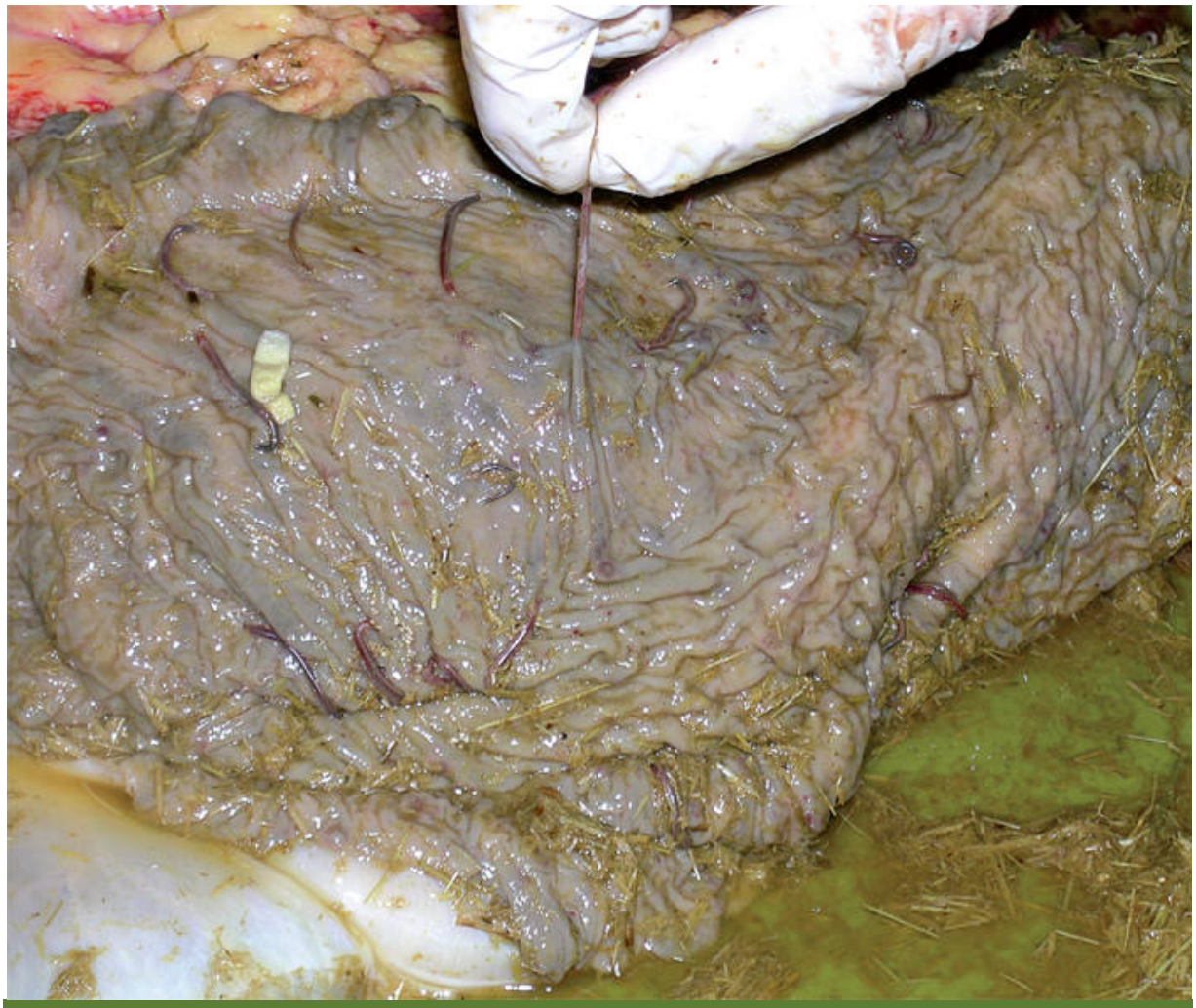
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Buccal capsule of *Strongylus equinus* . This species of strongyle is similar in size to *Strongylus vulgaris* but has three teeth, one bisegmented, at the bottom of its capsule.



Buccal capsule of a cyathostomin. While the buccal capsules of cyathostomin species are less developed, in many species they contain structures that allow them to access the vessels of the submucosa.

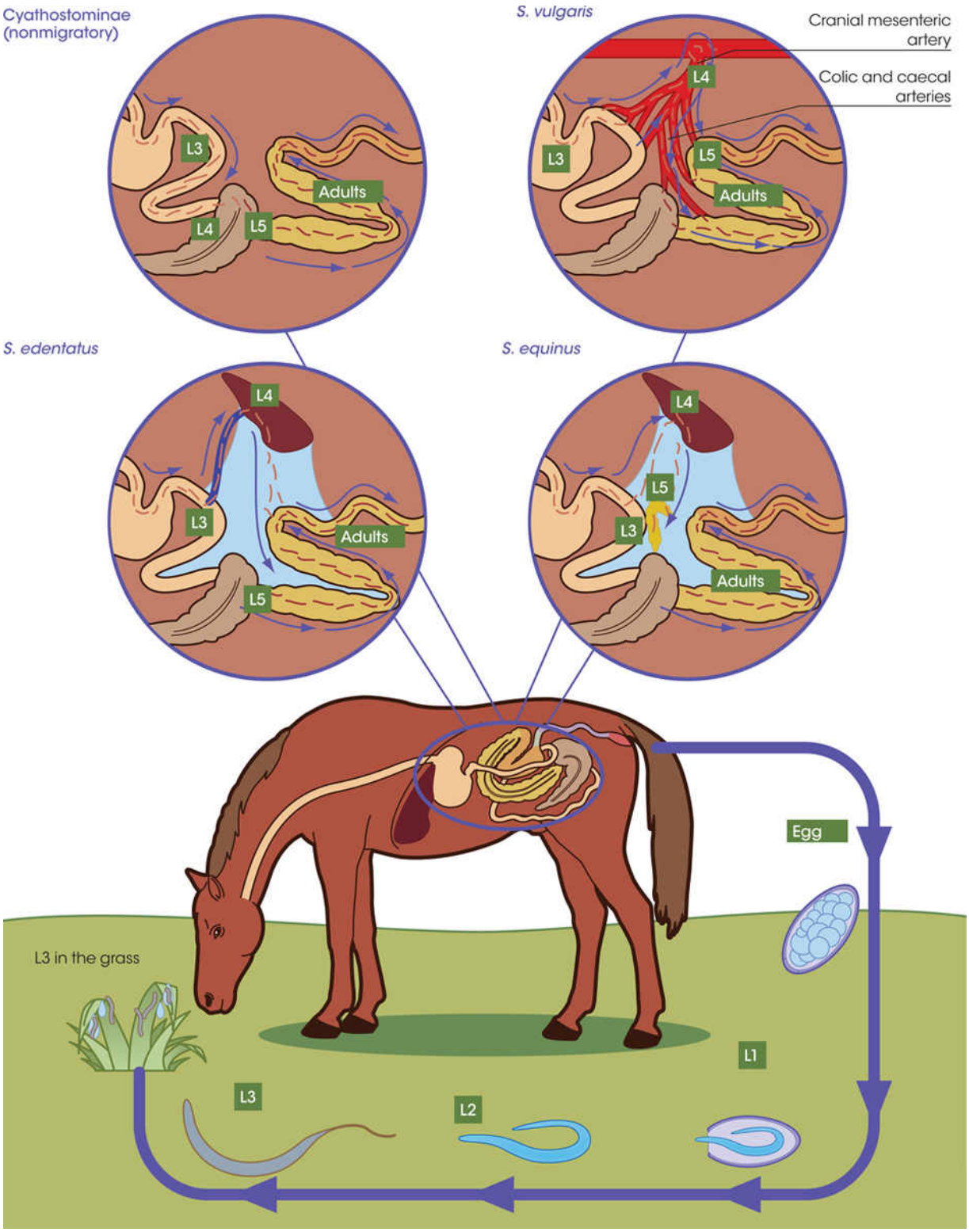
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Strongyle attached to the mucosa. The powerful suction generated by the oesophageal musculature of strongyles is evidenced upon attempting to remove live worms, which are firmly attached to the intestinal wall.

Life cycle of strongyles

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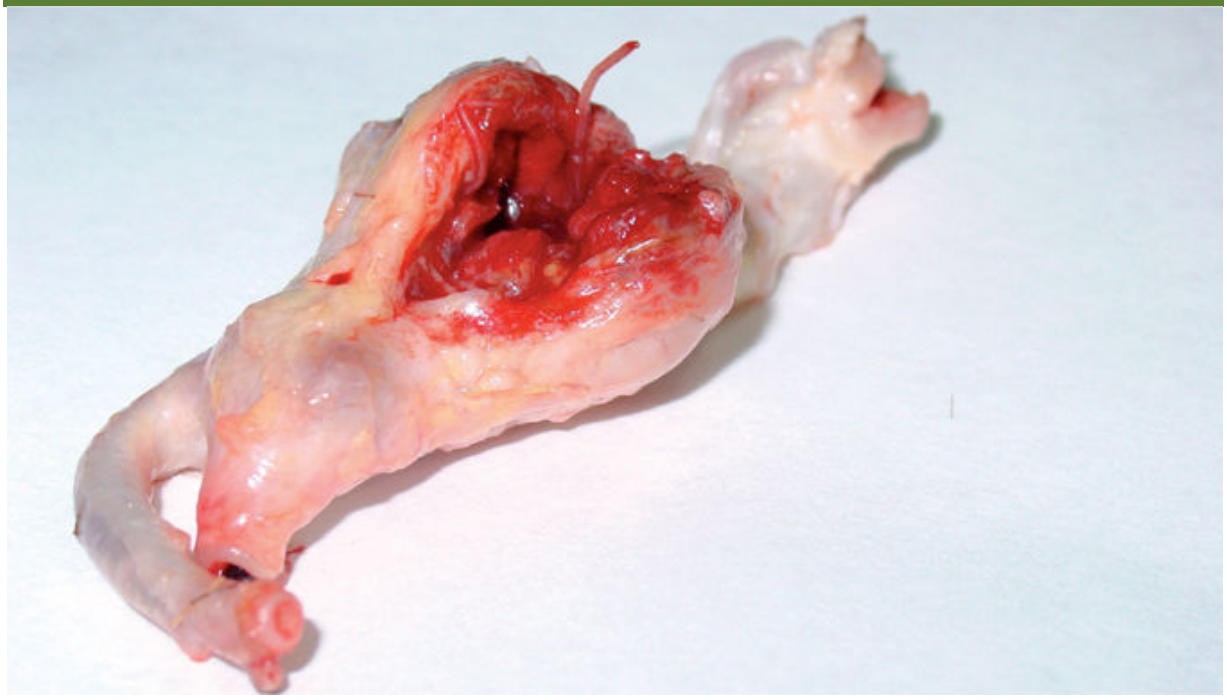


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15 Can *Strongylus vulgaris* larvae be observed in the mesenteric arteries?

A necropsy is performed on the body of a horse that died as a consequence of acute colic. The animal had previously had numerous and varied episodes of colic, and had permanent access to pasture. Opening of the abdominal cavity reveals extensive necrosis in the caecum with rupture of the wall and peritonitis. Upon opening the mesenteric artery, worms can be observed inside.

1.How big are they? What kind of nematode is this?
2.What lesions are observed in the artery?
3.How has the pathological picture observed at necropsy developed?



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1 How big are they? What kind of nematode is this?

The lesion produced is known as verminous thromboendarteritis, caused by fourth- or fifth-stage larvae (L4 or L5) of *Strongylus vulgaris*. These stages can reach up to up to 24 mm in length. Third-stage larvae (L3) ingested by animals while grazing invade the intestinal mucosa and evolve to fourth-stage larvae (L4), which in turn migrate through the arterioles against the direction of blood flow to reach the trunk of the mesenteric artery.

2 What lesions are observed in the artery?

The presence of larvae for long periods of time in the wall of the mesenteric artery and its branches (up to 4 months) causes endoarteritis with thrombus formation and degeneration of the arterial wall. This can give rise to alterations in the elastic and collagen fibres and dilation of the arterial wall, which is markedly thickened as a consequence of the inflammatory response. This alteration is known as an aneurysm (albeit incorrectly, given that no weakening of the vascular wall is observed). Some thrombi can be released. The emboli move through the bloodstream to occlude smaller calibre vessels, causing ischaemia and tissue hypoxia in areas irrigated by these vessels. Generally, these ischaemic areas are irrigated in a compensatory manner by vicarious circulation. However, necrosis and/or rupture of the affected area usually occur when the ischaemic area is very large.

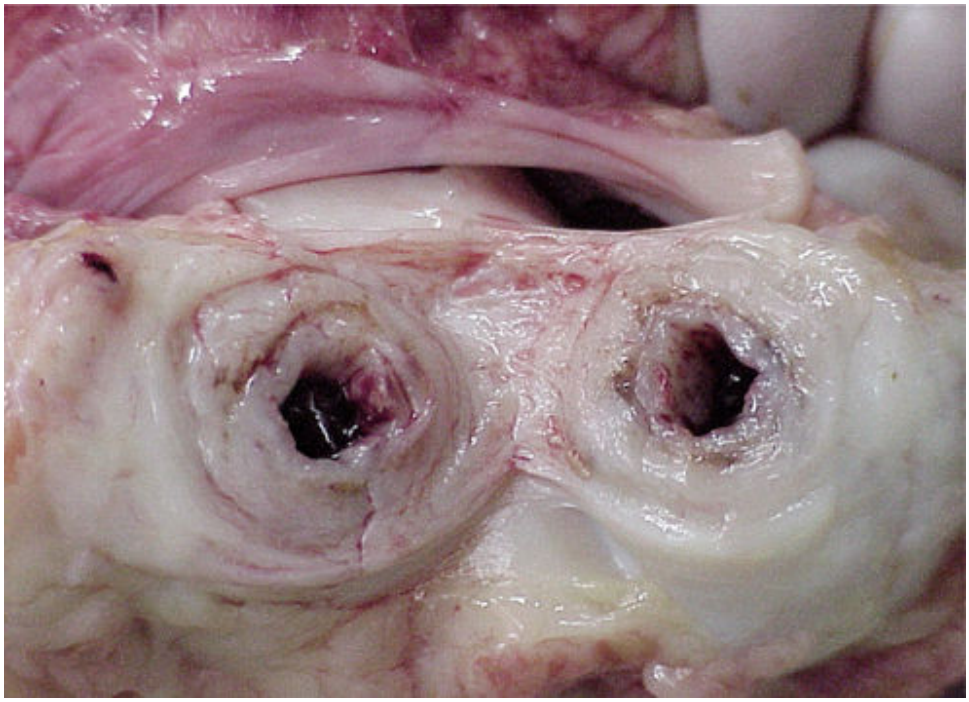
During necropsy, the mesenteric artery is easily found, located near the ileocaecal valve. Palpation reveals an increase in thickness and upon sectioning the presence of lesions and worms is confirmed.

3 How has the pathological picture observed at necropsy developed?

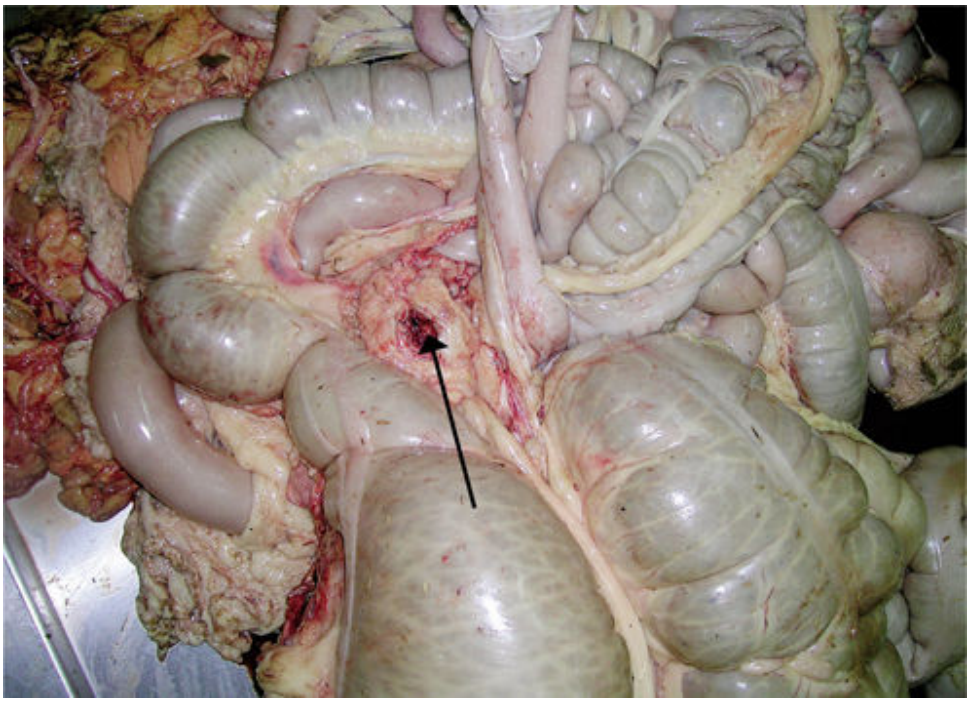
The severity of the alterations caused by the larvae of *S. vulgaris* varies depending on the number of larvae and the period during which they were ingested, the number and size of affected arteries, and the immune status of the infected equids. Acute clinical signs occur when receptive foals consume several hundred or thousands of larvae over several weeks. On the other hand,

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if resistant adult horses ingest fewer larvae over a longer period of time, they will develop chronic signs.



Sectioned artery containing larvae and exhibiting endoarteritis. During the second week of infection, the fourth-stage larvae migrate upstream through the arteries and accumulate in the trunk of the cranial mesenteric artery and its branches. They cause endoarteritis with thrombus formation and the arterial walls become hyperplastic and inflamed.



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The trunk of the mesenteric artery is easy to locate during necropsy; it is an uneven branch of the aorta located near the site of attachment of the mesentery (serosa). It is also easily located in the peritoneal fat; after removal of the intestinal mass, it can be observed situated close to the ileocaecal valve.

The fourth-stage larvae (L4) develop in the intestine and make their way to the arterioles, where they occupy the lumen and ascend to larger calibre vessels. These vessels contain an internal elastic membrane, along which the larvae move, producing lesions without piercing the membrane. These lesions lead to the formation of thin layers of fibrin that increase the size of larger calibre arteries, resulting in a reduction in the rate of migration of the larvae and, ultimately, the formation of a thrombus. If the larvae end up in a small vein or lymphatic vessel, as opposed to an arteriole, they are ultimately carried to the liver or mesenteric lymph nodes, where they die.

In primary infections, the migrating larvae often reach the aorta. However, the high blood pressure in this area hinders their progress, and they are dragged backwards to the anterior mesenteric artery. Intense parasitosis leads to the appearance of thrombi and chronic endothelial inflammation, caused and maintained by larval excretions and secretions, among other factors. Once these thrombi form most larvae cease their migration, thereby increasing the parasitic load in thicker arteries, such as the ileocaecocolic artery. Larger larvae, which are already developed by about 4 months, are carried away by the blood flow and return via the intestinal arteries, where they induce local inflammation in their search for the surrounding intestinal tissue. They become encapsulated for 3 or 4 weeks beneath the intestinal mucosa, giving rise to verminous nodules of 1 to 2 cm. Their emergence from the nodules produces an ulcer, and subsequently a scar. The prepatent period lasts about 6–7 months.



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Left: open nodule in which L5 stages of *Strongylus* and purulent content are observed.
Right: intestinal mucosa with numerous nodules containing L5 stages of *Strongylus vulgaris* ; and verminous thromboarteritis, in which L4 stages of *Strongylus vulgaris* are visible.

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16 Which nematode migrates via the right parietal peritoneum?

In a slaughterhouse in which equids are slaughtered, a veterinary inspector has detected haemorrhages and sinuous tracts in the adipose tissue of the right abdominal wall of some carcasses. These lesions are observed predominantly in carcasses from which livers have been condemned. The livers contain whitish spots, and a squeaking sound is produced when they are cut with a knife. Although infrequent, in some cases the area of the hepatic ligament is completely white, as if coated with wax. Upon cutting through haemorrhagic areas of the abdominal wall, large worms are observed.

1. What kind of migratory worm could this be? How could it be identified?
2. Upon cutting the liver, the knife makes a squeaking sound. Why? Will worms also be observed?
3. What is the cause of the lesions observed in the area of the hepatic ligament?
4. What measures should be taken to prevent carcasses with these types of lesions from entering the market? What action should be taken by those selling to slaughterhouses in order to minimise condemnations?

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1 What kind of migratory worm could this be? How could it be identified?

Lesions of the adipose tissue are caused by the presence of migratory *Strongylus edentatus* larvae. This parasite is easy to identify owing to its size and the absence of teeth in the bottom of its buccal capsule. The larvae, once ingested by the animals at pasture, cross the wall of the digestive tract and reach the liver via the venous circulation.

2 Upon cutting the liver, the knife makes a squeaking sound. Why? Will worms also be observed?

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The presence of this parasite in the liver tissue and its moulting in this location results in the appearance of small areas of focal hepatitis that tend to ultimately calcify, producing a type of lesion known as calicosis. Contact between the knife and these calcium deposits causes the aforementioned squeaking sound.
Worms are not easily seen as it is a chronic lesion.

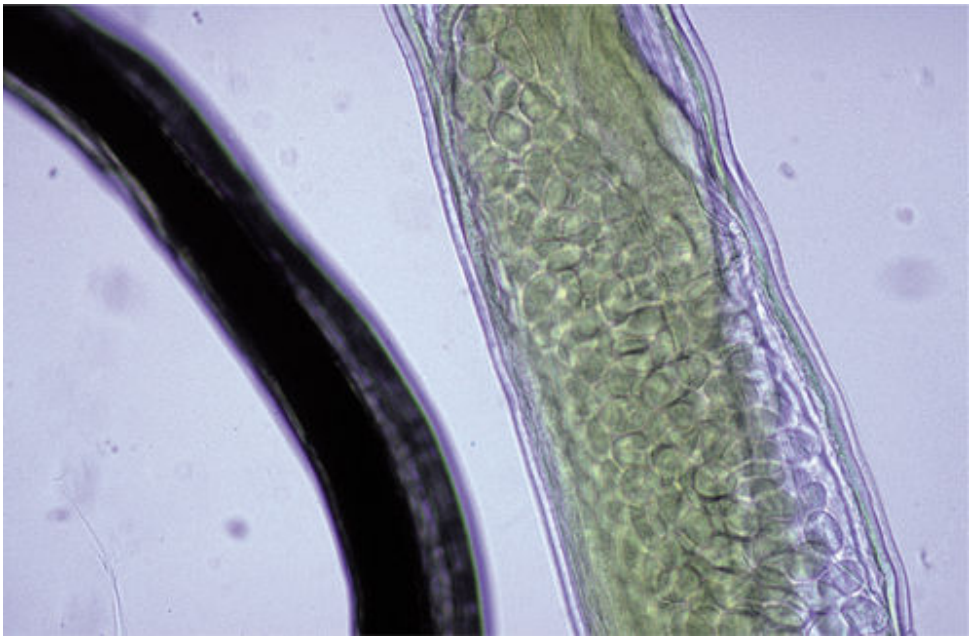
3 What is the cause of the lesions observed in the area of the hepatic ligament?

Larvae of *S. edentatus* migrate through the liver via the hepatic parenchyma, rupture the capillaries and arterioles of the liver, and cause small confluent haemorrhages. These in turn give rise to lesions, the extent and consequences of which are correlated with the number of migrating larvae. The larvae return to the large intestine, leaving the liver via the hepatic ligament, in which collagen is deposited as a result of the passage of many larvae. Some larvae can be detected in the right abdominal wall on their way back to the intestine. Subsequent migration leaves haemorrhagic tracts in the fat of the parietal peritoneum on the right side of the abdomen. Penetration by the larvae of the serosa of the large intestine also produces sinuous tracts and nodules at the sites of entry. Large oedematous nodules (8–12 cm) with a central circular area of necrotic tissue and inflammation of the serosa of variable severity can be observed in the intestinal wall, although these lesions have not been shown experimentally to produce clinical manifestations. The prepatent period is estimated at 10–12 months.

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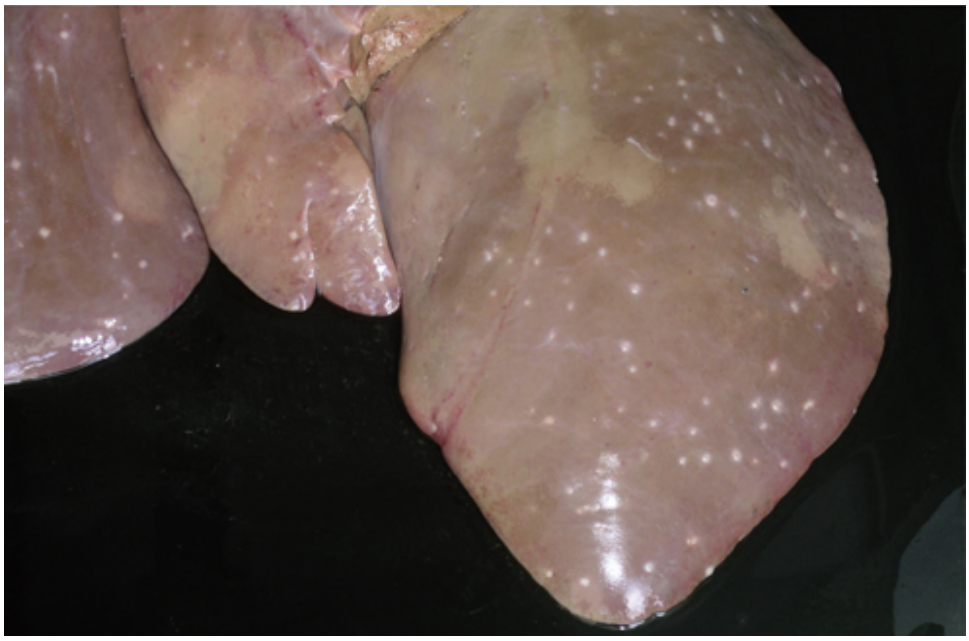


Section of peritoneum containing *Strongylus edentatus* larva. Lesions in the parietal peritoneum contain larvae of several centimetres that lack teeth at the base of the buccal capsule.



Strongylus treated with lactophenol. Observation of the internal structures of nematodes requires the use of specific products (Amman's lactophenol) to clarify the body wall and allow entry of the light of the microscope.

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Liver lesions. Nodules (1 cm), caused by the migration and moulting of *Strongylus edentatus* or *Strongylus equinus* larvae, can be seen in the hepatic parenchyma or beneath Glisson's capsule.

Other larvae found in the liver parenchyma include those of *Strongylus equinus*. It remains unclear whether the formation of nodules in the intestinal walls during the first phase of the biological cycle has pathophysiological consequences in the intestine. The subsequent migration to the liver causes haemorrhages in the hepatic capsule and later in the parenchyma. These haemorrhages are more intense than those caused by *Strongylus edentatus* , and heal leaving extensive scarring. Larval invasion of the pancreas is a fairly common feature and causes atrophy of the secretory cells. The pathophysiological consequences of this atrophy have not been studied. However, it can be deduced that, in addition to a decrease in the secretion of pancreatic juice caused by cellular atrophy, there is also a reduction in the production of cholecystokinin, which influences pancreatic secretions and, indirectly, appetite. Aberrant migrations to the diaphragm, lung, omentum, and even the spermatic cords and testicles give rise to eosinophilic granulomas and morphological and functional alterations of the parasitised organs. When the larvae leave the pancreas, they can be found in the surrounding tissues. It remains unclear how larvae reach the caecum, although it is thought that they may penetrate the wall of the head of the caecum, close to the pancreas. Once in the lumen of the caecum and colon, they reach sexual maturity and mate, and the females begin to lay eggs. The prepatent period is 8–9 months.

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4 What measures should be taken to prevent carcasses with these types of lesions from entering the market? What action should be taken by those selling to slaughterhouses in order to minimise condemnations?

Carcasses with these haemorrhagic tracts will not necessarily be condemned, given that current legislation requires the removal of abdominal fat prior to sale. Buyers are advised to purchase animals that have been dewormed.

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17 How and when are equids infected by strongyle larvae?

Upon the arrival of a new veterinary surgeon at an equine hospital, it is decided to incorporate faecal analyses into the patient arrival protocol to determine the absence or presence of parasites in new arrivals. This will allow the staff to rule out parasitic aetiologies in cases of pathologies with compatible clinical signs. In general, foals from stud farms are most often positive for strongyle eggs.

1.What is the source of strongyle infections?
2.What are the most favourable environmental conditions?
3.Is parasitism influenced by the age or susceptibility state of the animal?

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1 What is the source of strongyle infection?

Newborns are free from strongyle parasites. Generally, this digestive parasitosis is observed in adult horses and foals of a few months of age. Young animals come into contact with the parasite as soon as they start to graze. Within about 2 months they begin to shed eggs. In temperate climates these eggs develop rapidly into infective larvae that can remain viable on pasture for several months. However, due to the characteristics of their life cycle, the excretion of *Strongylus* eggs does not occur until almost 1 year of age, and is not observed earlier.

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2 What are the most favourable environmental conditions?

Temperature and humidity are considered the most influential climatic factors from the epidemiological point of view. The temperature range at which egg development occurs is between 9 ºC and 38 ºC. As the temperature rises, the rates of egg development and larval mortality increase proportionally. Egg development requires temperatures of over 3 ºC, and occurs very slowly at temperatures of less than 10 ºC, requiring almost a week to hatch. From this point, it takes almost 24 days to reach the third stage. Thus, in winter conditions eggs develop in 1 month.

At 20 ºC (i.e. spring and autumn) the eggs hatch within 1 or 2 days and the infective larvae appear within a week. In summer the larvae develop much faster, requiring only 3–4 days at 35 °C. Both the eggs and the L3 stage can tolerate freezing temperatures for some time.

Desiccation is lethal for the undeveloped egg. The resistance of free-living larvae increases with development: the L1 stage cannot withstand desiccation; the L2 stage is a bit more resistant, although it pauses its development to await the presence of water (rain); and the double-sheathed L3 stage is highly resistant.

In hot climates, the exogenous developmental stages show poor survival, but develop faster. The key limiting factors for development and survival appear to be humidity and temperature: high temperatures and low humidity have a sterilising effect on the pasture, while cold and wet weather provides optimal conditions for the survival of the infective stages.

Consequently, transmission patterns will vary from one region to another. In northern Spain conditions are likely adequate for the transmission of these parasites all year round, while in more southern regions transmission depends directly on the level of humidity of the grass.

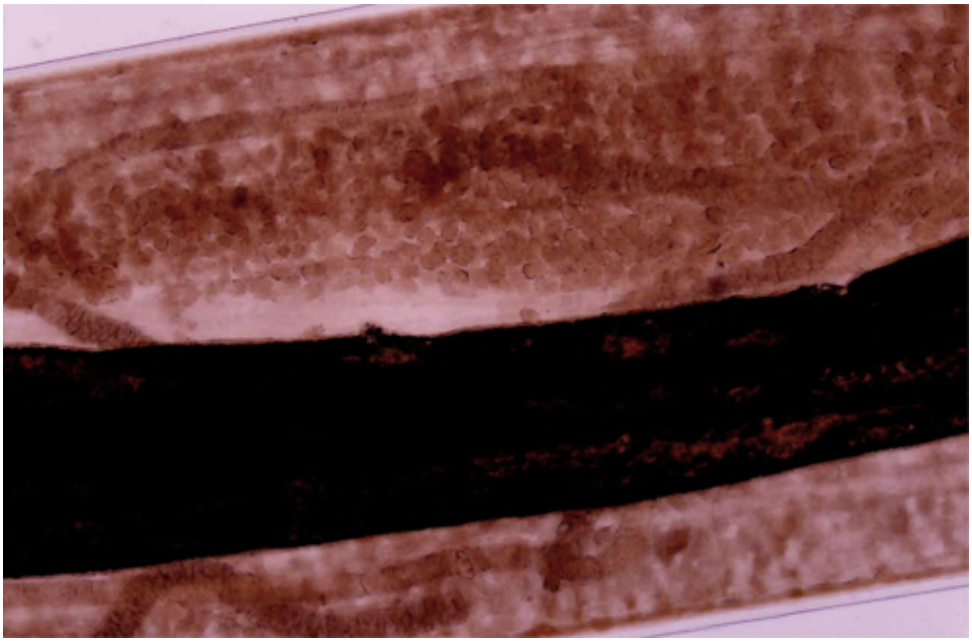
In field studies conducted in central Spain, larvae in the grass were most abundant in autumn and, in particular, in winter (at low temperatures development occurs more slowly but survival is increased), probably because winter conditions are not as extreme as in other countries. The largest numbers of larvae per gram of grass collected were detected between November and February. Throughout the rest of the year lower numbers of larvae were detected, with evident differences between irrigated and non-irrigated pastures during the warmest months.

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The intensity of parasitism is directly related to other factors such as the stocking rate and the rate at which eggs are shed by infected animals, which varies throughout the year. The fecundity of female strongyles can vary: it is estimated that large strongyles can lay up to 5,000 eggs per female per day, while in smaller strongyles this ranges from 100 to 200 eggs per day.

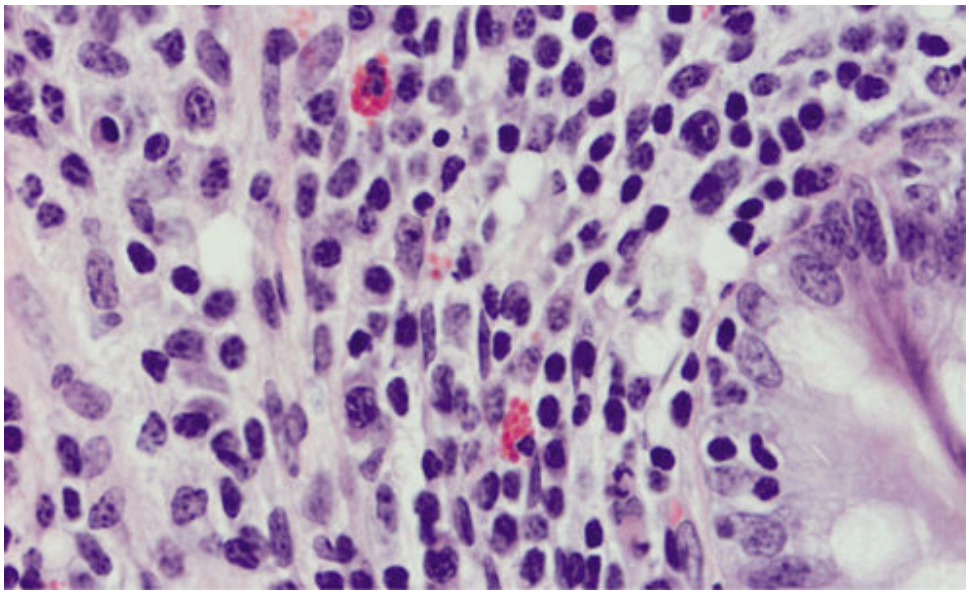


The great climatic differences in some countries prevent the standardisation of anthelmintic control programmes based on a rigid calendar; in some areas the snow can act as an insulating layer, protecting larvae in the soil, while in others the lack of moisture has a sterilising effect on pasture.



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Egg-filled uterus of a female Strongylus. Female migratory worms have a biotic potential far superior to that of nonmigratory species. A female can lay almost 5,000 eggs per day. It is thus common to find the interior of these parasites full of eggs.



Although the immune response is not fully competent, an inflammatory response in the intestinal wall can sometimes induce spontaneous expulsion of the adult parasitic population.

3 Is parasitism influenced by the age or susceptibility of the animal?

There is evidence of acquired immunity: after anthelminthic treatment, the egg reappearance period in younger animals is shorter and more eggs are excreted than in adult animals. This may be due to the presence in these animals of a greater number of inhibited larvae that are unaffected by standard anthelmintics. In general, older animals are more resistant because they shed fewer eggs and the egg reappearance period after deworming is longer. Some studies have suggested the existence of possible nonspecific self-healing phenomena, as described for other nematodes. The administration of infective larvae to animals grazing on contaminated pasture provoked mass elimination of adult and larval parasites, a phenomenon not observed in animals grazing on uncontaminated pasture.

Immunity is acquired, but is incomplete and does not protect adult animals from developing high parasitic loads in favourable environmental conditions. The immunological mechanisms associated with any digestive infection

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generally involve the production of cytokines by the Th2 subpopulation of T cells. These are considered partly responsible for the increases in eosinophils, mast cells, and IgE associated with immediate hypersensitivity response phenomena and immunity to gastrointestinal nematodes. Little is known about the immunological mechanisms associated with strongyle infections in equids. Most data are derived from experimental infections with *Strongylus vulgaris* . These studies have shown that infected animals express higher levels of Th2 cells (which produce cytokines and are partly responsible for the increase in eosinophils). No differences have been described in infections with cyathostomins, although the few studies performed are too limited to allow extrapolation of the results. One study reported an 8-fold increase in IL4 levels in an animal that spontaneously expelled its parasitic population compared with another animal that did not.

The phenomenon of hypobiosis continues to be poorly understood in these species. It appears to be a common process in the northern hemisphere. Several studies have reported a total absence of larval inhibition in tropical regions. This suggests that external conditions may be an important conditioning factor, but does not rule out the possible influence of other factors such as host immunity and population density. The presence of inhibited larvae in the intestinal mucosa is a much more common phenomenon in young animals than in adults. These larvae serve as a reservoir population for future generations of adults and facilitate the phenomenon of gene dilution by acting as a “refuge”.

Larvae inside a drop of water. The eggs evolve inside the faeces. The L1 stage hatches and feeds on bacteria and faecal matter. After two moults, the L3 stage retains the cuticle of the previous larval stage. This protects against adverse environmental conditions but also prevents feeding. This stage lives off its energy reserves. Thus, the higher the temperature, the sooner the reserves become depleted, and the shorter the survival period.

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18 How does thromboembolic colic occur?

A 4-year-old animal exhibits a clinical picture that began with the sudden onset of abdominal pain characteristic of acute spasmodic colic. The animal frequently turns its head towards its right flank and attempts to bite, lick, or nibble the flank and the lower part of the abdomen. A gradual increase in temperature and heart rate (70–80 beats per minute) is observed, together with tachypnoea, pale mucous membranes, and enlarged nostrils. The associated pain and clinical signs subside, and after a few hours the animal returns to normal.
The patient had previously experienced similar colics, after which a change in behaviour was observed: the animal became agitated and resisted saddling and mounting, and would hesitate immediately before jumping, regardless of the height.

1.What tests can be performed to determine the origin of the problem?
2.How has it occurred?
3.What are the less common effects of *Strongylus vulgaris* ?

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1 What tests can be performed to determine the origin of the problem?

This type of presentation could be caused by larvae of *Strongylus vulgaris* , which has been well studied as a potential cause of thromboembolic colic.

Auscultation *reveals* hypermotility of the left colon, and the rectal examination (which can be difficult in large animals) reveals the presence of a pulsatile structure of variable shape and size (chestnut- to orange-sized) in the mesenteric arteries near the left kidney. *Palpation* causes pain, risking detachment of a thrombus. An abdominal *ultrasound* confirms the presence of an orange-sized, pulsatile structure located just in front of the left kidney. *Arteriography* confirms dilation of the affected area and a reduction in blood flow.

When the artery is sclerosed, swelling, but no pulsation, can be detected. The animal shows signs of pain (resistance, erect ears, and kicking) in response to *percussion* with a rubber hammer on the spinous processes of the lumbar and sacral vertebrae and on the longissimus dorsi muscles. To allow

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the animal to adapt and to avoid false responses, it is necessary to begin percussion at the thoracic vertebrae and continue progressively backwards towards the lumbar and sacral vertebrae.

2 How has it occurred?

S. vulgaris it is the most pathogenic parasite of equids. During its migration through arterioles, at the beginning of the infection, it causes a severe reaction that manifests with the appearance of fibrin deposits along its trajectory that narrow the arterial lumen. When the larvae reach the mesenteric artery or its branches, their activity produces lesions in the endothelium and triggers blood coagulation. At the same time, the arterial wall becomes thickened by oedema and inflammation, and the elastic and muscular fibres of the tunica media are replaced by connective tissue, resulting in a local increase in the size of the artery. This can be erroneously considered an aneurysm, but is in fact localised endoarteritis.

This process includes platelet aggregation with continuous production of thromboxane and the formation of a thromboembolism. The larvae are located in the thrombi attached to the arterial walls. Embolisms are common in these parasitic diseases, mainly due to the fact that these thrombi can rupture, resulting in occlusion of the blood vessels. Circulation can be disrupted by occlusion of small and large arterial branches, but this action can be repaired and compensated by collateral circulation. Localised ischaemia with necrosis or hyperaemia with circulatory blockage can be observed, all of which culminate in colic of variable intensity. Usually, collateral circulation can compensate for the decrease in irrigation caused by these emboli, provided there is sufficient anastomosis.

Another pathology associated with the presence of larvae in the mesenteric arteries is nonconstricted infarction, which is a reduction in intestinal blood flow caused by partial intravascular obstruction. The degree of ischaemia varies and usually does not cause clinical signs.

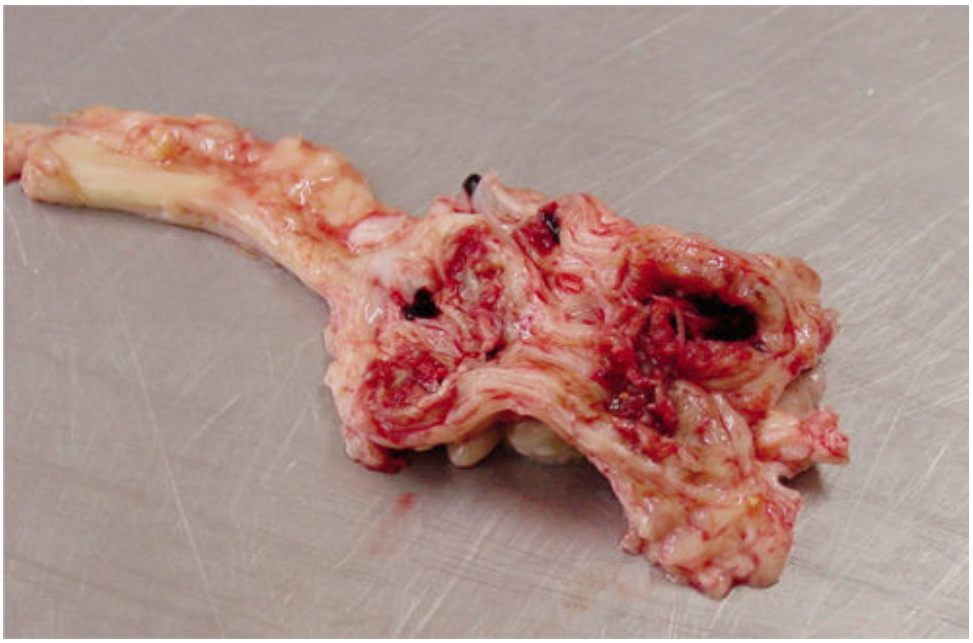
Intestinal flow can decrease by more than 50 % before the intestine is affected. This is due to the ability of this organ to increase oxygen uptake when blood flow is reduced and to divert blood from other areas to the mucosa. This is possible because equids have abundant anastomoses and vascular networks (rete mirabilis) interspersed along the length of an artery.

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These constitute a form of substitute circulation that can compensate for circulatory problems.

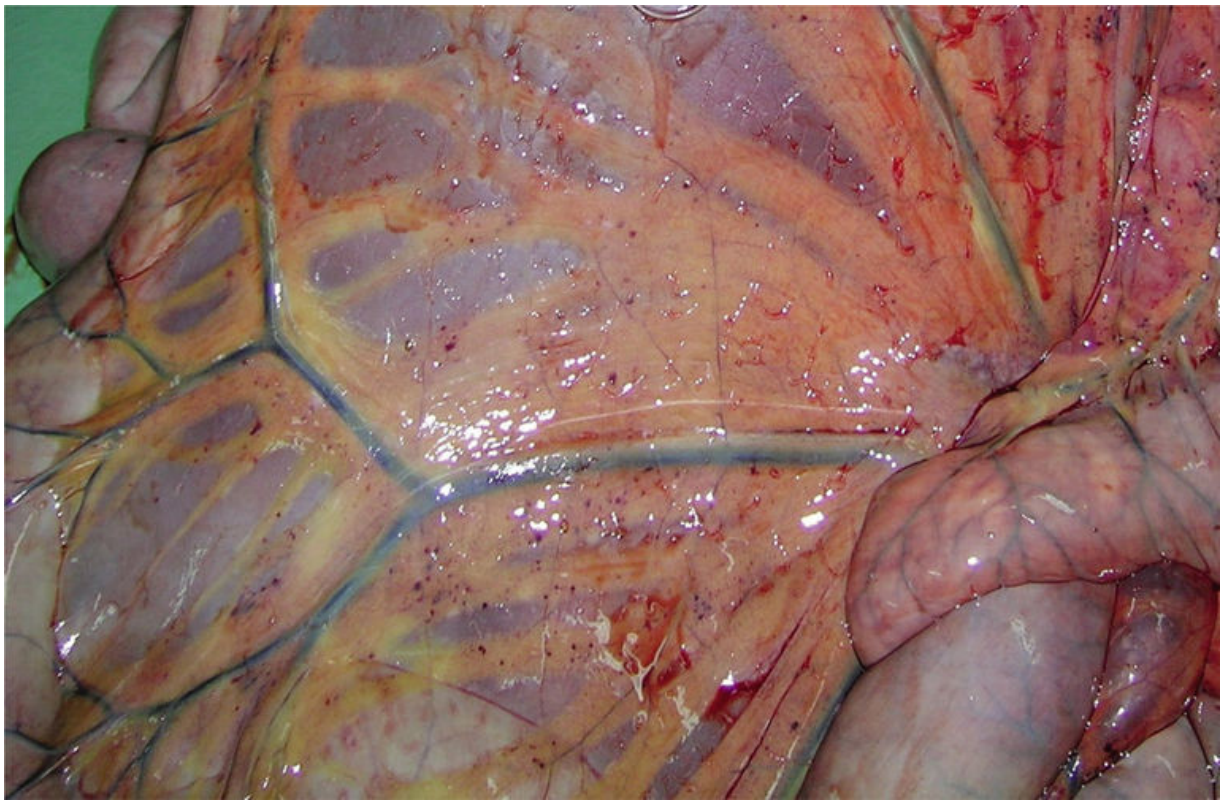


Pain is observed on percussion with a rubber hammer of the spinous processes of the lumbar and sacral vertebrae and the longissimus dorsi muscles. It is recommended to start percussion at the thoracic vertebrae and continue progressively backwards towards the lumbar and sacral areas.



The reduction in intestinal blood flow caused by a partial intravascular obstruction results in ischaemia of variable severity and, in general, does not cause clinical signs.

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Obstruction of some of the ileal arteries at the origin of the ischaemic intestinal lesion is observed at necropsy.

3 What are the less common effects of *Strongylus vulgaris* ?

Massive haemorrhage in the peritoneal cavity due to the rupture of an artery can result in sudden death, although this is very unusual.

Haemorrhagic infarction of a large area can cause paralysis of the intestine, resulting in retention of the intestinal content and abnormal fermentation with consequent gaseous distension. Paralysis involves alterations in the transmission of the peristaltic waves and can result in torsion, invagination, and strangulation of the intestine, caused when the healthy intestinal loops in active movement envelop paralyzed sections of the intestine, which then distorted or invaginate. The paralysis of large sections of the intestine can ultimately result in rupture of the intestine, stomach, or diaphragm due to the powerful backward movement of the peristaltic waves, although this is highly unusual.

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There has been much discussion as to whether the larvae of *S. vulgaris* are the main cause of colic in equids. Supporting this hypothesis, 90 % of colic cases are associated with lesions caused by this strongyle, thromboembolic colic is the main sequela of chronic infections, and the incidence of colic decreases with the application of effective prophylactic programmes against *S. vulgaris* . However, retrospective studies of colic have demonstrated an aetiological role of *S. vulgaris* in thromboembolic infarctions in only a very small percentage of cases.

The decrease in intestinal motility can be caused by reduced blood flow, resulting in intestinal disorders that cause colic. Long-lasting lesions give rise to marked alterations of the arterial system, such that a decrease in blood flow due to causes other than infarction, microthrombi, or the release of vasoactive substances that can predispose affected animals to intermittent colic. Similarly, alterations of the innervation of the intestine caused by vascular lesions, the release of toxins by degrading larvae, and allergic or hypersensitivity reactions can also give rise to colic attributable to *S. vulgaris* .

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19 What is exercise-related lameness of parasitic origin?

Veterinary attention is requested for a 5-year-old horse with exercise-related lameness. The patient is a polo horse that has just arrived from Argentina. Shortly after beginning to trot, the animal stops and shows clear signs of intense pain. On examination, the hindlimb is cold and dry, unlike the rest of the body. After a period of rest the animal recovers and can walk normally. Once other possible aetiologies have been ruled out, a parasitic condition is suspected.

1. What species and developmental stage is compatible with these signs?
2. Why is the limb cold and dry? Could a similar clinical picture affect the forelimb?
3. What is the prognosis for this presentation?
4. What is the simplest and least expensive way to diagnose this disease in a live animal? What would be the appropriate treatment?

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The increased demand for blood supply when the animal performs physical exercise causes tissue ischaemia due to the loss of elasticity in the walls of the iliac arteries in which Strongylus vulgaris

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larvae reside.

1 What species and developmental stage is compatible with these signs?

Exercise-related lameness of parasitic origin is caused by the presence in the iliac arteries of developing *Strongylus vulgaris* larvae. These arteries are colonised in cases of massive and continuous parasitisation, in which both the cranial mesenteric artery and all branches of the caudal aorta are or have been affected. Although only the presence of larvae in the iliac arteries produces clinical signs, other pathologies, such as impaired renal function, are common in affected animals.

The continuous ingestion of infective larvae of *S. vulgaris* over an extended period results in colonisation of the arteries that irrigate the intestine, with a particular predilection for the main trunk of the mesenteric artery. In all these arteries, lesions are produced by the same mechanism. The consequences, in any location, are the result of narrowing of the arterial lumen and the consequent decrease in blood supply. Involvement of the external iliac arteries results in intermittent claudication, due not to the effects of embolisation but to the loss of elasticity of the arterial wall and thrombosis.

The presence of *S. vulgaris* and *Strongylus edentatus* larvae in various organs gives rise to granulomatous formations in the lung, kidney, liver, testes, and the central nevious system (the latter resulting in the greatest pathological repercussions). Certain cases of sudden death have been linked to the presence of larvae in coronary arteries and in the aortic arch. In these cases, examination of the aortic bulb reveals tracks, endarteritis with thrombosis, and fibrous nodules, usually on the cranial curvature and in the region of the coronary sinuses. These lesions cause valvular endocarditis with insufficiency, stenosis or rupture of the coronary arteries, and myocardial infarction. Involvement of the renal arteries can result in renal infarction while migration to the brachiocephalic trunk and the carotid arteries leads to parasitic brain embolism with encephalomalacia and/or encephalomyelitis.

2 Why is the limb cold and dry? Could a similar clinical picture affect the forelimb?

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The cause of the clinical signs is a lack of irrigation of the affected limb due to loss of arterial elasticity and the presence in the arterial lumen of larvae and thrombi, which prevent the increase in circulatory flow required when exercise demands greater muscular oxygenation. The pathogenesis differs from that of colic given the absence of emboli occluding the arterioles. If this were the case, recovery of the animal would be very complicated.

The limb is cold and dry because of the lack of blood supply. It is practically impossible for this type of clinical picture to affect a forelimb. The larvae would have to migrate upstream through the aorta until they colonise the brachial arteries in sufficient numbers to cause lesions compatible with this clinical presentation.

3 What is the prognosis for this presentation?

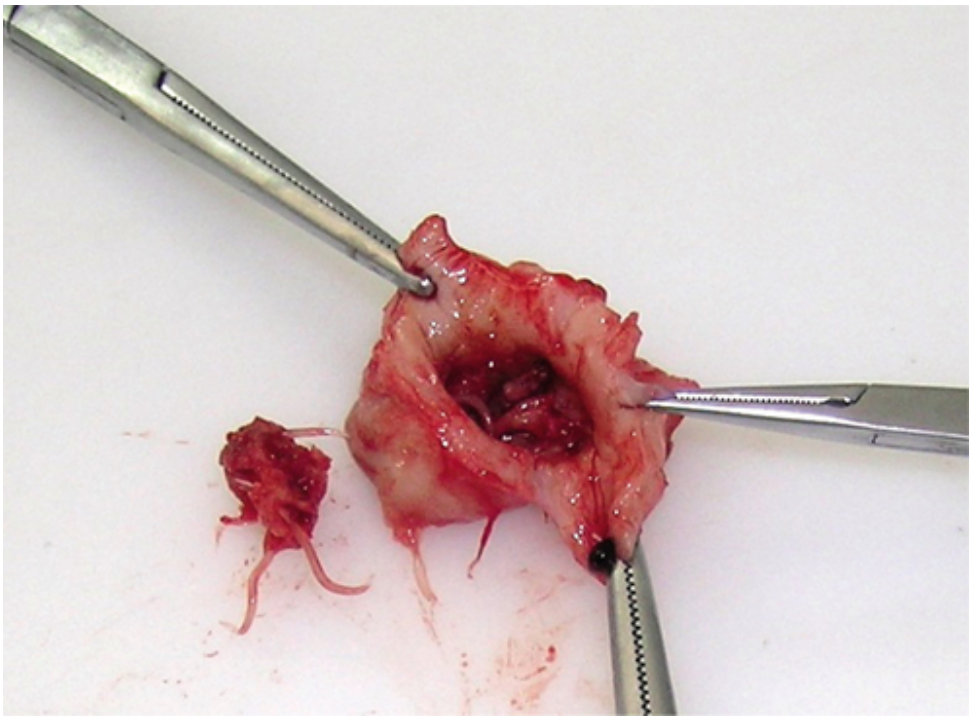
The prognosis is reserved, given that a large number of arteries are already affected when these signs appear.

4 What is the simplest and least expensive way to diagnose this disease in a live animal? What would be the appropriate treatment?

First, all animals belonging to the club of origin of the affected animal should undergo faecal analysis and culture to confirm the presence of *S. vulgaris* . In those that test positive, a strict control programme should be initiated using larvicidal drugs such as macrocyclic lactones every 2–3 months for at least 2 years.

The aim of aortoiliac thrombosis treatment is to eliminate the thrombus and promote collateral circulation to compensate for the lack of elasticity of the iliac artery. Surgical intervention by catheterisation is an alternative, as is slow intravenous infusion of sodium gluconate at a dose of 450 mg/kg (prior administration of prednisolone is recommended to avoid associated systemic reactions). Other protocols include the monthly administration of a larvicidal anthelmintic and phenylbutazone. However, the improvement of treated animals will depend on the progressive development of collateral circulation, which can be promoted with moderate, daily exercise.

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The wall of the artery becomes inflamed and the collagen and elastic fibres degenerate and are replaced by fibrous tissue, which is unable to dilate in response to the increased demand for oxygen.

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20 What pathology do cyathostomins cause?

On a stud farm with an adequate antiparasitic control programme based on the application of appropriate zootechnical and therapeutic measures, a diarrhoea outbreak affects animals of under 5 years of age after deworming in April. The diarrhoea is haemorrhagic and incoercible, and several animals die. Large quantities of small reddish parasites are detected in the faeces.

1.Which parasitic process is compatible with this clinical picture?
2.Are the collected worms larvae or adults?
3.Can cyathostomins cause colic?

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1 Which parasitic process is compatible with this clinical picture?

The cause of the haemorrhage is the massive and simultaneous exit of nonmigratory strongyle larvae from the intestinal wall. The vast majority are species of the subfamily Cyathostominae. The resulting condition is known as larval cyathostominosis.

When the condition is diarrhoeic and the presence of cyathostomin larvae has been verified, treatment should consist of the administration of antidiarrhoeal agents (e.g. codeine phosphate) and hydroelectrolytic support. Oral or nasogastric tube administration is effective. Anti-inflammatories should be administered to treat typhlitis and colitis, avoiding the use of nonsteroidal anti-inflammatory drugs, which can exacerbate protein-losing enteropathy. Repeated administration of low-dose corticosteroids (prednisolone, dexamethasone) is recommended, as this also enhances the effect of anthelmintics by attenuating the immune mechanisms that contribute to arrest of larval development in the mucosa.

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2 Are the collected worms larval or adult forms?

Diarrhoea is caused by the destruction of the intestinal epithelium by thousands of larvae that have remained in a hypobiotic state beneath the mucosa and are simultaneously activated after deworming. There is a hypothesis that in order to maintain the host-parasite equilibrium, the presence of adult worms emits a signal that somehow inactivates the development of the larvae that are ingested daily. The disappearance of the adult forms following administration of a non-larvicidal drug can, in some circumstances, result in simultaneous activation of all inhibited larvae, which then continue their development. Thus, within about 2 weeks the larvae emerge in masse into the intestinal lumen, causing haemorrhagic enteritis with a very poor prognosis.

To avoid this situation, larvicidal products should be included in the antiparasitic control programme.

3 Can cyathostomins cause colic?

The pathogenic effects of adult forms, which are located in the large intestine, colon, and caecum and can form abundant populations, are directly related to their feeding habits. Adult cyathostomins are blood suckers and, unlike larger strongyles, exhibit some degree of selectivity in their choice of location in the large intestine, usually moving to more posterior locations over time (the most mature parasites are generally found in the colon). The worms frequently change location and the wounds continue to bleed due to the persisting effect of anticoagulant substances after detachment of the worm, resulting in small ulcers. These later give rise to round scars that can be observed on the mucosa.

In natural infections, a decrease in ileocaecocolic motility and a reduced response of the colon to food intake have been observed, suggesting slowing movement of the ingested food, which in turn would explain the loss of appetite, malabsorption, and decreased weight gain.

Nonetheless, the pathological effects of cyathostomins continue to be directly related to the number of larvae in the mucosa. Invasion of the mucosa or deeper tissues causes a localised nodular inflammatory reaction with a marked predominance of eosinophils. This gives rise to neutrophilia and eosinophilia in the peripheral circulation that is detectable 3 weeks after

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infection. Many larvae die in the process of invasion, subsequently releasing components that exacerbate the eosinophilia. The epithelial lesions result in the loss of albumin; hypoalbuminaemia can be confirmed 4 to 6 weeks after infection, and a decrease in serum fructosamine 5 weeks after infection.

The presence of larvae in the mucosa appears to induce caecocolic mechnical disorders. Although the precise underlying mechanism remains unknown, inflammatory changes at the level of the mucosa that affect microcirculation and nerve endings appear to play an important role.

The development of cyathostomin larvae in the tissue depends on the parasite. Superficially located larvae do not produce a tissue reaction, and are visible as reddish structures, generally of less than 1 cm, rolled up beneath the mucosa. Larvae located deeper cause the formation of nodules containing a rounded cyst, inside which the larva is located. These cysts are easily removed upon sectioning the mucosa. Lesions of the intestinal mucosa caused by encysted larvae can cause alterations in intestinal motility and favour the development of colic and diarrhoea.

Encysted or non-encysted fourth-stage larvae that are located under the mucosa, but are always visible, should not be confused with inhibited third-stage larvae. The latter, which are not visible to the naked eye, undergo synchronous activation giving rise to the aforementioned clinical signs.



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The synchronous resumption of the development of thousands of hypobiotic larvae leads to massive destruction of the intestinal epithelium within about 2 weeks.



Inflammatory changes in the mucosa that are caused by larvae, not adults, and that affect microcirculation and nerve endings appear to play an important role in the pathogenesis of colic caused by cyathostomins.

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21 Are any equine strongyles resistant to anthelmintics?

A faecal sample from a horse is sent to a veterinary diagnostic laboratory to verify the presence of worms. The animal has just arrived from the USA. Since its arrival the horse's general condition appears poor; its hair is limp and dull and localised perspiration is evident. The mucous membranes are slightly pale, compatible with mild anaemia. Periods of constipation alternate with periods of diarrhoea, or the production of foul-smelling soft or malformed stools. The anamnesis confirms that the animal has spent time at pasture, has had repeated episodes of colic, and has been fed daily with pyrantel-medicated feed. The Spanish veterinary surgeon suspects a possible case of anthelmintic resistance to this product.

1. Is the suspicion of resistance justified?
2. How can it be avoided?
3. How can selection pressure for anthelmintic resistance be avoided?
4. Should special programmes be designed to minimise the development of resistance?

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1 Is the suspicion of resistance justified?

Since the turn of the millennium, meetings of equine nematode specialists have reviewed advances in the biology and control of equine cyathostomins, after an apparent decrease in the prevalence of *Strongylus vulgaris* in several countries. Their aim is to establish a set of guidelines for the standardised diagnosis and treatment of anthelminthic resistance.

The widespread use of anthelminthic control programmes at intervals based on the egg reappearance period has reduced the number of clinical cases, particularly those caused by “large strongyles”. However, these practices constitute a form of selection pressure for anthelminthic resistance, especially in cyathostomins. Since then, reports of resistance and therapeutic failures have increased in number.

Field studies carried out over the last 10 to 12 years have demonstrated a high prevalence of cyathostomin resistance to benzimidazoles, and some studies have reported resistance to pyrantel. Resistance is therefore an

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emerging problem, as also illustrated by macrocyclic lactone resistance in cyathostomins and ivermectin resistance in *Parascaris* spp.

Consequently, the control of equine nematodes should not rely so much on anthelmintics, but rather on practices that reduce environmental contamination, such as grazing methods combined with selective treatments based on faecal egg counts.

2 How can it be avoided?

Following the lessons learned in sheep, it is imperative to ensure that the largest number of parasites possible are not exposed to anthelmintics, and hence not subjected to selection pressure. The populations that are not selected by drug treatment are known as "refugia", and consist of all adult worms and larval stages present in animals that have not been treated (but are included in selective parasite control programmes based on egg removal), all free-living stages (over 90 % of the total population of a parasitic species lives outside the host), and hypobiotic larvae in the intestinal mucosa that have inhibited their metabolism and can therefore withstand most of the active compounds used to treat equids.

3 How can selection pressure for anthelmintic resistance be avoided?

To date, the only clear guidelines to avoid the development of parasite populations with high levels of resistance to all available drugs (as previously described in sheep) recommend reducing prophylactic treatments and applying anthelmintics in a more rational manner, and emphasise careful and routine evaluation of deworming recommendations.

This is best achieved by performing quantitative faecal analyses. The number of eggs per gram (epg) of faeces does not correlate with the worm burden, but rather is an indication of the degree of contamination and the potential for parasitic transmission. Faecal analyses must be carried out in at least 10 % of each of the different groups of horses before deworming. The risk of infection is different in stabled animals versus those that graze permanently. Within a stud, young animals (foals, weanlings, and horses up to 3 years of age) and adults should be considered as independent groups.

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Furthermore, the distribution of parasites in a host population is not homogeneous; a few animals may harbour many parasites, while a majority may have very low parasitic loads. By identifying these “high egg shedders”, treatments can be specifically applied to a small group of animals, avoiding unnecessary treatment of those with low parasitic loads.

It is advisable, once a year, to confirm the efficacy of the product used by performing a second coprological analysis 14 days after deworming. If the control programme is effective, less than 10 % of the samples analysed after deworming should be positive, and/or an 80–90 % reduction in the number of positive samples should be observed after versus before treatment. If the results indicate that the treatment has not been effective, incorrect administration of the product should be ruled out before suspecting anthelmintic resistance of the parasite population.



In response to the emergence of populations of equine parasites that are resistant to anthelmintics, specialist working groups have sought to avoid a repeat of the situation seen in sheep.

Several factors can influence the rate of development of anthelminthic resistance in a worm population.

Owing to the increased availability of antiparasitic drugs, the routine administration of anthelmintics by horse owners, without any veterinary control or prescription, has become a common practice. This has significantly hindered the fight against anthelminthic resistance.

The role of equine veterinary surgeons should not be reduced to knowledge of the most recently released antiparasitic drugs; it should include all the actions inherent to the design and control of an adequate antiparasitic programme. The recent appearance of this problem in equids has

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demonstrated that the techniques described for the detection of anthelminthic resistance in sheep cannot be extrapolated to horses. As such, there is currently no reliable method to compare data at the international level.

4 Should special programmes be designed to minimise the development of resistance?

Control programmes should be adapted to the circumstances of each farm, considering young animals, adults, those permanently at pasture, and stabled animals as independent groups.

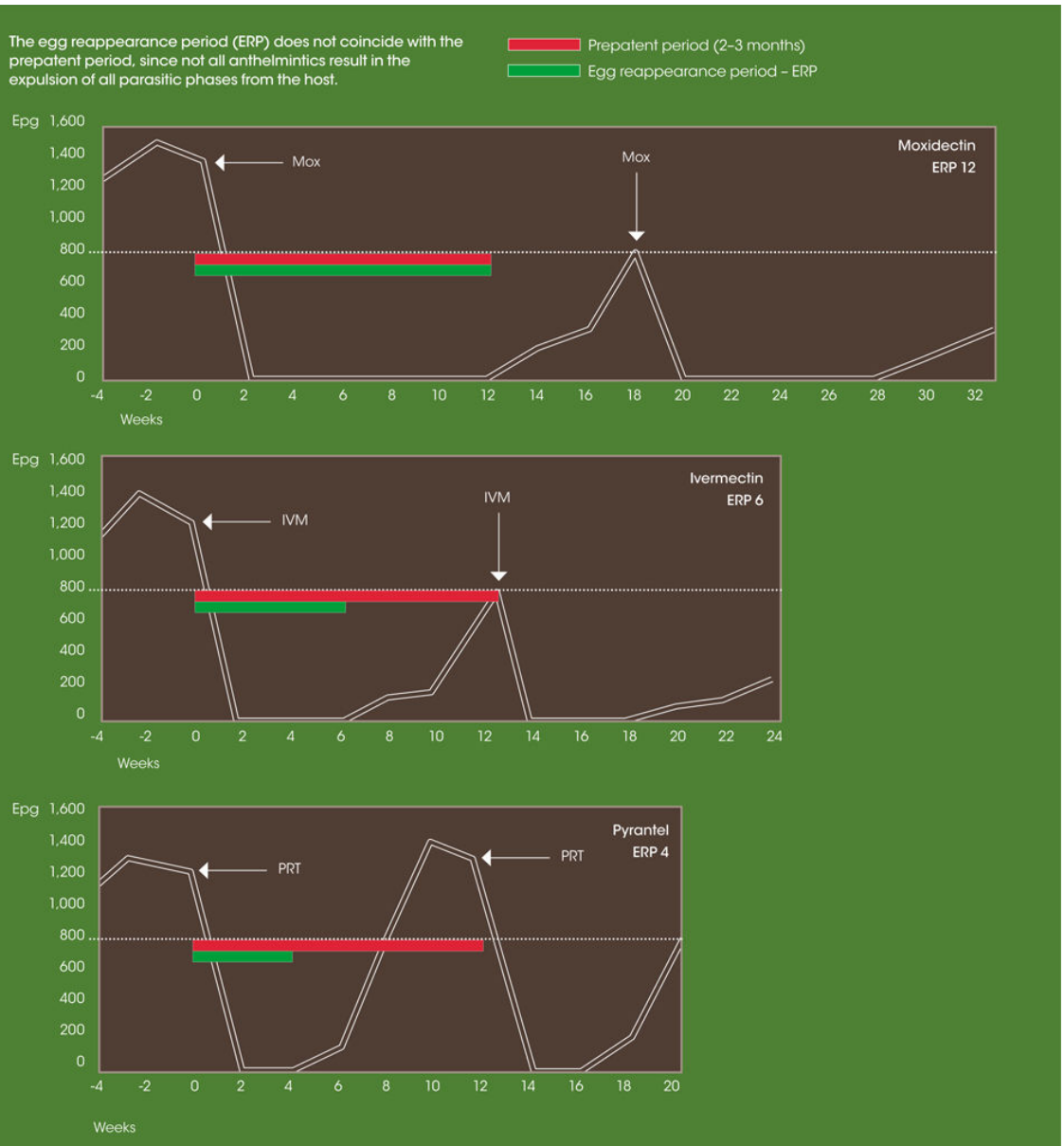
The greater susceptibility of young animals requires the use of programmes tailored to the surrounding environmental conditions to minimise the risk of pasture contamination.

For example, in cases of long egg reappearance periods, drugs such as moxidectin can be administered at the most appropriate times for development up to L3 (no eggs = no larvae). In high-risk situations, up to the end of the prepatent period (no adults = no eggs) repeated moxidectin treatment can be combined with zootechnical measures (e.g. removing faeces, decreasing the stocking rate).

By contrast, in adults and stabled animals it is recommended to identify high egg shedders by counting the number of eggs in the faeces and selectively administering drugs to animals with an epg of over 100–200. Maintaining the hygiene and cleanliness of facilities, including communal pastures and riding rings, with biweekly stool removal, is fundamental to reduce the risk of infection and, therefore, the need for drug treatment. Once per year (usually in the spring), all groups should undergo an efficacy test, whereby coprological analyses are repeated 14 days after the administration of the antiparasitic product. In foals, it is advisable to rotate the products used, and to annually evaluate the efficacy of each.

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The egg reappearance period (ERP) does not coincide with the prepatent period, since not all anthelmintics result in the expulsion of all parasitic phases from the host.

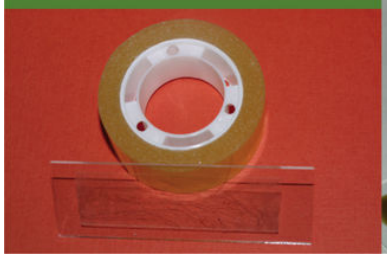


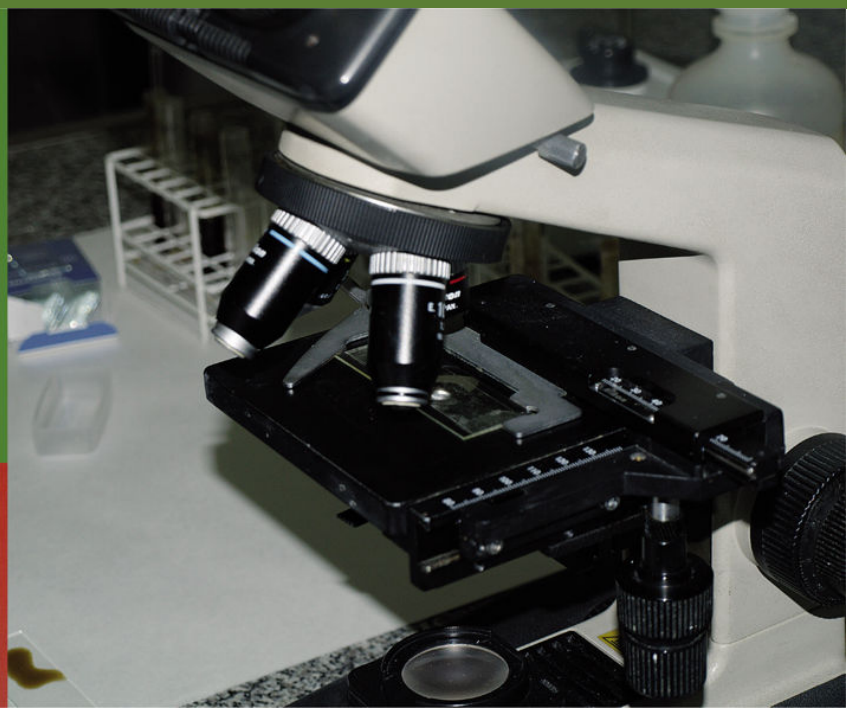
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22 How is an oxyurosis diagnosed?

In the month of June, the owner of a farm contacts the veterinary surgeon and reports that his animals are restless and frequently rub their hindquarters against walls and trees. They had not been dewormed in spring; coprological analyses revealed less than 100 strongyle epg, and the veterinary surgeon did not consider treatment appropriate. The owner reports that he has seen white parasites in the faeces. Based on this information, the presence of pinworms is suspected

1.What is the basis for the suspicion of pinworms?
2.Why were they not detected in the routine coprological analysis?
3.How is oxyurosis adequately diagnosed?





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1 What is the basis for the suspicion of pinworms?

Oxyurids of horses generally refers exclusively to *Oxyuris equi* , a white nematode with a highly acuminated tail that resembles an awl. This species is colloquially known as the pinworm. Females are visible to the naked eye, and reach almost 10 cm in length, while males do not usually exceed 1 cm.

They live in the large intestine, colon, and rectum. In females, the vagina is located in the anterior third to facilitate oviposition, which is performed externally, in the perineal region around the anus of the host. The movements of the females, the presence of eggs, and the sticky substance that the female excretes to help the eggs adhere provoke intense pruritus in the host, which attempts to alleviate the pruritus by rubbing against nearby surfaces.

Only large numbers of *O. equi* larvae can cause inflammation of the intestinal mucosa, sometimes resulting in small ulcers caused by ingestion of the mucosa using the buccal capsule. Small numbers of larvae tend not to cause intestinal alterations, nor do adults exert pathogenic effects in the intestinal lumen. The sole alteration caused by this species is the result of the laying of eggs in the perianal region, and in particular the release of substances from the adult females, the bodies of which burst open during egg laying. These remains cause irritation, as do the eggs, which are surrounded by a sticky, yellowish substance.

The migratory movements of the females and the irritation of the skin of the perineum caused by the presence of eggs and the substances released from the adults induce behaviours aimed at mitigating the anal pruritus (i.e. rubbing against hard surfaces), ultimately leading to erosion of the hair on the surface of the tail-mass.

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The strong pruritic effect causes affected animals to rub the tail-mass against walls, columns, posts, etc. This results in inflammation and erosion of the skin of that area, and the hair becomes broken and tangled. The inflammation, wounds, and erosions on the skin can become complicated by bacterial infections. Pruritus causes restlessness and prevents animals from carrying out their normal activities. This can alter the general condition of the animal, and result in weight loss and abnormal behaviours.

2 Why were they not detected in the routine coprological analysis?

Since oviposition does not occur in the intestine, coprological analyses will only detect this species if the sample analysed is taken from the initial portion of a stool, in which eggs may be found. The best method is direct observation of the large numbers of usually yellowish eggs around the perineum, and

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collection of the eggs with adhesive tape. Direct observation of the adhesive strip under the microscope allows identification of the characteristic oxyurid eggs.

Another species of equine oxyurid is *Probstmayria vivipara* , which has totally different biological characteristics. The female produces live larvae that continue their development inside the host, and thus can reach high parasitic loads.

3 How is oxyurosis adequately diagnosed?

The main manifestation of the disease caused by *O. equi* is pruritus affecting the perianal region and surrounding areas. This causes affected horses to constantly scratch and rub these regions, giving rise to the skin and hair lesions described above.

The general condition of the animals, which lose weight, are restless, and become anorexic, may deteriorate. In some cases changes in behaviour are observed, and animals become irritable and unruly.

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Like other nematodes of the same group, Oxyuris females do not deposit the eggs in the intestine but migrate to the perineum where they frequently die during oviposition, covering the eggs with the their remains, which adhere to this area as a yellowish substance. The eggs can be collected using adhesive tape, which is then observed directly under a microscope.

The appearance of the tail is characteristic: hair is lost from the dorsal region of the tail-mass, on which broken bristles are observed (a condition known as “rat tail”). In the vicinity of the anus are small, waxy-looking masses adhered to the skin, as well as secretions from the anal opening. Parasites are frequently shed in the faeces. These parasites can be differentiated from the medium-sized *Parascaris* based on their length and the characteristic tapered tail of the female.

The lesions caused by the animals’ response to the pruritus and the presence of a sticky substance in the perianal region suggest oxyurosis, which is confirmed by direct microscopic observation of eggs collected from the area. Sometimes oxyurid eggs are detected in coprological analyses by chance if they have been dragged out by the faecal mass during its passage through the anal sphincter. It is essential to examine the skin of the perianal region for

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the presence of eggs. If the sticky clusters in which the eggs are embedded are not visible, adhesive strips can be applied to the skin near the anus and then directly adhered to a slide, which is then examined at 100x under a microscope.

The differential diagnosis should include entities that cause pruritus, including pediculosis, which is also caused by parasites.

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23 Can more than one pinworm species affect equids?

During routine reproductive control tasks, it is common to observe whitish parasites on the gloves when removing faecal material for examination. These are clear signs of inadequate parasite control in the animal in question.

1. What parasite is this?
2. How many oxyurid species can affect equids?
3. What are the morphological characteristics of pinworms?
4. What type of biological cycle do they have?

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1 What parasite is this?

Parasites large enough to be visible to the naked eye in the circumstances described above are typically roundworms. Among the nematodes that parasitise equids, the largest are those of the genus *Parascaris* , which are observed in large quantities in the faeces after administration of an anthelmintic. However, if many worms are present and are forced to compete for space and nutrients, they tend to be smaller, and often many perish. These dead worms, which are often partially digested, are expelled in the faeces.

Pinworms are smaller, white worms, characterised morphologically by a highly acuminated tail. Females of the species *Oxyuris equi* are visible to the naked eye, measuring almost 10 cm, while males do not usually exceed 1 cm.

Other, smaller macroscopic nematodes are also found in faeces. These include oxyurids of the genus *Probstmayria* (similar to *Oxyuris* but less than 2–3 cm in length) and cyathostomin larvae, which are less than 1 cm and have a characteristic reddish-brown colour caused by the ingestion of blood.

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2 How many oxyurid species can affect equids?

Several species of nematodes belonging to the family Oxyuridae parasitise equids. The most important of these is *O. equi* , which causes oxyurosis, characterised by pruritus in the tail and perineal regions, a loss of appetite, decreased production, and alterations in the animal's general condition. In addition to this species, another species with similar morphological characteristics (*Oxyuris poculum* ; von Linstow, 1904), but with a longer spicule (440 μm) was found in a horse in former Ceylon (Sri Lanka).

The second cosmopolitan species is *Probstmayria vivipara* , a small nematode that can be found in large quantities in the large intestine and does not appear to be pathogenic to its hosts.

3 What are the morphological characteristics of pinworms?

The family Oxyuridae includes small- and medium-sized nematodes that have three poorly developed lips, and an oesophagus with a well-defined posterior bulb. The males have several large papillae around the cloaca and in the females, which are generally larger than the males, the vulva is located at the anterior end of the body and the caudal tip is acuminated to form a long, pointed tail.

The male *O. equi* is 0.9–1.2 cm long (females measure 4–15 cm) and is whitish or greyish-white. It has a truncated posterior end and caudal wings supported by costiform papillae. The single, pin-shaped spicule measures 120–165 μm. The body of the female is whip-like: the anterior portion of the body is thickened and curved, while the posterior region tapers to form a long tail. The vulva is located in the anterior portion of the body, about 7–8 mm from the mouth. The females are oviparous.

The eggs are asymmetric, with an operculum at one of the poles. They measure 85–95 μm × 40–45 μm and are embryonated at the moment of laying.

Within the same family, the subfamily Probstmayriinae includes the species *P. vivipara* , a small oxyurid (males and females measure approximately 2 mm and 29 mm, respectively). The paired spicules of this species are of unequal length, measuring 58–67 mm. The vulva of the female is located in

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the middle portion of the body. The viviparous female has two uterine branches, which contain eggs and free embryos.

4 What type of biological cycle do they have?

Both species have a direct life cycle. When the two branches of the uterus of the female *O. equi* are full with eggs, the female crawls to the anus to deposit the eggs in the perianal region. During oviposition, the body of the female frequently bursts due to the high internal pressure, and the eggs are disseminated throughout the tail mass and perineal region, where they adhere to the yellowish waxy substance derived from the inside of the female. Within 1 to 1.5 days, the first larval stage begins to develop inside the egg. The infective larvae form within 3–5 days.

The eggs containing the infective larvae do not hatch, and are shed when the animal scratches. They fall to the ground and contaminate feed and drinking water, and are transmitted to other hosts, in which the larvae are released in the small intestine. The third larval stage enters the crypts of the mucosa of the ventral colon and caecum, and transforms into the fourth larval stage 3 to 10 days later.

These larvae have an obvious buccal capsule and feed on the intestinal mucosa. It has not been established whether this stage is hematophagous, although this is suspected based on their occasional reddish-brown colour. Within about 2 months (approximately 50 days), the larvae transform to the fifth larval stage and complete their development in the intestinal lumen, beginning egg-laying about 5 months after infection.

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Oxyuris females are much larger than the males. Once filled with eggs, the females migrate through the rectum to lay their eggs in the perineum. During oviposition, the females often burst due to the high internal pressure, releasing a sticky substance that facilitates adhesion of the eggs to the perineal area.

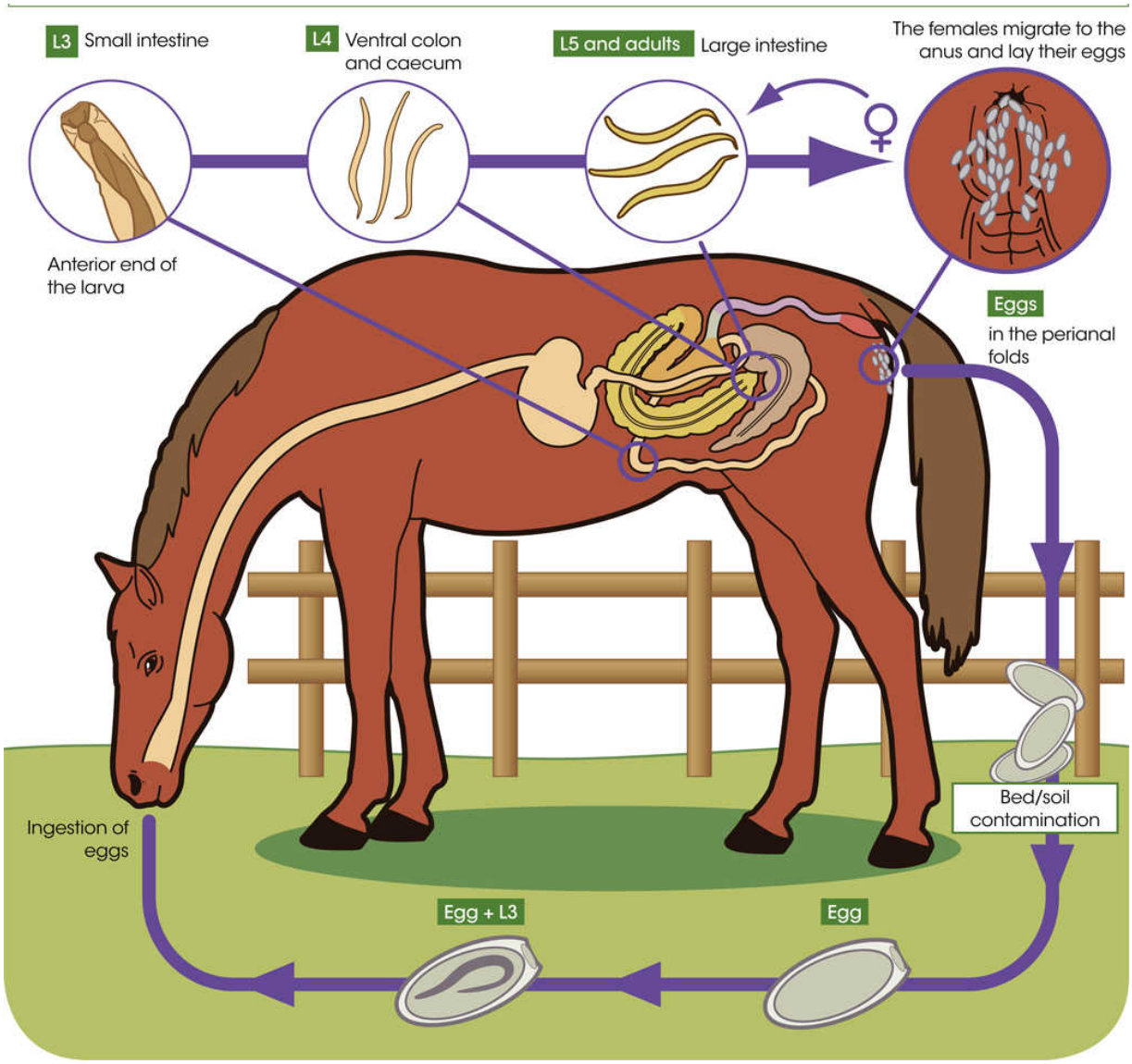


The anterior location of the vagina in Oxyuris females is a clear functional adaptation, which facilitates oviposition outside the large intestine.

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The life cycle of *P. vivipara* is also direct, but occurs completely within the host. Females produce larvae that are almost a large as them. These develop and reach maturity in the lumen of the colon and, after mating, give rise to a new generation of worms, ultimately generating populations of thousands of individuals. Larvae appear to be shed in the faeces, and subsequently contaminate feed and drinking water, through which they reach other hosts.

Life cycle of *Oxyuris equi*



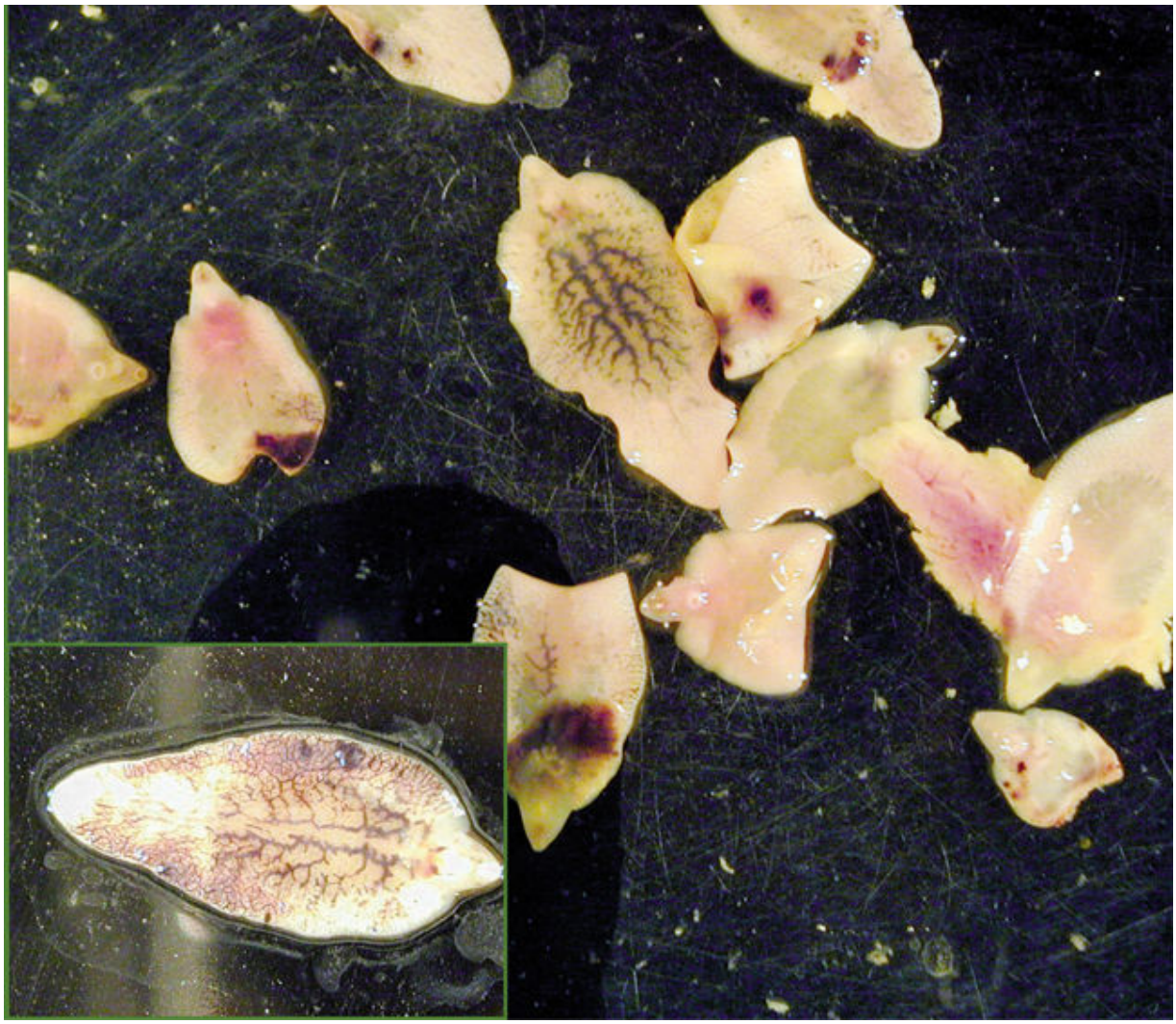
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24 Is *Fasciola* commonly found in equids?

Faecal samples from a group of horses from the province of Pontevedra, Spain, are sent for analysis. The horses exhibit loss of appetite and poor overall productivity caused by a suspected liver problem. Blood tests reveal a slight increase in GGT (γ-glutamyl-transferase) (118 U/l), hypoproteinaemia (4.0 g/dl), and haematocrit levels of 31 % after mild exercise. A laboratory in northern Spain detects the presence of *Anoplocephala* and *Fasciola* eggs in large quantities. The veterinary surgeon observes no improvement after treatment with triclabendazole and requests confirmation of the previous diagnosis.

1. What coprological methods are used to confirm the presence of eggs of *Fasciola hepatica* in faeces?
2. Is it common to detect eggs of *Anoplocephala* and *Fasciola* ?
3. What is the prevalence of fasciolosis in Spain and in other countries?
4. Where is this parasite found in horses?

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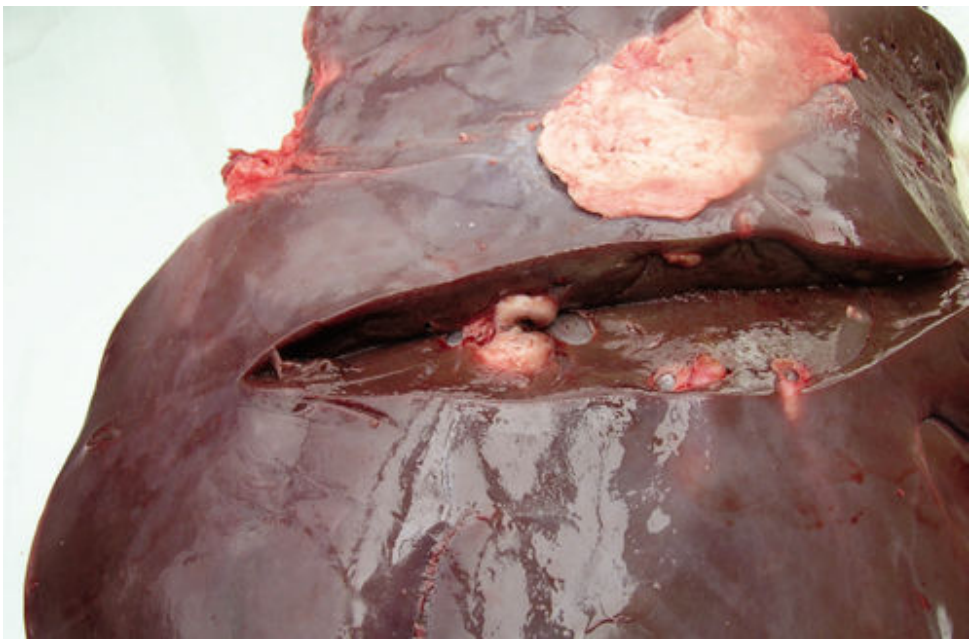
1 What coprological method should be used to confirm the presence of Fasciola hepatica eggs in faeces?

In equids, the most commonly used coprological method is flotation, which evaluates the ability of parasitic forms to float in solutions denser than water. However, the eggs of some parasites (e.g. fasciolae or paramphistomes) form a sediment when emulsified in running water, in which the faeces should be sufficiently diluted. In very dense solutions (e.g. sucrose, zinc sulfate) F. hepatica eggs will float, but collapse and are difficult to identify.

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2 Is it common to detect eggs of Anoplocephala and Fasciola?

In the present case, it should be borne in mind that eggs of Anoplocephala spp. are not excreted continuously in the faeces of equids, and are not detected with high sensitivity using coprological methods. Therefore, even if an animal is parasitised, eggs will not necessarily be detected. The presence of F. hepatica eggs is much less common; even in experimental infections in which this species has reached sexual maturity the faecal shedding of eggs is very scarce and intermittent.



Fasciolae are very likely to be observed upon sectioning the bile ducts of horses or donkeys that graze during the summer on damp pastures to which parasitised ruminants have had access. Adult forms cause periangiocholitis, as evidenced by the presence of thick and whitish cords beneath Glisson's capsule, especially on the ventral surface.

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The eggs of *Fasciola* are oval, measure approximately 150 x 90 µm, and have a highly characteristic light yellowish-brown colour.

The great weight of *Fasciola* eggs necessitates the use of cup sedimentation with water or flotation techniques using solutions denser than water, following the McMaster method. In both cases, it is necessary to use larger amounts of faeces than in other routine methods.

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It is very unusual to observe Fasciola eggs in the faeces of infected horses (fasciolae are more commonly found in donkeys). This is because none of the solutions used for faecal analysis of horses are sufficiently dense to allow the flotation of Fasciola eggs, which are very heavy. Moreover, fasciolae are not very fertile in these animals, and there is no correlation between the number of parasites present in the bile ducts and the number of eggs excreted in the faeces. In horses, the proportion of animals that test positive in coprological analyses is very low (25 % in some studies). By contrast, in ponies this figure can be as high as 89 %.

Therefore, coprology is not recommended to detect infection, but can be useful if very dense solutions are used (although some such as potassium iodomercuriate are prohibited), or if faeces are processed by sedimentation, using quantities of 10–30 g.

3 What is the prevalence of fasciolosis in horses in Spain and in other countries?

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Data on the prevalence of fasciolosis in horses in Spain are scarce. In some slaughterhouses, adult worms have been retieved from livers that were condemned due to the presence of fasciolosis lesions. There are very few reports describing the excretion of eggs, and analyses performed by multiple authors have all proven negative. Moreover, serodiagnosis by ELISA detection of antibodies appears to be unreliable due to the possibility of cross-reactions with other parasites. Seroprevalence, as determined by detecting the presence of antigens in faeces, is very low in Spain (<1 %), in line with reports from other countries, including Denmark, France, and South Africa.

Studies of the prevalence of fasciolosis in other countries are equally scarce. Post-mortem studies from the last century reported very high rates in Ireland (77 % in horses and 91 % in donkeys) and Yugoslavia (25 %), while in France rates calculated based on reports from slaughterhouses did not exceed 2 %. Much lower rates have been reported in studies using coprological analyses (less than 1 % in the Netherlands, Germany, and Australia). All studies have confirmed that this infection is much more common in donkeys than in horses. Seropositivity varies considerably depending on the method used. Haemagglutination appears to be the most reliable method for the diagnosis of fasciolosis. Using this technique, positive results have been obtained in 45 % of French horses with clinical signs compatible with fasciolosis (281) over the last 15 years.



The intermediate host is a small snail of the genus Lymnaea that lives in lakes, pools, and marshes with clay soil bottoms. In the snail, the miracidium transforms into a sporocyst, which in turns evolves to the

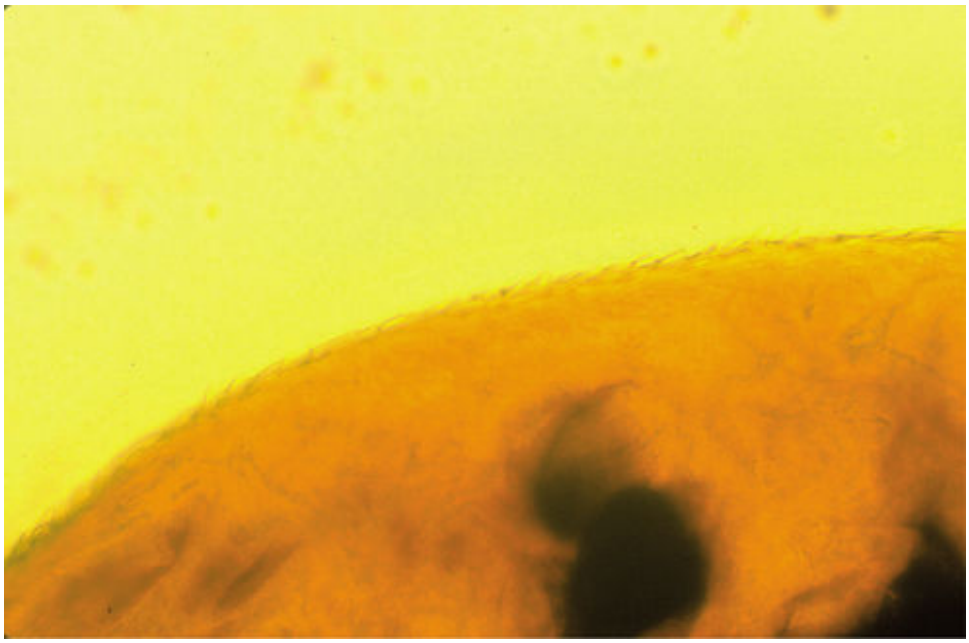
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redia stage. If the conditions are unfavourable, second-generation rediae are formed, but in general cercariae are formed directly after about 8–10 weeks. The snails emit the cercariae, which shed their tails and encyst in the grass and water, transforming themselves into infective forms known as metacercariae.

4 Where is this parasite found in horses?

The juvenile form of this parasite resides temporarily in the right parietal peritoneum and the liver parenchyma. Once it reaches maturity, it relocates to the bile ducts.

In horses, the presence of Fasciola impairs physical activity. Affected horses require longer to recover after normal exercise, and in those that routinely participate in hunts recovery may require weeks instead of days. Reports have also described poorer performance in show jumping animals and prolonged recovery periods in dams after foaling. Other alterations that may be observed include capricious appetite, diarrhoea associated with dietary changes, urticaria, and even colic. In some cases visible mucosae are icteric, and signs of chronic hepatitis or hepatic overload are observed. Secondary digestive alterations may occur, accompanied by poor general health, as evidenced by a poor haircoat and general weakness. Neither hyperthermia nor oedema are observed. Histology reveals subacute interstitial hepatitis associated with secondary portal hypertension and bile duct hyperplasia.



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Fasciola hepatica is a flat, leaf-shaped trematode, the entire surface of which is covered with small microscopic spines that erode the liver parenchyma and the bile ducts.

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25 Which parasites are implicated in diarrhoea of newborn animals?

A search for information on diarrhoea in foals reveal a monograph that states the following: “diarrhoea in foals is one of the most common clinical problems in animals of up to 2–3 months of age. The severity of diarrhoea ranges from acute forms that rapidly weaken affected animals to chronic diarrhoea that can persist despite treatment, affecting vitality, slowing growth, and increasing susceptibility to other gastrointestinal or respiratory diseases as a consequence of immune system compromise. Generally, diarrhoea is not the consequence of a single cause, but is complicated by other debilitating problems such as septicaemia, respiratory processes, arthritis, internal parasitoses, and some inflammatory diseases. The risk of mortality is very high in foals of less than 2–3 weeks of age given their lower resistance and limited reserves. However, in older animals, although less severe, diarrhoea constitutes a potential source of transmission of pathogens that cause diarrhoeal diseases, and has an impact on growth”.

1. What agents can cause diarrhoea in foals?
2. Is *Cryptosporidium* a primary or an opportunistic pathogen?
3. How can an outbreak of cryptosporidiosis in foals be confirmed?
4. What measures should be taken in the case of an outbreak of cryptosporidiosis in foals?
5. What zoonotic agents can be detected in horse faeces?

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1 What agents can cause diarrhoea in foals?

Most foals develop diarrhoea at some point. Clinical signs are usually mild, without general manifestations, and affected animals recover after a short time. The relative importance of the aetiological agents is not well understood, although colibacillosis likely plays an important role. Rotavirus and coronavirus infections can also cause diarrhoea. Other factors include nutritional causes, especially changes in diet, and infections caused by Strongyloides westeri. The colour, smell, and consistency of faeces, as well as increased body temperature, clinical signs of colic, and the age of the animal provide important clues as to the possible aetiology of diarrhoea.

Among the possible direct causes of diarrhoea in young equids are infections caused by Salmonella, Rhodococcus equi, Actinobacillus, Clostridium spp.,

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Escherichia coli, Cryptosporidium, Giardia, and Eimeria.

2 Is Cryptosporidium a primary or an opportunistic pathogen?

The role of Cryptosporidium spp. in diarrhoea in foals is controversial. In immunocompetent animals infections are self-limiting, although cases of fatal enterocolitis and chronic diarrhoea have been reported. The only clinical cases of cryptosporidiosis described in foals involve immunodeficient Arabian horses.

In these cases, infection is characterised by watery diarrhoea in foals with very low immune response capacity, and usually occurs secondary to other infections.

3 How can an outbreak of cryptosporidiosis in foals be confirmed?

The clinical signs of cryptosporidiosis are nonspecific. Although epidemiological data and clinical signs provide information on the severity of the process on a given farm, it is always necessary to establish an aetiological diagnosis using laboratory tests.

All microscopic methods of diagnosis are based on visualisation and recognition of the morphology of cryptosporidia. Cryptosporidium oocysts, which are relatively small in size and can be shed by the host in relatively small numbers or intermittently, are identified based on their selective and partial uptake of dyes.

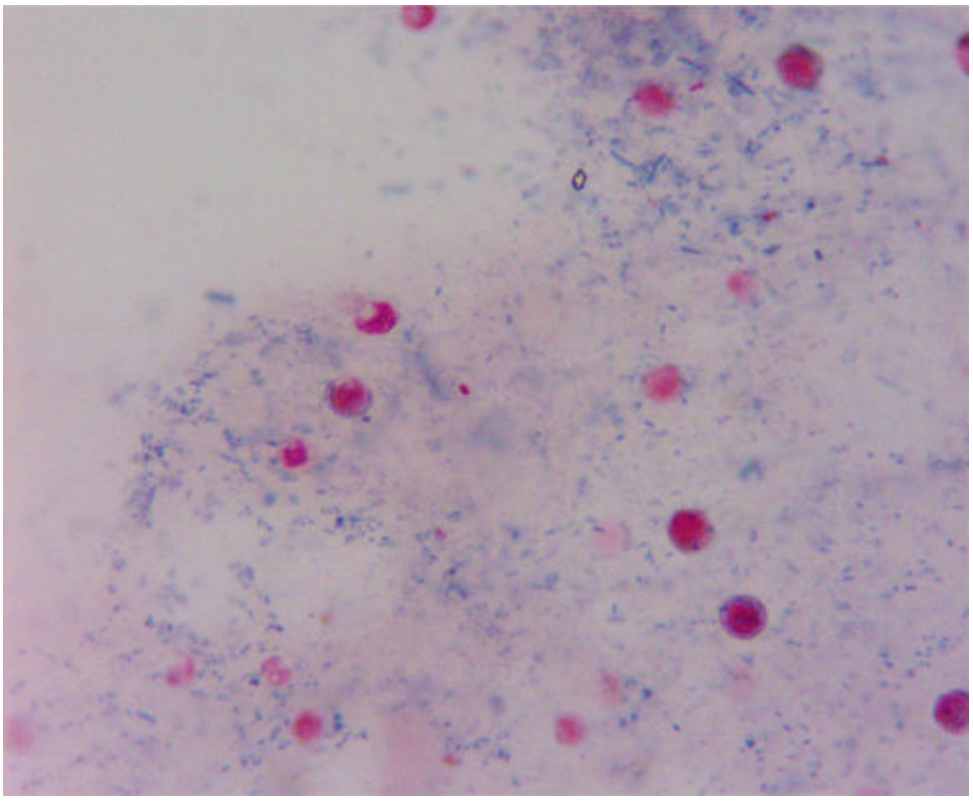
Several simple techniques are used to detect oocysts excreted in the faeces. Direct staining of faecal smears results in uptake of dye by either the oocyte or the medium, allowing visualisation of parasites. An alternative approach involves the use of concentration techniques, sometimes in combination with specific staining techniques. Immunological techniques provide a third option.

Among the most commonly used staining techniques are Giemsa staining, acid-fast staining, negative staining, and techniques based on the uptake by the parasite of fluorochromes such as phenol-auramine, auramine-rhodamine, auramine-carbol fuchsin, acridine orange, or DAPI (4, 6-diamidino-2-

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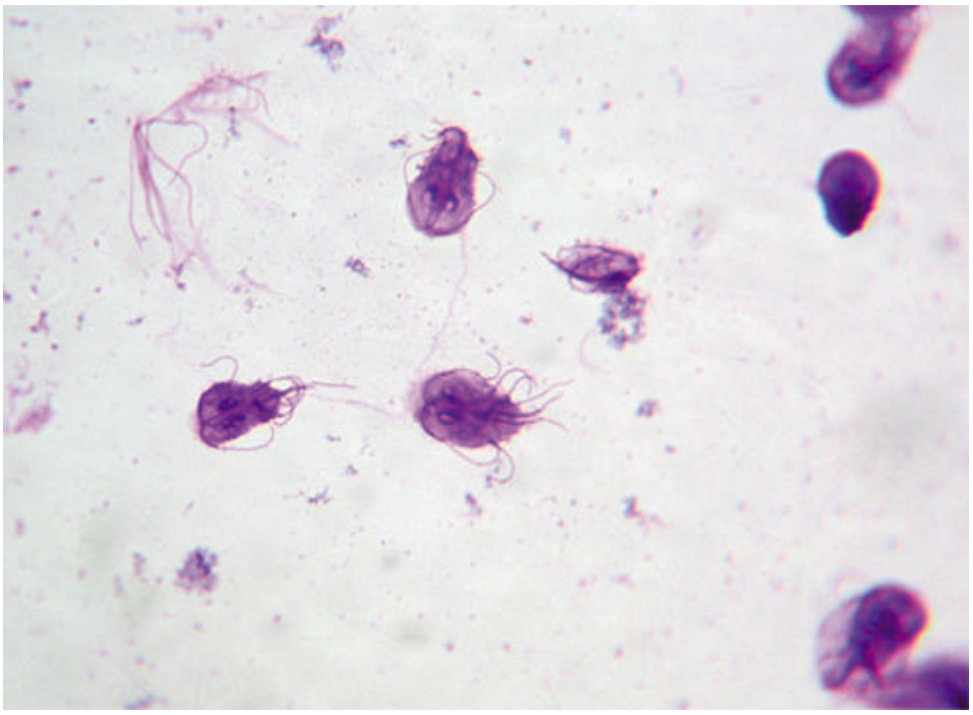
phenylindol dihydrochloride). Although there are studies that indicate that the Ziehl-Neelsen technique modified by Henriksen and Pohlenz produces very good results, other authors claim that techniques using auramine offer greater sensitivity and specificity.

Staining is performed directly on faecal smears or concentrated material. Faecal concentration techniques can be used to increase the sensitivity of oocyst detection. These methods include flotation (Sheather's sugar solution, zinc sulfate, or sodium chloride). Sedimentation in formalin-ether and formalin-ethyl acetate has also been described. After concentration, the oocysts can be observed directly by phase contrast or by applying some of the aforementioned staining techniques.



The oocysts of Cryptosporidium spp. are oval or subspherical and measure 5.6–7.4 µm. Upon excretion in the faeces they are sporulated and fully infective. Furthermore, they survive very well in the environment, and are highly resistant to variable environmental conditions.

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Trophozoites measure 12–17 x 7–10 µm. They attach to the intestinal mucosa and actively divide by binary fission. Upon detachment they are carried to more distal areas of the digestive system where they form cysts, which are then expelled with the faeces and mediate dissemination and transmission. Occasionally, large amounts of trophozoites are expelled; most are destroyed in the stomach, but some pass into the intestine, attach to the mucosa, and continue the biological cycle.

Immunological techniques can also be used to detect oocysts in faeces. These include latex agglutination, direct immunofluorescence, and indirect immunofluorescence using polyclonal or monoclonal antibodies.

4 What measures should be taken in the case of an outbreak of cryptosporidiosis in foals?

Of the wide variety of drugs tested for the treatment of cryptosporidiosis, very few show demonstrated efficacy, although some contribute to a decrease in oocyst shedding and in some cases ameliorate diarrhoea.

Given the lack of effective drugs, symptomatic treatment is one of the priorities once an outbreak of diarrhoea occurs on a farm. Moreover, given the self-limiting nature of the infection, the introduction of adequate measures can prevent mortality in the group or reduce losses due to morbidity to reasonable levels.

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The first measure should consist of oral or parenteral administration of electrolyte solutions (Na+) together with glucose or amino acids. However, the establishment of adequate hygiene/sanitary measures and improvement of the management of farms can prevent clinical manifestations of infection, and decrease infection pressure in areas in which cryptosporidiosis is considered endemic.

5 What *z* oonotic agents can be detected in horse faeces?

Equids can eliminate Giardia cysts and Cryptosporidium oocysts, but the prevalence of both parasites in this species means the probability of transmission to humans is practically nil.

The strains or isolates of Cryptosporidium species recovered from foals with diarrhoea in certain countries have been identified as genetic variants of Cryptosporidium parvum that closely resemble human and bovine isolates, and carriers of GP60 IIaA18G3R1 alleles, indicating their zoonotic potential.

The species primarily responsible for human infections in many places is Cryptosporidium hominis. About 50 % of outbreaks in the United Kingdom have been attributed to this species. Studies of GP60 in C. hominis isolates collected during outbreaks in the United Kingdom have allowed identification of nine subtypes, of which the IbA10G2 subtype predominates.

Given the marked genetic diversity among isolates and because taxonomy has not yet answered all the pertinent questions regarding host-parasite specificity, it is impossible to confirm the zoonotic potential of the isolates, even though some exhibit clear signs of zoonotic potential.

Giardiasis does not usually occur with diarrhoea, but has a significant impact on health given that the varieties of the species found in equids correspond to human varieties and are therefore considered potential zoonotic agents.

Studies have reported prevalences of giardia infections of up to 20 % in horses. The majority involve asymptomatic animals (clinical disease is rare). Giardia is the pathogen most commonly identified in outbreaks of digestive processes (e.g. diarrhoea) acquired from water by humans.

Giardias belong to the genus Giardia, which is a highly heterogeneous group. Initially these species were identified based on the morphology of the median body (MB), and later based on the electrophoretic characteristics of

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some of their enzymes or their chromosomes. Current molecular techniques have clearly identified several species, of which the most prevalent in vertebrates is Giardia duodenalis (also known as Giardia intestinalis, Giardia lamblia). This species is the only one capable of infecting humans and more than one host species.

The identification of genotypes has enabled verification of the existence of several human genotypes in equids, such as genotypes A1 and A2 of G. duodenalis, as well as others known by their species names (genotype B4 or Giardia enterica and genotype E or Giardia bovis). Currently there is no direct relationship between diarrhoea in equids and the presence of cysts in the faeces, and co-infection with Cryptosporidium is possible in very young animals.

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2

Q & A
Endoparasites of the **respiratory system**

Chapter 2

Q & A Endoparasites of the respiratory system

Introduction

This chapter covers clinical cases and pathologies caused by various endoparasites of the respiratory system of equids. Most of the pathologies discussed are caused by parasitic species that live in other organs or body parts, but migrate through the respiratory system, in which they settle temporarily. The exceptions are pathologies that specifically affect the respiratory tree (mainly the bronchi and bronchioles) and those that are restricted to the nostrils and sinuses, both of which are caused by parasites that reside in the respiratory system.

In most animal species, the presence of highly pathogenic parasite species in the respiratory system is rare. Nonetheless, several parasite species frequently found in the respiratory system of equids are discussed in this chapter.

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One such disease is dictyocaulosis, which is caused by bronchopulmonary nematodes that produce clinical pathologies in horses that share facilities and/or grazing areas with donkeys, in which infection is almost always asymptomatic. The low level of specificity of *Dictyocaulus arnfieldi* , a natural parasite of donkeys and horses, leads to potentially serious pathologies, especially in foals. Given the importance of this disease, especially when it coincides with respiratory diseases of other aetiologies, it is important to be familiar with the appropriate diagnostic methods and techniques in order to establish adequate therapeutic and prophylactic measures.

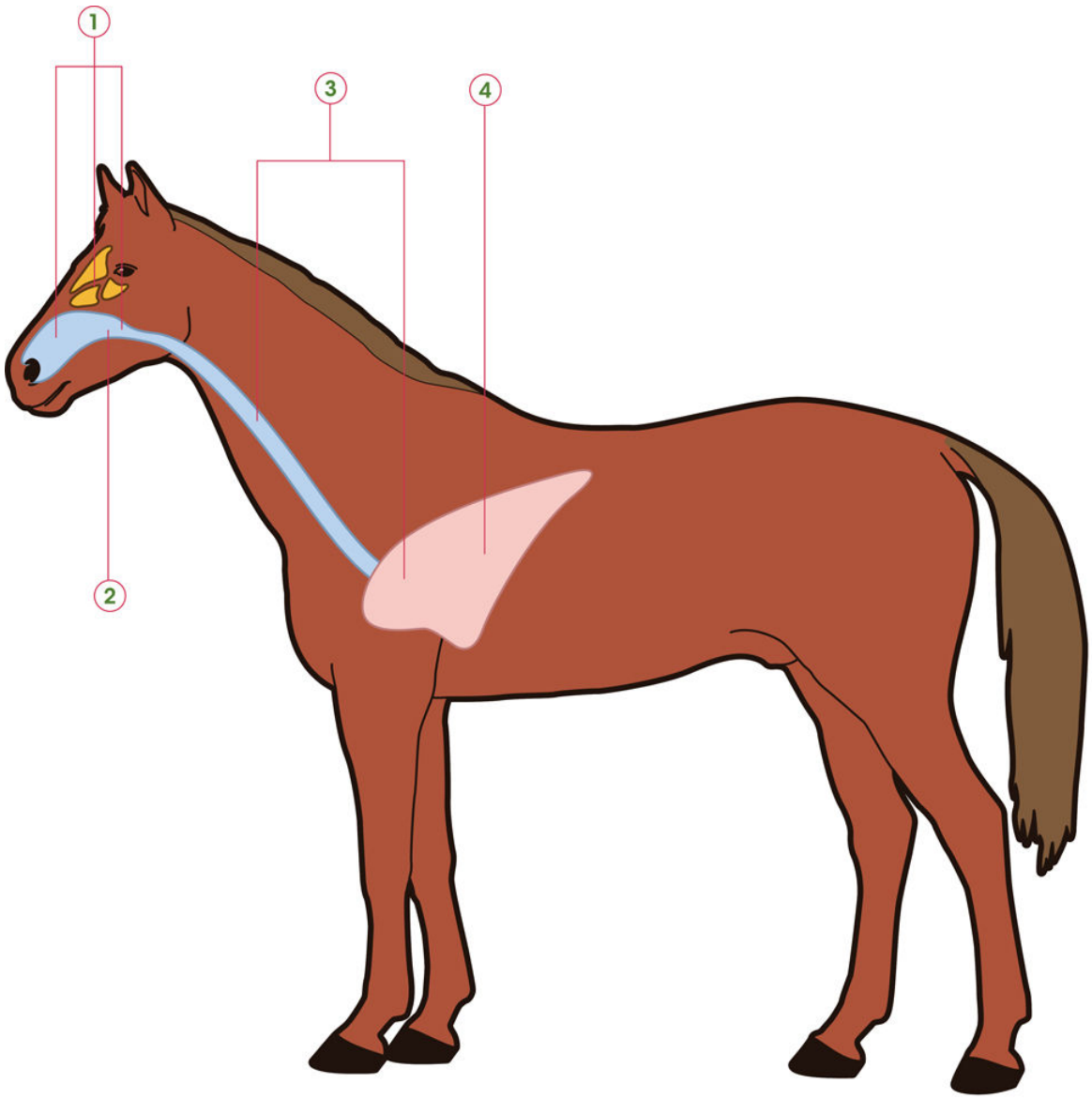
Cavity myiasis caused by the presence of *Rhinoestrus* spp. larvae in equids is another parasitic process specific to the respiratory system. The presence of larvae of these oestrids in the nostrils, nasal cavities, and sinuses of equids causes various alterations that can be confused with more severe processes, especially when affected animals exhibit neurological signs and behavioural changes. This type of myiasis can occasionally affect humans (ophthalmomyiasis), and should therefore be considered a parasitic zoonosis.

The other conditions are not exclusively respiratory, since the pulmonary disorders caused by the parasites responsible are the consequence of their migration through the respiratory system. These include parasitic diseases caused by various species of *Gasterophilus* , which temporarily reside in the oral cavity and adjacent areas before definitively settling in the host's stomach and intestine.

Infections caused by *Parascaris* are of particular interest for several reasons. As described in the chapter on endoparasites of the digestive system, *Parascaris* spp. can give rise to serious disorders, especially in young foals. These disorders are the result of two distinct factors. The first is intra-organ migration, which begins and ends in the intestine. This migration route is considered by many authors to be a consequence of the loss of an ancestral intermediate host, and includes the lung, where the parasite causes significant clinical repercussions. The second factor is the underlying eosinophilia, which is not exclusive to parascariosis, or even helminthosis. It is induced by the migration of larval forms through the lung, resulting in characteristic clinical signs that are frequently observed in equids as well as other species of domestic animals.

Major parasites of the equine respiratory system

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1. *Rhinoestrus*: nasal cavity, sinuses, and pharynx
2. *Gasterophilus*: pharynx and soft palate
3. *Dictyocaulus*: trachea and bronchi
4. *Parascaris*: lung (migrating larvae)

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26 How is dictyocaulosis diagnosed in a horse?

An adult horse develops a severe respiratory process with suspected pneumonia after being introduced into a riding school where horses and donkeys closely coexist. Coprological analyses are performed every 3 months to assess the need for anthelmintic treatment. The results of the most recent McMaster test had been negative for most animals, including the patient. Embryonated eggs are detected in tracheal aspirate samples.

1. Why were embryonated eggs detected in the tracheal aspirate?
2. Are adult forms of *Dictyocaulus* easy to identify?
3. What type of coprological analysis should be performed to rule out dictyocaulosis?

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1 Why were embryonated eggs detected in the tracheal aspirate?

The aetiological agent of dictyocaulosis in equids is *Dictyocaulus arnfieldi* . This whitish nematode of over 5 cm in length is found in the bronchi and bronchioles of donkeys. The presence of *Dictyocaulus* has no major pathological effects in these animals, which can harbour heavy parasitic loads without developing clinical signs. Donkeys therefore rarely exhibit clinical signs, except for certain chronic and nonspecific respiratory processes such as cough or dyspnoea.

The parasite causes severe lesions in the lungs of horses. These occur specifically at the beginning of the infection process, when the developing larvae reach the respiratory system via the bloodstream. In horses these nematodes rarely reach sexual maturity and the biological cycle is not completed. Consequently, the embryonated eggs are excreted, are

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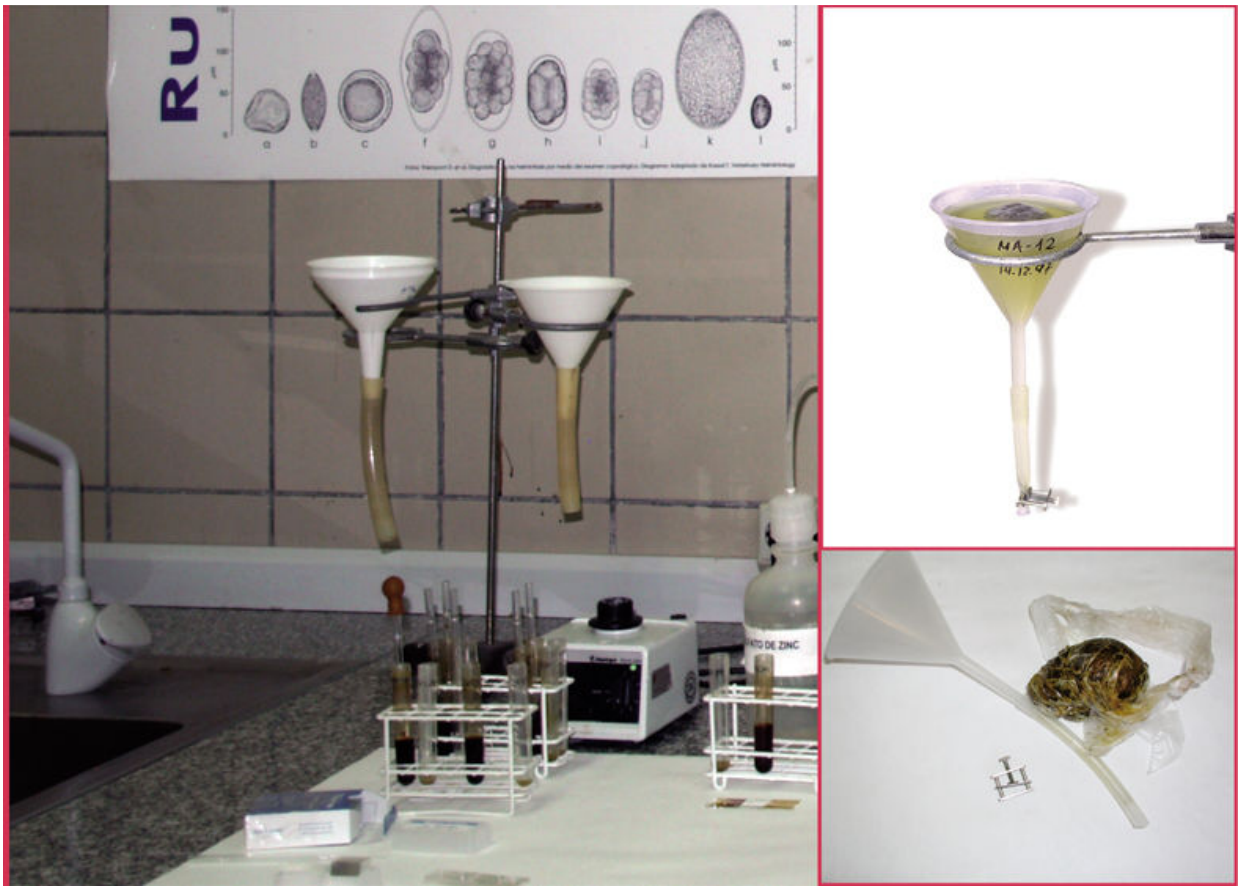
subsequently ingested and hatch in the digestive tract, and the resulting L1 form is shed in the faeces. By contrast, the cycle is completed in donkeys, which shed large numbers of larvae.

2 Are adult forms of *Dictyocaulus* easy to identify?

The adults are whitish and filiform. The males measure 35–43 mm and the females 45–65 mm. The male has a non-lobed copulatory bursa, a rudimentary gubernaculum, and two thick, paired spicules. The caudal end of the female is rounded and the vulva is located towards the middle of the body, covered by a flap similar to that of other *Dictyocaulus* species. The uterus contains embryonated eggs that measure 85–95 µm.

To perform the Baermann technique, a glass funnel of the appropriate size is placed in a ring attached to a vertical support. A rubber tube, which can be closed using a metallic clamp, is attached to the funnel. Faeces are introduced into the funnel, either in a gauze bag or upon a metal filter, and the funnel is filled with warm water. After 5 to 7 hours, the larvae emerge from the faecal mass and accumulate in the rubber tube above the level of the clamp. A volume of 0.5–1.0 ml is collected in a centrifuge tube, the larvae are concentrated by centrifugation, and the sample is examined under a microscope.

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3 What type of coprological analysis should be performed to rule out dictyocaulosis?

Given that larvae are shed in the faeces, it is advisable to carry out a larval migration inhibition test; these larvae are difficult to detect using routine flotation techniques (e.g. McMaster technique), especially in adult animals and those with chronic processes and nonspecific clinical signs. Flotation methods can also be used to assess the concentration of eggs. These techniques are more effective for the identification of carrier animals.

In horses values of 20–40 larvae per gram of faeces (LPG) generally give rise to a pathological process, while values >100 LPG are compatible with an evident clinical process. However, in donkeys the number of larvae shed in faeces is usually greater, and values of 150–300 LPG are considered normal. In addition to the aforementioned tests for parasitic forms, the detection of certain cell types, such as eosinophils and mast cells in

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bronchial secretions obtained by aspiration (in which embryonated eggs and/or L1 forms can also be observed), can help confirm diagnosis.

In any case, in equids it is important to always rule out other possible respiratory pathologies, the frequency of which is usually higher.

Dictyocaulosis is often definitively confirmed post mortem, based on the presence of lesions and adult parasites in the respiratory system.



Larvae of *Dictyocaulus arnfieldi* are small, lack a cephalic button, and have a short, pointed tail. This image shows a larval form of *Dictyocaulus filaria* (sheep) in which the cephalic button is evident.

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27 Why do donkeys transmit dictyocaulosis to horses?

A Spanish association, Asociación Amigos del Burro ("Friends of the Donkey") requests the necessary documentation to apply for membership of the British organisation The Donkey Sanctuary. The latter organisation began as a shelter, but now focuses on the breeding and conservation of the majority of donkey breeds, and promotes the creation of satellite centres in other countries for the conservation of native species. In addition, it promotes awareness of the use of the donkey as a companion animal for horses that are kept alone in private fields, although the organisation provides clear information on the risk of transmission of diseases such as dictyocaulosis.

1. Does *Dictyocaulus* have different pathological effects in these two species?
2. What type of biological cycle does it have?
3. What are the pathological effects?

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1 Does *Dictyocaulus* have different pathological effects in these two species?

Due to interspecies and parasitic differences, it is not recommended that horses, ponies, and donkeys share the same pastures given the risk that donkeys, which are asymptomatic carriers of *Dictyocaulus* , may contribute to clinical outbreaks in horses, especially young foals. Foals are more susceptible to infection than adult horses.

The main host of *Dictyocaulus arnfieldi* is the donkey, in which evident clinically detectable pathologies are rarely observed. However, because *Dictyocaulus* can live for up to 5 years in these animals, it can cause long-lasting contamination of the environment. This parasitosis is more common in humid and temperate zones, and data on its prevalence in Spain are scarce.

Horses are “less specific” hosts, and therefore develop more severe clinical signs (almost always chronic), although these signs are not pathognomonic.

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Infections are characterised by obvious respiratory signs and shedding of the L1 form in faeces is common. Clinical suspicion can be confirmed based on the affected animal's grazing history (shared grazing by horses and donkeys) and their response to treatment.

2 What type of biological cycle does it have?

The biological cycle of *D. arnfieldi* is very similar to that of the *Dictyocaulus* parasites of ruminants, with the difference that eggs are embryonated when shed. After 12–20 hours the L1 form hatches, provided that thermal and hygrometric conditions are adequate, and moults twice to produce the infective L3 form within 5–7 days, depending on temperature and humidity. Sporangia of *Pilobolus* spp. contribute to the dispersal of these larvae, as also observed for *Dictyocaulus* parasites of ruminants. The L3 form can survive at temperatures of 10–35 °C. Larvae in grass or drinking water are ingested, shed their cuticle, cross the intestinal wall, and reach the mesenteric ganglia, where they moult to produce the L4 form after 4 days. From the mesenteric ganglia they migrate to the heart via the lymphatic circulation and enter the bloodstream to reach the alveoli and bronchioles, where they moult again to the pre-adult or L5 form, which matures sexually and settles in the bronchi.

The first L1 forms appear in the faeces of parasitised animals after about 14 (donkeys) or 30–40 (horses) days, and are shed for 1.5–6 months by horses and 5 years or more by donkeys.

3 What are the pathological effects?

Clinical signs are generally mild, and can be absent altogether in donkeys. In some cases the following clinical signs may be observed: mild hyperpnoea, dry cough, mucous discharge, and nasal discharge, which is rich in eggs and/or larvae and is purulent in cases of superinfections; and, less frequently, dyspnoea, weight loss, and poor general health.

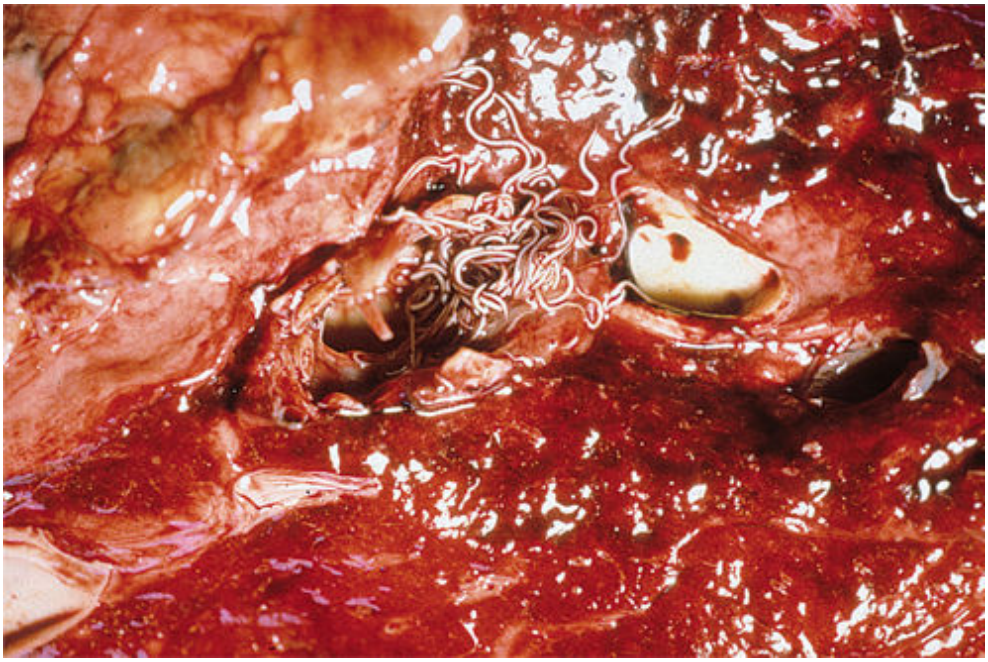
Infection in horses is usually chronic, and tends not to result in evident clinical signs, although respiratory difficulty after exercise may be observed. Death occurs in rare cases; there are reports in the literature of animals that

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died and were found to have large numbers of adult parasites in the respiratory tree, as well as concurrent viral infections.

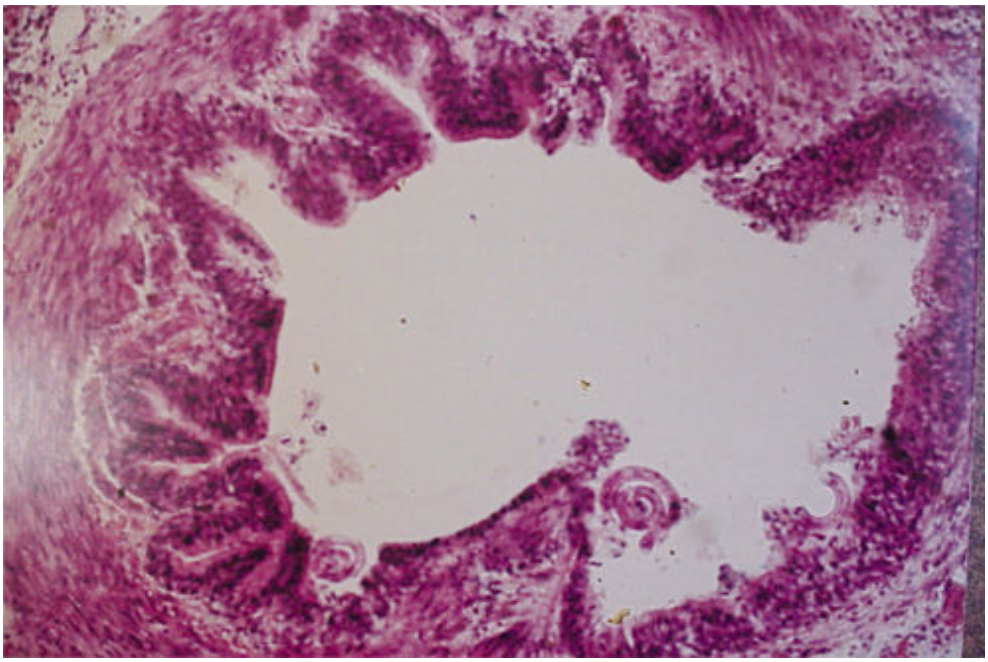
Macroscopically, pulmonary alterations are characterised by the presence of raised, circumscribed lesions (about 3 cm in diameter) with irregular borders on the pulmonary surface. The lumina of the bronchi contain abundant mucus as well as immature and adult worms. The clinical picture is completed by the presence of areas of condensation and emphysema corresponding to bronchopneumonia.

Microscopy reveals areas of bronchitis and chronic catarrhal bronchiolitis, with cellular infiltrates and areas of alveolar haemorrhage.



Adult worms are found in the smallest bronchi and bronchioles, which become almost completely obstructed.

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In affected areas, the bronchiolar epithelium appears hyperplastic (thickened) and contains abundant mucus. Intense cellular infiltration is also observed around the bronchioles. These alterations prevent proper expulsion of inspired air.

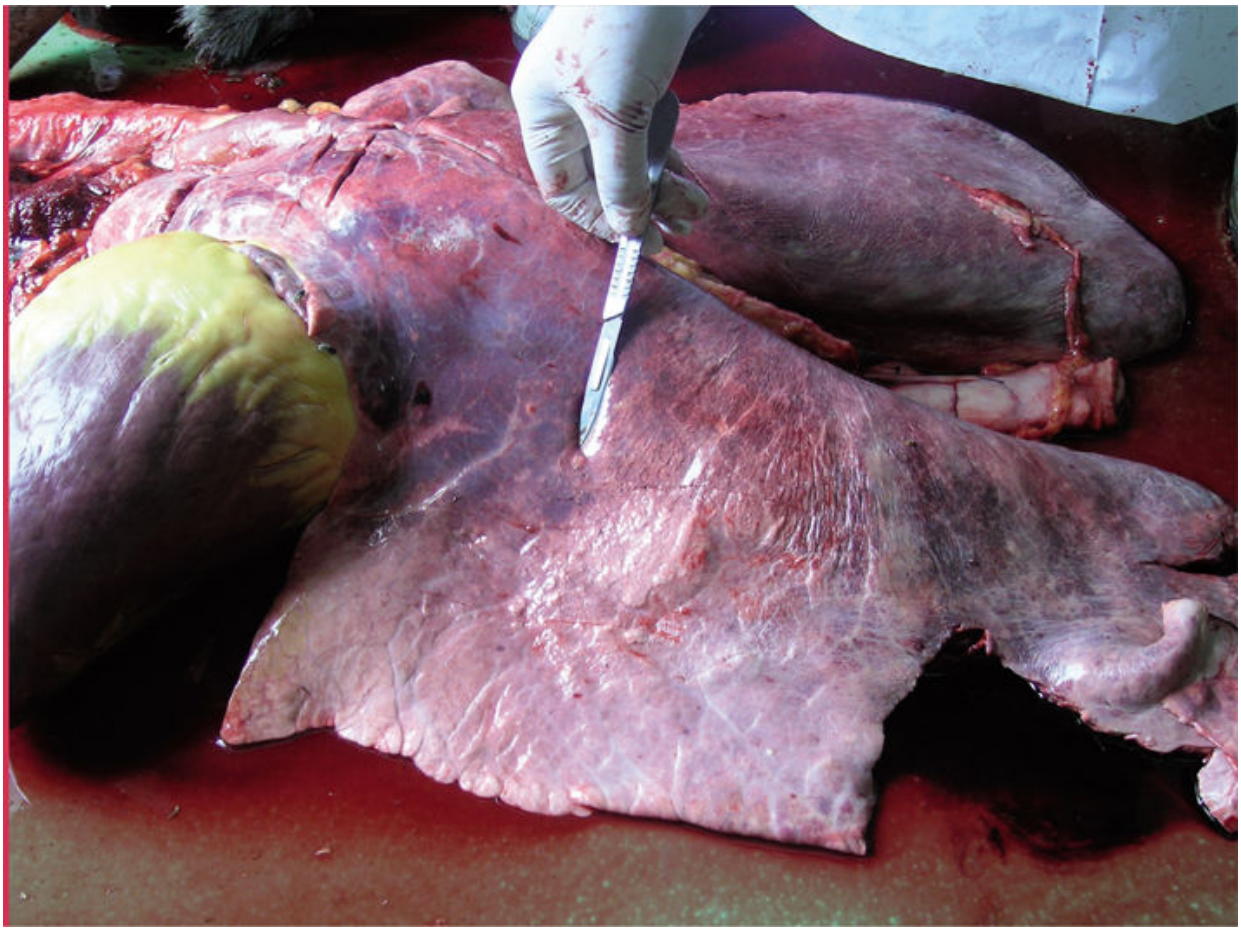
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28 Why are so many eosinophils produced in cases of parasitic bronchitis?

During necropsy of a 5-month-old foal, spherical subpleural nodules of 0.3–1 cm in diameter are clearly evident in the lung. They are greyish and translucent, and protrude slightly. Samples are taken for further analysis. The most notable histological finding is marked eosinophilia. The interlobular septa and subpleural areas are enlarged due to the presence of oedematous fluid and abundant eosinophils. Areas of collapsed alveoli and eosinophilic alveolitis, together with alveolar haemorrhage, are also evident. No nematode larvae are identified in any of the sections analysed.

1. Which parasite causes this type of lesion?
2. What lesions are caused in the liver by migration of the parasite?
3. Why are there so many eosinophils?
4. Is eosinophilia observed in this phase of infection?

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1 Which parasite causes this type of lesion?

The presence of eosinophils has always been associated with infections caused by parasites and with allergic reactions. During *Parascaris* invasion of the lungs, the most significant reactions are eosinophilic bronchitis and bronchiolitis.

2 What lesions are caused in the liver by migration of the parasite?

Migration of the larvae through the hepatic parenchyma during the invasion phase results in the rupture of numerous capillaries, leading to haemorrhages beneath the capsule and in the parenchyma as early as 2 days after

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penetration. However, even in cases involving thousands of larvae, this is a temporary process and is not usually accompanied by any clinical signs.

3 Why are there so many eosinophils?

It is important to remember that larvae of *Parascaris equorum* moult in the lung parenchyma, unlike other species in which this process occurs in the liver parenchyma. For this reason the greatest pathological effects are observed in the lung. The cuticle of the larvae of ascarids contains a substance that interferes with mast cell degranulation. Eosinophils are attracted to sites of parasite-host contact, giving rise to transient eosinophilia that leads to lymphocyte invasion after about 3 weeks and the subsequent formation of subpleural nodules.

These processes explain the respiratory alterations observed in parasitised animals. Alveolitis, bronchiolitis, and bronchitis lead to respiratory dysfunction and disorders of variable severity, which can manifest between 15 and 30 days after infection.

The final stage occurs in the small intestine. There, adult parasites obtain nourishment from the substances ingested by the host, resulting in physiological decline and hypoalbuminaemia of variable severity. Although appetite is unaffected, the general condition of the host deteriorates, as evidenced by decreases in weight gain and protein absorption.

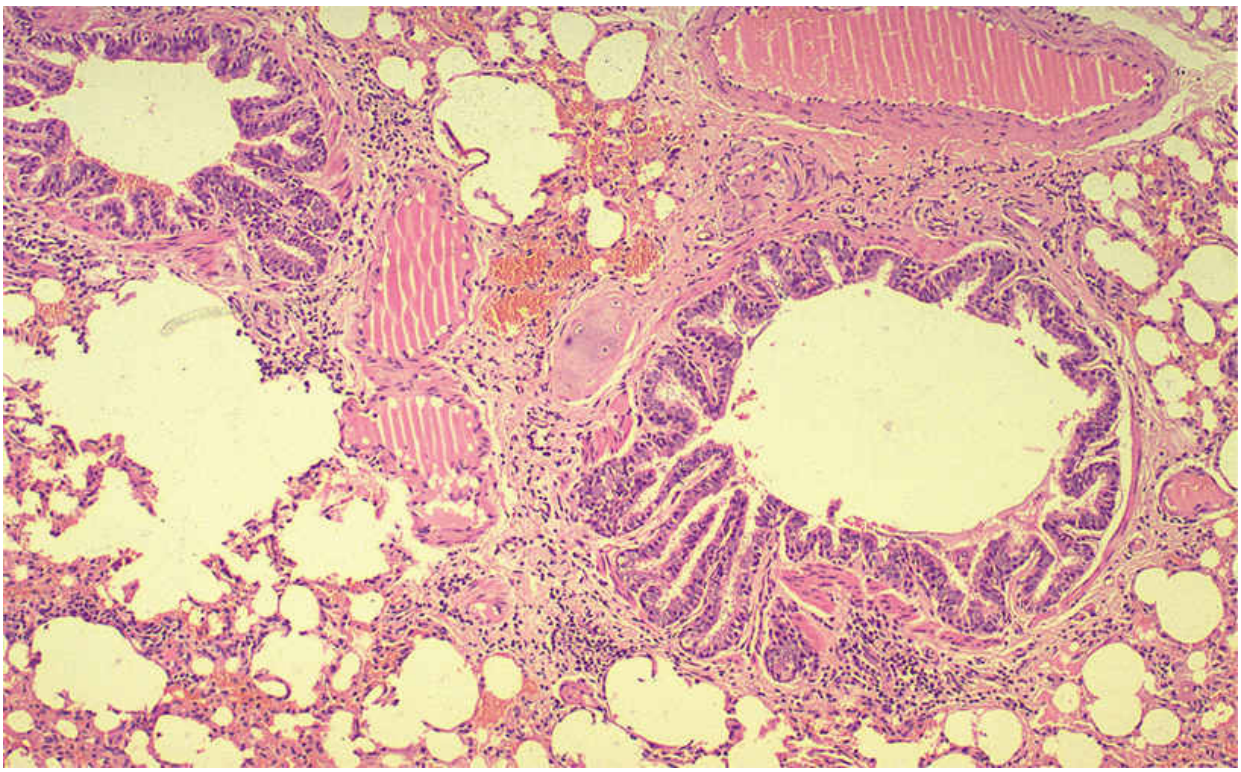
4 Is eosinophilia observed in this phase of the infection?

Alterations in certain blood parameters coincide with intraorgan larval migration. For example, marked eosinophilia, the intensity of which is correlated with the infective dose, appears 10–40 days after infection, and subsequently diminishes or practically disappears. Eosinophilia may recur in cases of reinfection.

Eosinophilic infiltrates in the interlobular septa and subpleural areas lead to alveolar collapse. Larvae and abundant mucus can also be seen in histological sections.

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The nodules consist of a lymphocytic mass surrounded by a fibrous capsule and eosinophils.



Intense eosinophilic cellular infiltration is observed in affected areas. The septa and subpleural areas are filled with oedematous fluid containing eosinophils. Collapsed alveoli and haemorrhagic areas are also observed. Cross sections of larvae may be visible.

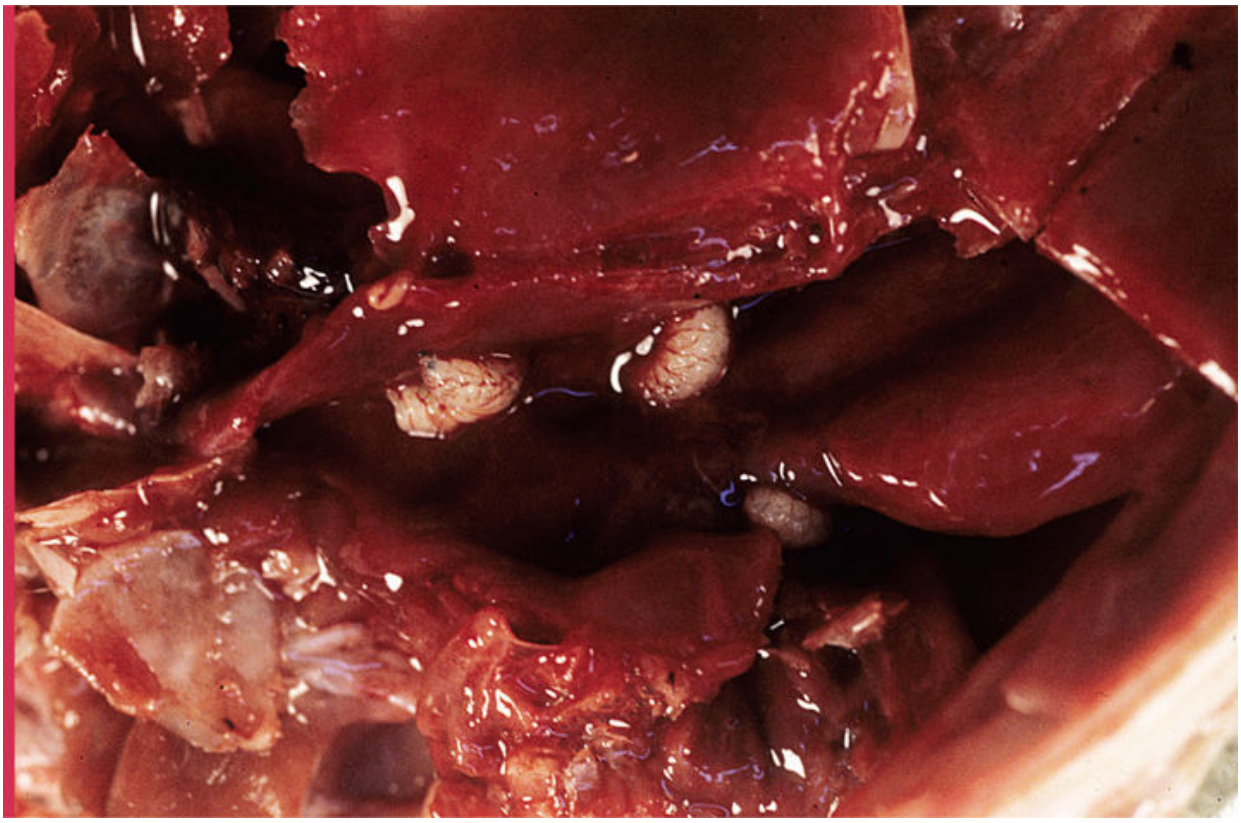
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29 What fly larvae can cause catarrhal processes in horses?

During a summer stay on the island of Sicily, veterinary advice is requested for a very peculiar case. A female foal aged 14 months had abruptly begun to exhibit neurological signs compatible with encephalomyelitis, including a radical change in behaviour, agitation that was exacerbated during the day in the presence of flies, and jerking and abnormal movements of the head. When the animal was tied to a wall ring and thereby forced to raise her head, she would back away and throw herself to the ground with such force that she had to be released. The foal had also developed a catarrhal process of the upper airways with cough and dyspnoea, and respiratory sounds indicative of laryngeal stenosis were audible. Given the animal's failure to respond to all treatments administered, it is decided to opt for euthanasia. Necropsy reveals no brain involvement, but does confirm an acute inflammatory process in the pharyngeal cavity, nasal passages, and frontal sinuses, the effects of which extend to the larynx and oesophagus. In total, 65 fly larvae in all stages of development are recovered.

1. What species of fly larvae can parasitise the nostrils and surrounding tissues of horses?
2. Is there any risk to humans?

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1 What species of fly larvae can parasitise the nostrils and surrounding tissues of horses?

Rhinoestrosis is a parasitic disease caused by larvae of *Rhinoestrus* spp. (Diptera, Oestridae), which settle in the nasal cavities, sinuses, and pharynx of horses, donkeys, and zebras. It was thought to be restricted to Asia and Africa, but since the beginning of the 21st century numerous cases have been reported in Europe, especially in southern Italy.

Female flies deposit the first-stage larva (L1) during flight near the nostrils or eyes of equids. Upon reaching the nasal cavity, the larva moults to produce the third-stage (L3) form. Aided by headshaking movements of the host, this form abandons the host and buries itself in the ground, where it pupates.

A female can produce 700–800 larvae. The yellow larvae reach up to 17 mm in length and have spines and protuberances on the dorsal and ventral surfaces. As in other oesterids, the stigmatic plates, which mediate

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respiration, are located in a half-moon-shaped depression in the last abdominal segment.

The presence of *Rhinoestrus* larvae causes local inflammation, and is characterised by clinical pictures ranging from mild dyspnoea and impaired athletic performance to death by encephalitis as a result of invasion of the ethmoid bone and meninges. Parasitation causes significant morbidity and mortality, particularly in domestic horses. In severe infestations, the number of larvae per animal can exceed 150, and mortality rates can reach 82 %. In these cases, the larvae are not limited to the nasal cavities but are also found in the pharynx. Larvae may also be found in the vicinity of the ethmoid bone, from where they can potentially affect olfactory nerves and areas of the cerebellum.

Interestingly, although the pathogenic effects of these larvae are well known, this particular myiasis remains poorly understood. Parasitised animals show clinical signs compatible with other respiratory processes, such as bordetellosis or herpesvirus infections.

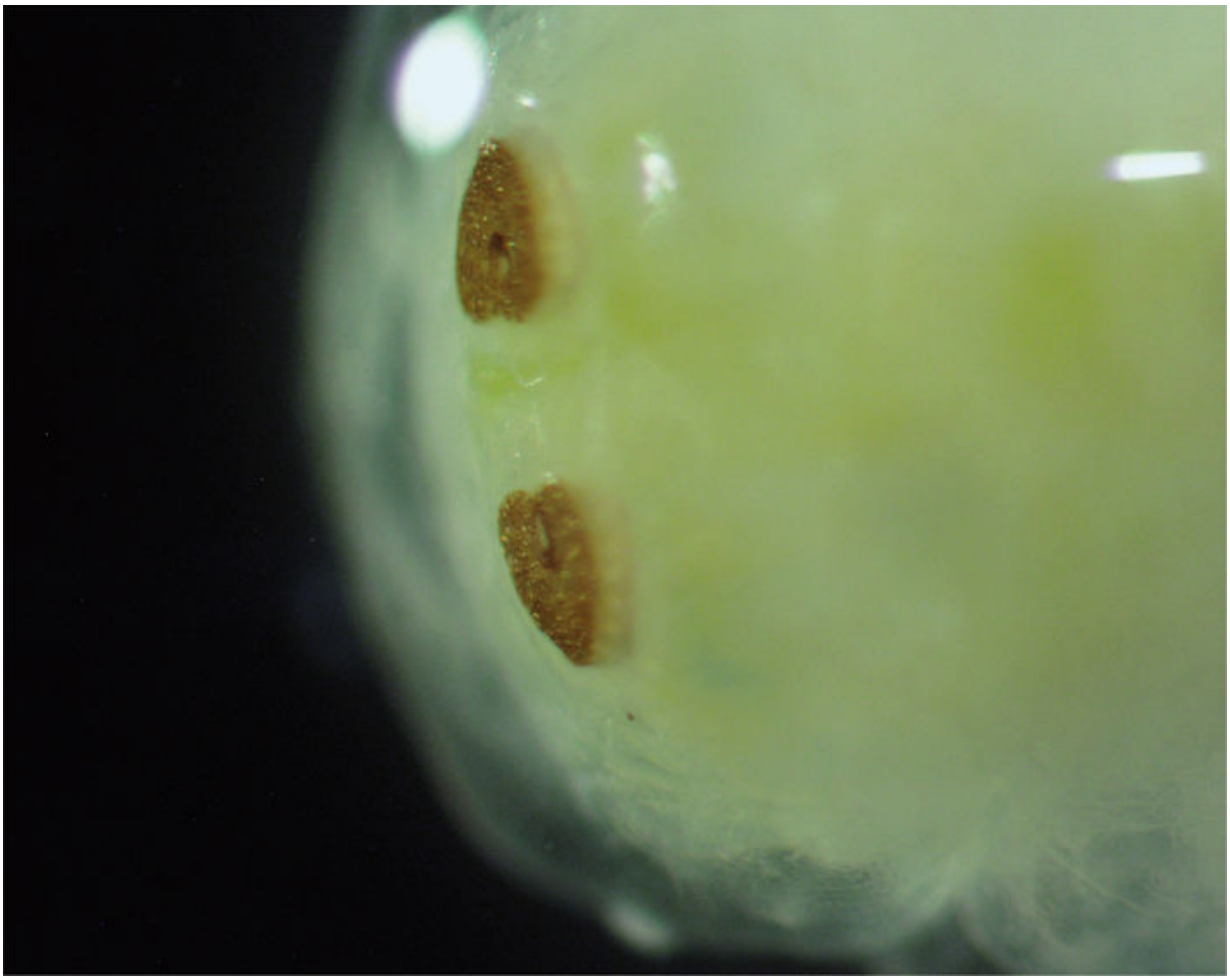
Diagnosis is established upon inspection of the nasal cavities or identification of expelled larvae. However, even when endoscopy is performed, *in vivo* diagnosis is very difficult, as it is almost impossible to access the area behind the soft palate. Postmortem diagnosis is straightforward, but is not a routinely performed task in slaughterhouses, and necropsies are not usually performed in cases of respiratory processes.

Currently available molecular and immunological tools allow diagnosis of equine rhinoestrosis.

2 Is there any risk to humans?

Parasitisation by *Rhinoestrus purpureus* , and other species of the same genus that affect horses, is considered a zoonosis, and can cause ophthalmomyiasis in humans that have close contact with equids. In both animals and humans, female flies propel larvae into the nasal cavities and the conjunctival sac. In horses, the larvae descend to the pharyngeal cavity and nasal passages where they continue their development. In humans these larvae cause painful ocular processes.

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The stigmatic plates, which have a characteristic morphology that allows specific identification of the larvae, are located in a half-moon-shaped depression in the last abdominal segment.

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30 What parasite can cause laryngeal stridor?

A group of 4-year-old horses with temporary dysphagia begins to develop more serious problems when provided with food, characterised by a dry cough and spastic mastication in the absence of food in the mouth. In the days preceding the examination, the episodes of coughing become more frequent and coincide with a whistling sound indicative of obstruction of the passage of air through the upper airways. After opening of the mouth of one of the animals to inspect the pharynx, numerous (43) larvae of *Gasterophilus* are observed attached to the bottom of the soft palate and in the pharynx.

1.How big are the larvae and why are they there?
2.What species is this and how prevalent is it?

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1 How big are the larvae and why are they there?

The larvae of *Gasterophilus pecorum* differ to those of other gasterophilids, since the eggs are deposited in the soil (leaves and stems of plants), but do not adhere to the hair of the hosts. The females live for about 4 days, during which time they lay numerous eggs (1300–2500) that are deposited in groups of 10–15 scattered across the pasture.

Embryonic development occurs over 5–8 days, although the larvae remain inside the egg and only hatch when ingested with grass by horses and donkeys. Hatching occurs rapidly (within 3–5 minutes). Immediately afterwards the larvae penetrate the mucosa of the lips, gums, cheeks, tongue, and hard palate, which they perforate, and migrate towards the base of the tongue and the soft palate. Here, numerous larvae can be found, and can remain for 9–10 months until reaching the L3 phase. The "older" L3 forms pass into the stomach (gastric fundus) where they transform into the "mature" L3 stage.

They can also be swallowed together with food and settle in the wall of the pharynx, oesophagus, or stomach.

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2 What species is this and how prevalent is it?

G. pecorum is one of the most commonly found and most pathogenic species of parasite in horses. Serious outbreaks of infestation by this species have been described, resulting in many deaths, with a high parasitic load (up to 250 larvae) observed in some animals; in other outbreaks, infestations affect young animals, which have difficulties in swallowing and exhibit clinical signs that include muscular hypertrophy in the oesophagus as a result of severe myositis.

The larvae of *Gasterophilus pecorum* can attach to the soft palate, where they remain for several months until reaching almost 2 cm, causing ulcers and a marked fibrous reaction.



Parasitisation of humans by *G. pecorum* has been described; the parasite penetrates the skin, but most larvae become encapsulated and die after penetration.

This parasitosis has been reported in horses in Spain, in the provinces of Burgos, Cáceres, Castellón, and Córdoba, and in Portugal.

Male adult flies of this species have yellow hairs and a characteristic black stripe on the back of the thorax. Females are brown with black hairs and have a very short ovijector.

The eggs (1 mm) are black, oval, and elongated and are deposited directly on plants and objects located on the ground in the vicinity of equids, and, on

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rare occasions, on their limbs. The larvae hatch in response to moisture and an increase in temperature (a stimulus produced upon swallowing of the eggs). The first-stage larvae hatch in the oral cavity and attach to the pharynx, where they remain.

The presence of the parasite does not usually cause serious alterations; although cases have been reported in which 100–250 larvae were found on the palate of horses after death, in these cases the cause of death was unclear.



Image courtesy of Dr D. Otranto

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3

Q & A
Systemic
endoparasites

Chapter 3

Q & Systemic endoparasites

Introduction

This chapter describes a heterogeneous group of important parasites that affect many animal species, although many of them have a limited impact in equids.

In particular, we will focus on infections known to cause zoonotic diseases (i.e. those that can affect humans). One example is trichinosis, which often causes outbreaks in humans in countries in which horsemeat has traditionally been consumed. Toxoplasmosis and related processes can also potentially affect human populations.

By contrast, no such risk has been demonstrated for equine hydatidosis; *Echinococcus equinus* does not infect humans, although for some time it was believed that all isolates of *Echinococcus* could give rise to hydatid cysts in humans.

The clinical cases presented here provide examples of several important pathologies of horses. Some of these processes have not yet been reported in

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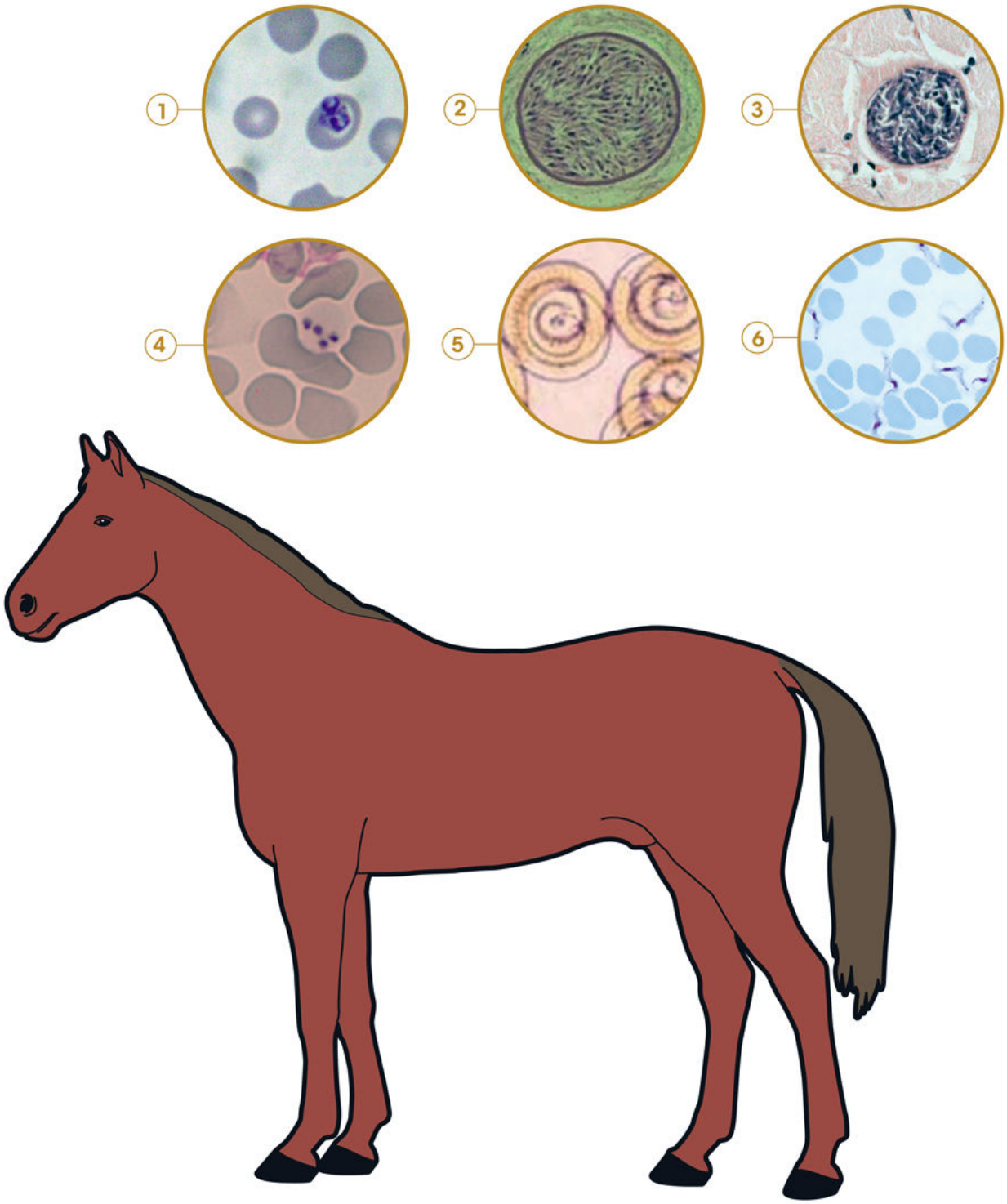
Spain, but have been diagnosed in other parts of Europe. Examples include equine protozoal myeloencephalitis, the spread of which is favoured by the trading of horses worldwide.

A section in this chapter is also dedicated to dourine, a disease that was eradicated in Spain many years ago but still persists in North Africa and other areas. It is unlikely that the prevalence of this disease will return to the levels seen during the first half of the 20th century in Spain. Nonetheless, some knowledge of this infection is warranted given that the parasite responsible, *Trypanosoma equiperdum* , cannot be morphologically distinguished from other trypanosomes such as *Trypanosoma evansi* . According to Spanish regulations, a specific certificate is required in order to export purebred Andalusian horses to the United States. This certificate must state that the animal has not spent time in any location in which certain diseases (specified in an official list), including dourine, have been reported.

Finally, infections caused by babesias and theilerias (incorrectly referred to as piroplasmosis) are of particular interest. In addition to the known consequences of these tick-borne parasitoses, piroplasmosis and other arthropod-borne infections warrant further discussion given that climate change may favour their spread, requiring the development of new epidemiological models.

Major systemic endoparasites of equids

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1.*Babesia*
2.*Neospora*
3.*Sarcocystis*
4.*Theileria*

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5.*Trichinella*
6.*Trypanosoma*

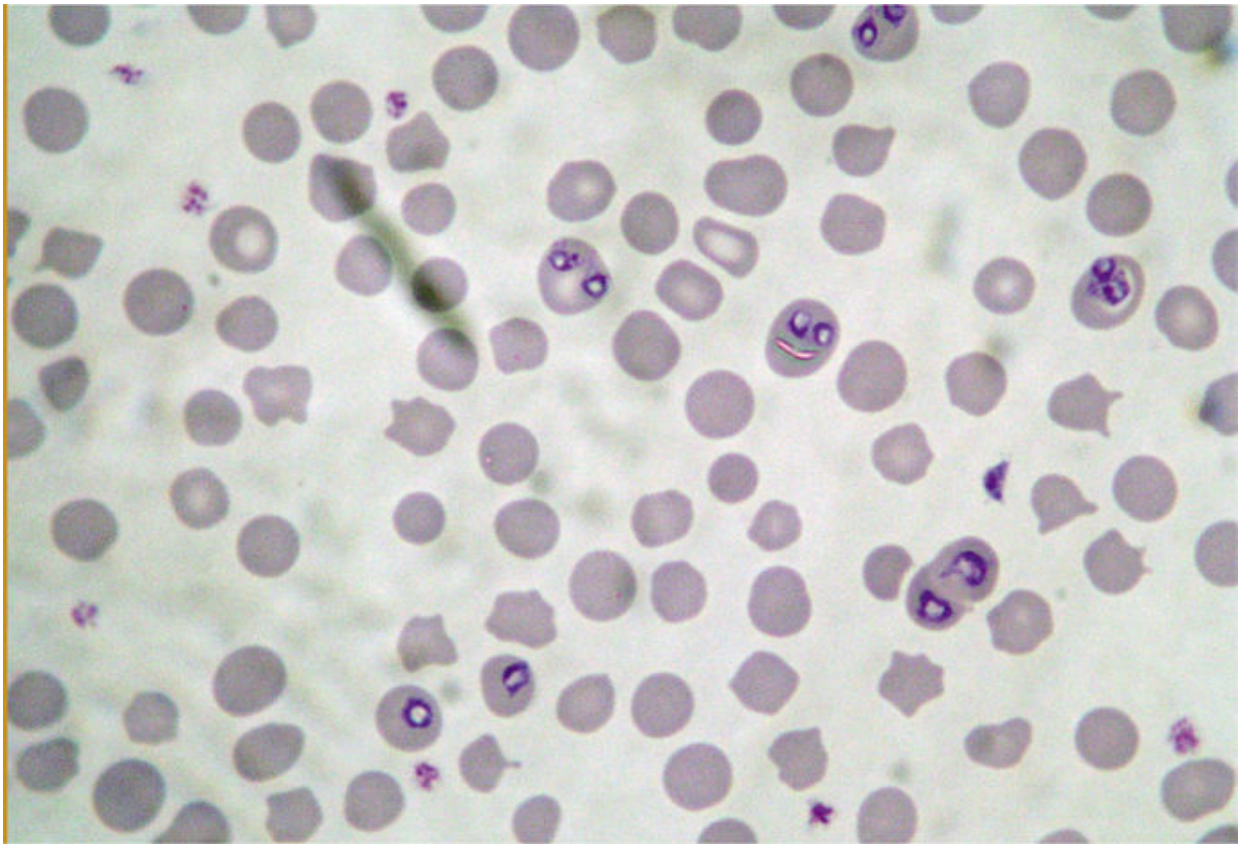
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31 What is the best method to diagnose piroplasmosis?

Blood samples are taken from an animal with fever and anaemic mucous membranes and sent to a new laboratory that performs diagnostic testing for piroplasmosis using molecular techniques (polymerase chain reaction, PCR). Serum samples are sent to the usual laboratory for serological diagnosis (indirect immunofluorescence, IFA). Conflicting results are obtained by the two laboratories: PCR indicates that the animal is positive, while the IFA result is negative for *Theileria equi*.
Since the horse has stopped eating, is prostrate, and has increasingly high peaks of fever, imidocarb is administered 4 times (4 mg/kg every 72 hours) and the analyses repeated 2 weeks later. New blood and serum samples are sent to the same two laboratories. Surprisingly, the second analysis reveals very low PCR values and very high IIF values.

1. What could explain the disparity between the results obtained using the two techniques?
2. Is this animal considered parasitised or not?
3. Is PCR more reliable than IIF?
4. What is the diagnostic value of a blood smear examination?

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1 What could explain the disparity between the results obtained using the 2 techniques?

In primary infections, once contagion has begun the parasite multiplies and antibodies against the parasitic antigens are produced. Molecular techniques are very sensitive and can detect much lower numbers of parasites than blood smear examinations. In an animal with fever, blood smear examinations and PCR can detect infection shortly after it has occurred. This may explain the positive PCR result obtained in one of the clinical laboratories.

By contrast, IFA only detects infection at later stages, when dynamic multiplication of the parasite results in sufficiently high antibody levels.

In conclusion, it is almost certain that the first blood samples sent to the laboratories for analysis were collected immediately after infection, before the initiation of an adequate immune response. PCR detected the presence of a parasite, but IFA did not.

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Traditional laboratory methods can be used to confirm the diagnosis of many veterinary diseases, and are simple, fast, and highly specific. It is advisable to consider establishing a definitive diagnosis before resorting to other techniques, the results of which require careful interpretation.

One such example is the blood smear examination. In blood smears obtained from animals with suspected disease, the presence of characteristic intraerythrocytic parasitic forms can be verified. This technique is highly sensitive in patent infections. The high rate of multiplication of *Theileria* , once it invades the bloodstream after multiplying in the lymphoid organs, gives rise to sufficient numbers of intraerythrocytic parasites to enable visualisation. However, microscopic examination of blood samples from potentially infected animals is not always sufficient to confirm diagnosis. It is usually difficult to detect piroplasms in the blood of animals with mild parasitaemia.

Fever is a key clinical sign of piroplasmosis, and must be present in order to establish a correct diagnosis. Parasitaemia is readily detectable in blood tests (PCR or blood smear), and is accompanied by peaks of fever that facilitate interpretation of the serology results.



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The results obtained in the second analysis are a direct consequence of the treatment administered. The drugs used to treat piroplasmosis, when administered according to the manufacturer's recommendations, destroy a large proportion of the parasites. Therefore, microscopy is not recommended in animals recently treated with an effective drug, given the likely scarcity of piroplasms in the blood. However, assuming that infection occurred shortly before the first analyses were performed, the time elapsed between collection of the first and second samples would be sufficient to allow the levels of antibodies detectable by IFA to increase enough to produce a positive result, with a very high titre.

In summary, it is important to have adequate knowledge of the techniques used to confirm diagnosis and to properly interpret the results.

2 Is this animal considered parasitised or not?

Yes, the animal was parasitised at both timepoints. In *T. equi* infections, pathogenic alterations initially affect lymphocytes and later, with increasing parasitaemia, erythrocytes.

Once the host overcomes the infection, it develops immunity that protects against future infections. Consequently, it becomes a latent or inapparent carrier; parasites locate themselves in different organs and cannot be detected in peripheral blood. Seroprevalence persists in these animals for years, and in some cases for life. This state of equilibrium in parasitised animals can be disrupted by stressors, treatments with immunosuppressive effects, or coinfections caused by pathogens with immunosuppressant effects.

3 Is PCR more reliable than IFA?

The suitability of a given technique depends on several factors. Some techniques are very specific and sensitive at certain phases of the infection process. Others are sensitive and can rapidly detect infection, but are less specific, and can produce false positives caused by cross reactions with pathogens that resemble the causative agent.

Polymerase chain reaction (PCR) can be considered a reliable technique, in that it allows rapid confirmation of the presence of the parasite. However,

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in carrier animals fever must be present to confirm a positive result obtained using PCR or a blood smear examination. Indeed, a blood smear performed and analysed by an experienced technician can be as reliable as PCR. In a study in which experimental infections were diagnosed using a range of available methods, PCR produced a weak signal only 2 days before piroplasms were detected in a blood smear. When selecting a particular technique, several factors should be considered: sensitivity, specificity, speed, and cost.

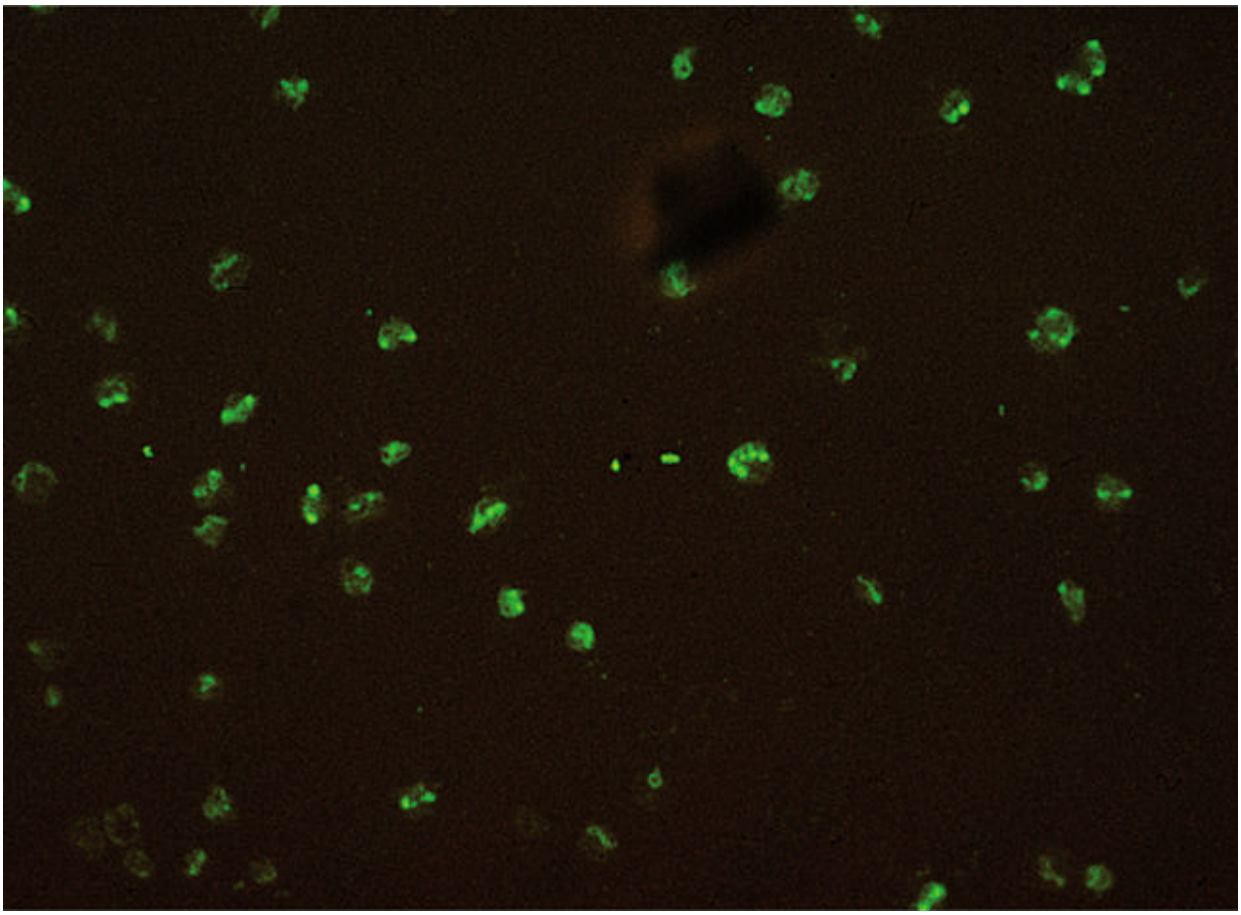
4 What is the diagnostic value of a blood smear examination?

The blood smear is a simple, rapid, and highly specific method that should always be used to confirm or rule out suspected piroplasmosis.

As described above, blood smear examination allows detection of intraerythrocytic parasitic forms in animals with suspected piroplasmosis, but in some cases can produce false negative results. In certain stages of the infection, in animals that have been treated with an effective drug, or after repeated clinical episodes, parasites can be very difficult to observe in peripheral blood smears, despite the impressive multiplication capacity of some piroplasms.

A blood smear examination is therefore of limited use in carrier animals and those without fever. Nonetheless, this is the recommended first-choice method for confirmation of piroplasmosis.

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Positive IFA. Carriers are detected by quantifying the immune response. It is essential to dilute sera before titrating.

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PCR is highly specific and sensitive for the detection of piroplasms, but for confirmation of diagnosis clinical signs compatible with a theileriosis, such as impaired performance, fever, anaemia, or icterus, must be present.

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32 Can piroplasms cause death?

Sinchán is a 10-year-old male pony who is stabled at the facilities of the Veterinary Teaching Hospital of Madrid, where he is used as a blood donor and for veterinary practical classes. He undergoes a splenectomy after a powerful kick from another horse ruptures his spleen. After 29 days, pyrexia is observed, followed by tachycardia and anaemia. Three days later, the mucous membranes are pale. Five days after fever begins the patient's condition worsens significantly. The urine is a dark brown colour. At this point it is decided to euthanise the animal and perform a necropsy. Acute theileriosis is diagnosed. To confirm the diagnosis, a foal that was donated to the centre is splenectomised and receives a transfusion of cryopreserved blood (parasitaemia, 40 %) taken from Sinchán at necropsy. The foal develops similar clinical signs and is euthanised after 15 days. Parasites are detected 7 days after transfusion and peaks of fever (40.2–40.6 °C) coincide with increased parasitaemia. On the day of euthanasia, haemoglobinuria is detected and parasitaemia exceeds 40 %.

1. How is the infection of both animals explained?
2. Do the same mechanisms underlie the pathogenic effects of *Babesia caballi* and *Theileria equi* ?
3. What lesions likely to be found at necropsy?

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1 How is the infection of both animals explained?

The majority of the world's equine population lives in areas enzootic for babesiosis. In these areas, animals are regularly infected by tick bites during the first year of life. Neonatal infections are usually inapparent, owing to the protective effect of maternal antibodies. Clinical cases are relatively rare in these areas, and most infected animals remain as inapparent carriers. If the animals are removed from contact with ticks, this protection against *Babesia caballi* is lost within 6 months. However, this is not observed with *Theileria equi* ; this parasite remains in the body and the animal remains an inapparent carrier. The extent of the immune response is key to controlling proliferation of this protozoan. Therefore, immunocompromise can result in the development of clinical piroplasmosis in previously asymptomatic animals. In both the horses described here, splenectomy was a key determinant of the fatal outcome. The first animal was a carrier of *T. equi* , and the second was infected by blood inoculation.

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2 Do the same mechanisms underlie the pathogenic effects of *Babesia caballi* and *Theileria equi* ?

No. There are important differences between these two parasites, although both can target and destroy erythrocytes, giving rise to anaemia, icterus, and fever.

B. caballi only parasitises the erythrocytes of horses. Trophozoites are 3–4 µm long and pyriform. Pairs of merozoites (3–5 µm) can also be found. Trophozoites are circular structures, while merozoites are usually located in the centre of the erythrocyte.

Immediately after infection of a horse with *T. equi* , this parasite multiplies first in lymphocytes and later in erythrocytes. The schizonts of *T. equi* can be found in the lymph nodes 12–14 days after a horse is bitten by an infected tick. The intraerythrocytic forms of *T. equi* are small (1–2 µm) and spherical, pyriform, or amoeboid (2–3 µm). The parasite divides in erythrocytes, first into two and then into four forms, which are arranged in a characteristic "Maltese Cross" pattern.

There are two main mechanisms by which piroplasms cause lesions at both the cellular and tissue levels. The primary mechanism is erythrocyte destruction as a result of parasitic multiplication. Haemolytic anaemia gives rise to anoxia and secondary inflammatory lesions in various organs, particularly the liver and kidneys. The second mechanism underlying the pathological effects of babesiosis involves electrolyte abnormalities, alterations in coagulation, complement activation, and the release of active compounds that cause vascular alterations and hypotension/shock.

Intravascular haemolysis is generally proportional to parasitaemia, which in *T. equi* can exceed 40 %. It is caused by the emergence of the protozoa from parasitised cells and by an increase in the fragility of non-parasitised erythrocytes as a consequence of surface deposition of immune complexes. Haemolysis results in anaemia and tissue anoxia, haemoglobinaemia, and haemoglobinuria (only in *T. equi*), bilirubinaemia and icterus, the release of endogenous pyrogens, and fever.

Products of the parasite's anaerobic metabolism, such as lactic acid, cause metabolic acidosis, triggering capillary dilation and vascular stasis and consequent increases in tissue anoxia.

In some severe cases of babesiosis, especially those caused by *B. caballi,* vascular phenomena predominate over anaemia, leading to death by

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hypovolemic shock. Various pathogenic mechanisms are involved. One such mechanism involves the adherence of erythrocytes to the capillary endothelium of certain organs, especially the brain. This phenomenon is mediated, in part, by the appearance on the surface of erythrocytes of button-like protuberances, the number of which is directly related to the virulence of the strain of the parasite. These protuberances can consist of parasitic antigens or of auto-antigens produced in response to the action of various parasitic proteases.



Conjunctival anaemia and icterus. Affected animals have a tendency to become suddenly immobile, and in some cases lie down and do not respond to any stimulus. Absolute anorexia and fever peaks (40 °C) are observed. Oedema affects lower body areas (limbs, head, abdomen). The faeces contain mucus and colic is common. Haemoglobinuria is uncommon. The mucous membranes are pale and icteric.

The direct consequence is obstruction of local circulation, particularly in certain organs such as the brain. This accounts for many of the nervous clinical signs observed in some cases of clinical babesiosis.

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In some cases thrombi or multiple microthrombi appear in different organs, including the lungs, renal glomeruli, and hepatic sinusoids, altering organ irrigation and function. The release of parasitic proteolytic enzymes is associated with activation of the kinin–kallikrein system. Activation of this system is observed several days before numbers of the parasite reach levels detectable in blood. This explains the occasional appearance of clinical babesiosis in the absence of elevated parasitaemia. These enzymes favour the transformation of prekallikrein, which is naturally present in the blood, to kallikrein. In addition to increasing hypotension and capillary permeability, plasma kallikrein indirectly affects haemostasis by promoting the release of bradykinin (parasympathomimetic effect) and activating coagulation factor XII, which promotes intravascular coagulation.

The consequences are peripheral vasodilatation with increased vascular permeability, blood stasis, and hypotensive shock caused by the activation of vasoactive substances and complicated by the presence of microthrombi in different organs, especially the lung and brain.

In *B. caballi* infections the main pathogenic effects are caused by the release of pharmacologically active substances that induce blood stasis, intravascular coagulation, and shock. In *T. equi* infections, pathogenesis begins in lymphocytes and subsequently affects erythrocytes with increasing parasitaemia, giving rise to severe anaemia. In both cases a marked autoimmune response is observed.

Once the host overcomes the infection it develops immunity that protects against future infections. From this point on, the host is a latent or inapparent carrier, in which parasites locate themselves in different organs and cannot be detected in peripheral blood. Seroprevalence persists in these animals for years or, according to some authors, for the rest of the animal's life. This state of equilibrium in parasitised animals can be disrupted by stressors or by treatments or interventions that have immunosuppressive effects (e.g. splenectomy).

In acute cases, marked icterus is observed throughout the carcass. The spleen is very enlarged and very dark in colour, and the liver is swollen and dark. The adipose panniculus has a striking yellowish colour.

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The acute form of the disease is characterised by prostration, tachypnoea, weakness, a rapid pulse, anorexia, and polydipsia. Affected animals develop intermittent fever and icterus is evident in the ocular conjunctiva and the palpebral, buccal, and labial mucous membranes. Haemoglobinuria lasting 1 or 2 days may be observed in acute *T. equi* infections. Affected animals usually die before they can be treated.

Numerous less specific clinical signs have been described, including bilateral vision loss, lacrimation, palpebral haemorrhage and oedema, renal failure, gastrointestinal disorders such as colic, locomotion problems, especially paralysis of the posterior third, and oedema in ventral areas, all of which are related to the pathogenesis of the disease.

In Spain, the disease course is usually chronic and is characterised by nonspecific clinical signs, including weight loss, weakness (especially of the posterior third), and a predisposition to other concomitant processes.

Piroplasmosis caused by *T. equi* tends to be more severe, and is more often accompanied by fever, anaemia, and haemoglobinuria.

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3 What lesions are likely to be found at necropsy?

The anatomopathological picture varies in intensity and depends on the presentation (acute vs chronic). Lesions include generalised icterus, haemorrhages in the myocardium, liver congestion, dark colouration of the renal medulla, and the presence of abundant, deep-red urine in the urinary bladder. The most striking histopathological alterations are observed in the liver, kidneys, heart, and lungs, in which congestion, haemorrhage, tissue degeneration, and necrosis are observed.

Hepatic hypoxia can result in hepatocyte degeneration, giving rise to fatty infiltration and vacuolisation of the liver cells. The kidneys exhibit necrosis of the epithelial cells in the proximal tubules, as well as venous congestion, especially near the medullary cortical junction. Pulmonary oedema is common, and precedes pneumonia. Histology reveals distension of the capillaries, which are replete with blood. According to some authors, erythrocytes in the lung are more parasitised than those in peripheral blood. In acute presentations, ecchymosis is observed in the epicardium and endocardium, and in the myocardium in severe cases.

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33 Why is piroplasmosis not fatal in horses in endemic areas?

Castelo is a dark chestnut male Thoroughbred of 5 years of age who trains and competes at the Madrid Hippodrome. After surgery to remove several polyps from the glottis, he is moved in August to rest for 3 months in the stud in Aranjuez where he was raised as a foal. There, he shares a pasture with other horses. Surprisingly, 1 month after arrival, Castelo develops recurrent intermittent fever, with peaks of 41 °C, accompanied by mild tachycardia and congestive mucosa with conjunctival petechiae, and diarrhoea that lasts 1 day. Blood tests indicate mild anaemia (PCV = 25 %; Hb = 8.3 g/dl; RBC = 4.6/10 µl; bilirubin = 5.9 mg/dl) and urine tests reveal no abnormalities. Piroplasms are observed, but only at moments of peak hyperthermia. All animals in the stud undergo blood tests. Mild parasitaemia is detected in two animals in which no clinical signs are evident. The seroprevalence of *Babesia caballi* and *Theileria equi* is 7.5 % and 45 %, respectively.

1. Why did Castelo become ill at the stud?
2. Why did none of the animals in the stud develop clinical signs?
3. How are piroplasmids transmitted?

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1 Why did Castelo become ill at the stud?

Age is a very important determinant of susceptibility to these diseases, since the foals (up to about 8 months of age) are protected by antibodies acquired via the mother's colostrum. This may explain why Castelo avoided illness while still a foal. Equids that live in enzootic areas in which the vector and parasite are also found develop a very durable form of immunity (premunity or non-sterile immunity) to piroplasmosis.

For this reason acute presentations are very rare in enzootic areas, and are only observed in cases in which the host/parasite equilibrium is disrupted. Castelo's removal from an enzootic area and his stabling in a box at the stud prevented any contact with ticks that could re-inoculate him, resulting in clinical disease.

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2 Why did none of the animals in the stud develop clinical signs?

On stud farms it is common for animals to be regularly infected by tick bites during the first year of life. Contagion of a receptive host occurs via inoculation by another invertebrate host (a tick) of the family *Ixodidae* or as a result of transfusions or injections using contaminated material.

Because of the protection afforded by maternal antibodies, neonatal infections are usually inapparent. In enzootic areas, clinical outbreaks are relatively rare, and infected animals remain as inapparent carriers.

The majority of piroplasmosis infections result in the establishment of strong acquired immunity once the infection is overcome. If continuous contact with the parasite (antigen) is maintained, the immunity may be almost permanent. This immunity is intraspecific, and does not protect against the entry and partial reproduction of parasites that are inoculated later, particularly those of distinct strains. In natural conditions, one infection can overlap another without the appearance of clinical signs. However, infection with a different strain can result in transient fever and mild parasitaemia.

This is why in enzootic areas native cattle that are in continuous contact with the parasite rarely become ill. Those most at risk are young or recently introduced animals.

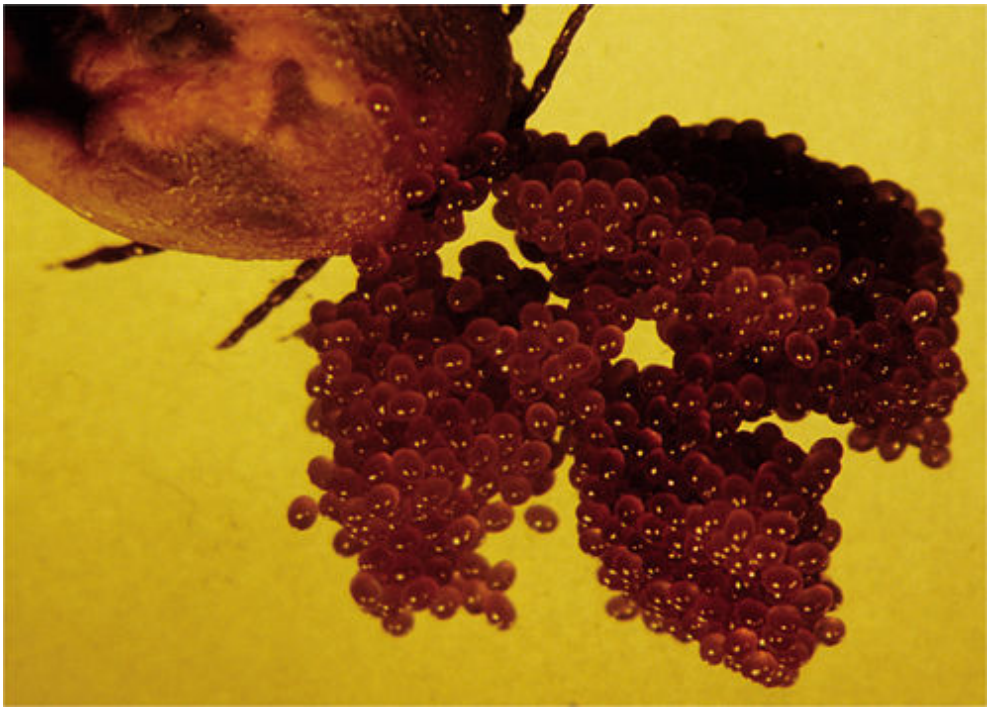
If contact with the parasite ends, either as a result of indiscriminate treatments or variations in the tick population, the animals lose their immunity and within about 6 months become as susceptible to clinical disease as animals that have had no previous contact with the parasite.

The ability of the host to respond to infection is as important as continuous contact with the antigen. In enzootic areas, the sudden appearance of clinical cases is directly related to stressors such as foaling, starvation, or other pathologies.

Another very important factor is the species and population density of intermediate hosts. The biological cycle of ticks consists of two distinct phases: the free-living phase is common to all species and encompasses the period between the descent of the gravid and satiated female from the host to lay her eggs and the attachment of the hatched larvae to another host. This phase is significantly influenced by environmental factors such as

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temperature, rainfall, and relative humidity, and therefore considerable regional differences are observed throughout Spain.



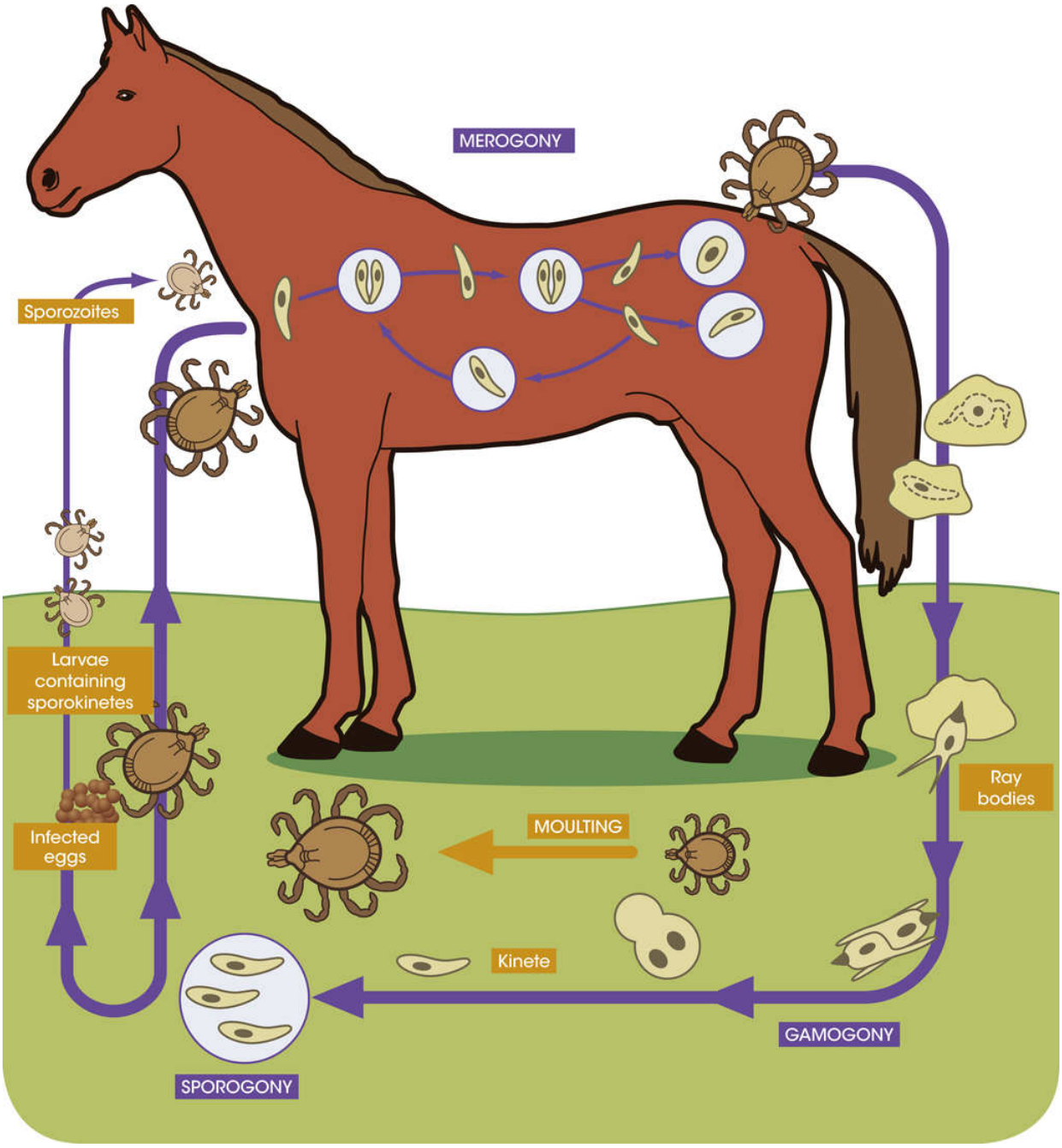
Babesia can infect the ovaries of ticks, resulting in disease transmission by the subsequent generation of ticks. Each parasitised female tick can infect many horses.



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Theileria does not infect the ovaries, only the salivary glands. Its vectorial capacity is more limited, since for each parasitised larva (top left) or nymph (top right), only one horse will be infected.

Life cycle of *Babesia*



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3 How are piroplasms transmitted?

In natural conditions, transmission of piroplasms occurs exclusively via the bite of infected ticks.

Thus, in parasitological terms, ticks are considered the definitive hosts. Sexual reproduction of piroplasms occurs in ixodids, or hard ticks, only some of which are suitable for reproduction of the parasite. The sexual (gamogonia) and asexual (sporogony) phases occur in the tick. The main tick species that mediate transmission are those of the genera *Dermacentor, Hyalomma* , and *Rhipicephalus.*

Once the parasite is ingested by the tick, the sexual phase of its biological cycle begins. While most of the parasites present in erythrocytes die immediately after the blood is ingested by the tick, others emerge from the erythrocytes and differentiate, giving rise to so-called *Strahlenkorper* or ray bodies. Two to 4 days after the ingestion of blood, the gametes fuse to form a spherical zygote containing a single nucleus. This elongates to form a new, mobile zygote called a kinete.

Kinetes invade the mucosa of the tick's intestinal epithelium, where they reproduce asexually (sporogony) to produce sporokinetes. Sporokinetes in turn invade other cells of the tick, including the Malpighian tubules, muscle fibres, and ovarian cells, where asexual reproduction occurs, and may continue up until the death of the tick.

When the oocytes become infected, the parasites remain dormant until the egg develops and the larva hatches. The sporokinetes continue to multiply in the tissues of the larva. After the larva ingests blood the sporokinetes invade the salivary glands to continue their biological cycle. Sporozoites, which can infect the vertebrate host, are produced in the salivary glands. Several thousand sporozoites are formed in each cell of the acini of the salivary glands, and mature within about 5 days. These sporozoites can therefore be inoculated into a new host while the larva continues to feed (21–28 days). Once the ovaries of a tick are infected, the next two or three generations of ticks produced will be infective. This type of transovarial transmission is typical of *Babesia* spp.

During their development ticks undergo metamorphosis twice, transforming from larva to nymph and from nymph to adult. Piroplasms ingested in one phase of the cycle can continue their development in the next phase and sustain the infection of the invertebrate host. In such cases, the

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parasite reproduces directly by infecting the salivary glands in one phase. As the next phase develops and begins to feed, sporozoites are produced and are transmitted to the new vertebrate host during feeding. This is known as transstadial transmission.

While transmission of *B. caballi* to the invertebrate host can be transovarial or transstadial, *Theileria equi* transmission is exclusively transstadial. In the case of *B. caballi* , tick larvae are born infected, but horses tend to be infected by nymphs or by adult ticks (after ingestion of infected blood by the nymphs). Horses are poor hosts of tick larvae.

Only nymphs and adults are involved in the transmission of *T. equi* . Both females and males participate; the latter ingest multiple blood meals and change hosts frequently.

Parasitisation by *T. equi* is much more frequent (3–6 times more) than that by *B. caballi* . Identification of the aetiological agent is extremely important. Depending on the causative agent, the frequency of presentation and severity of the clinical picture can differ considerably.

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34 Why do animals infected by *Theileria equi* continue to carry the disease?

In a stud, sera are collected from several animals destined for export and are sent for laboratory analysis. In addition, information is requested on the prerequisites for the control of piroplasmosis and the most appropriate type of analysis to perform to avoid problems during the export process.

1. What is the technique of choice in the US?
2. How useful are PCR, IFA, and blood tests when performed 3 months before export?
3. What recommendations can a veterinary surgeon give regarding animals that test positive or negative?

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1 What is the technique of choice in the US?

Complement fixation has been used for a long time with good results. It is a highly specific, but not particularly sensitive, technique that allows identification of infected animals. In recent years ELISA has been used. This is a much more sensitive technique, which allows better quantification of antibodies and detection of disease carriers.

2 How useful are PCR, IFA and blood tests when performed 3 months before export?

Before exporting a clinically “healthy” animal to one of these countries it is recommended to evaluate blood antibody levels approximately 3 months

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beforehand using indirect immunofluorescence (IFA) or enzyme-linked immunosorbent assay (ELISA). Like ELISA, IFA is a sensitive technique that can be used to detect carrier animals.

Many animals infected by *Theileria equi* remain as asymptomatic carriers. A sudden drop in immune function can lead to the appearance of clinical signs. In these animals, the levels of antibodies in the blood can produce a positive test result, depending on the technique used.

3 What recommendations can a veterinary surgeon give regarding animals that test positive or negative?

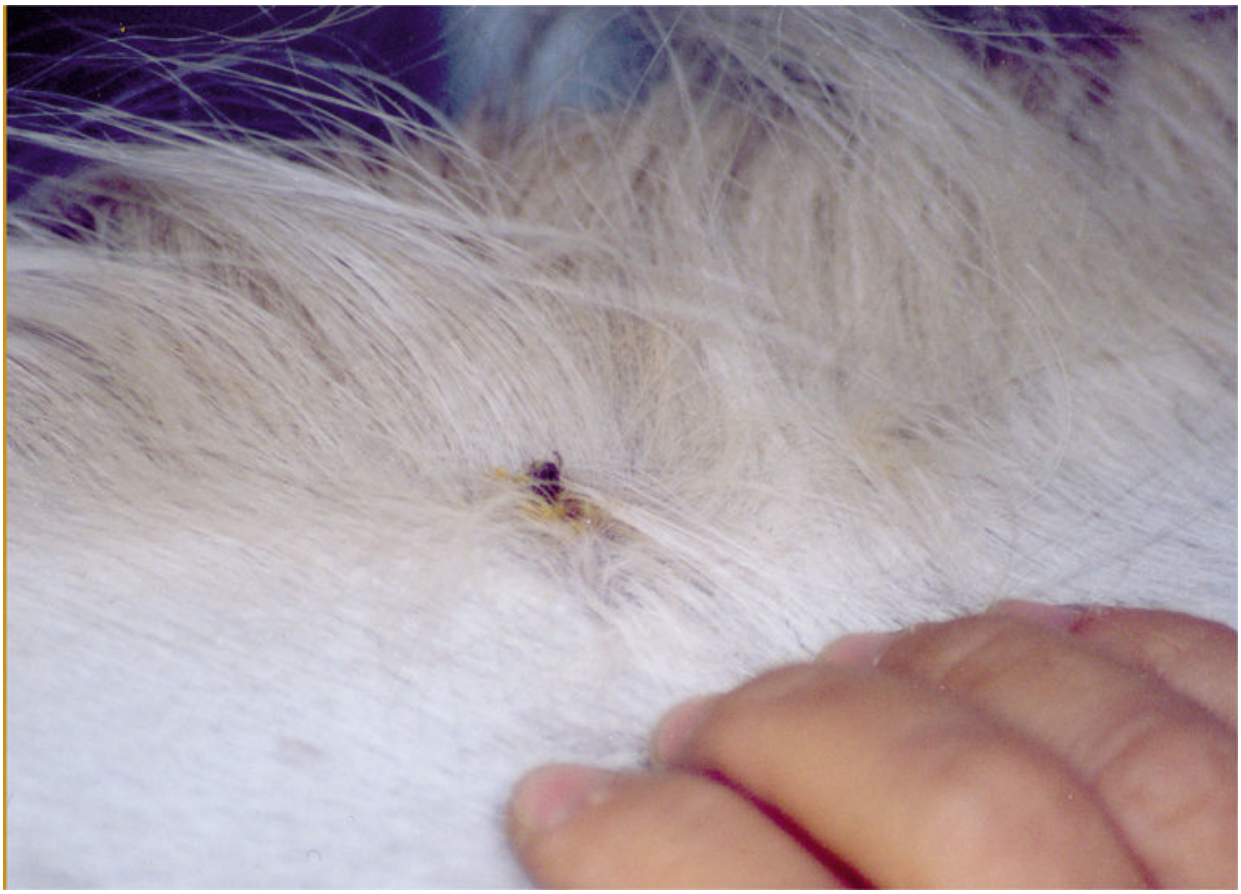
If the result is positive, the animal can be treated with a product of proven efficacy to try to reduce the antibody titre. If the result is negative, the animal should be restricted to a safe area and any contact with ticks that could transmit the parasites should be avoided. The tests that are currently used are so sensitive that it is recommended not to export animals that test positive. In Spain, where seroprevalence for *T. equi* is approximately 40 %, this has a serious negative impact on the trade and sale of animals.

There are few alternatives, and the only realistic recommendation is to transfer breeding to areas with a low prevalence of piroplasmosis, such as northern Spain.

Eradication of ticks from a farm is unrealistic; in endemic countries it is practically impossible to avoid the introduction of parasitised animals from other regions or the movement of wild animals that also serve as hosts for ticks. Another alternative is to promote the trade of animals with other countries in which these diseases are also endemic.

In Spain, one of the main vectors of *Theileria equi* is *Dermacentor reticulatus,* a tick found mainly at the base of the mane.

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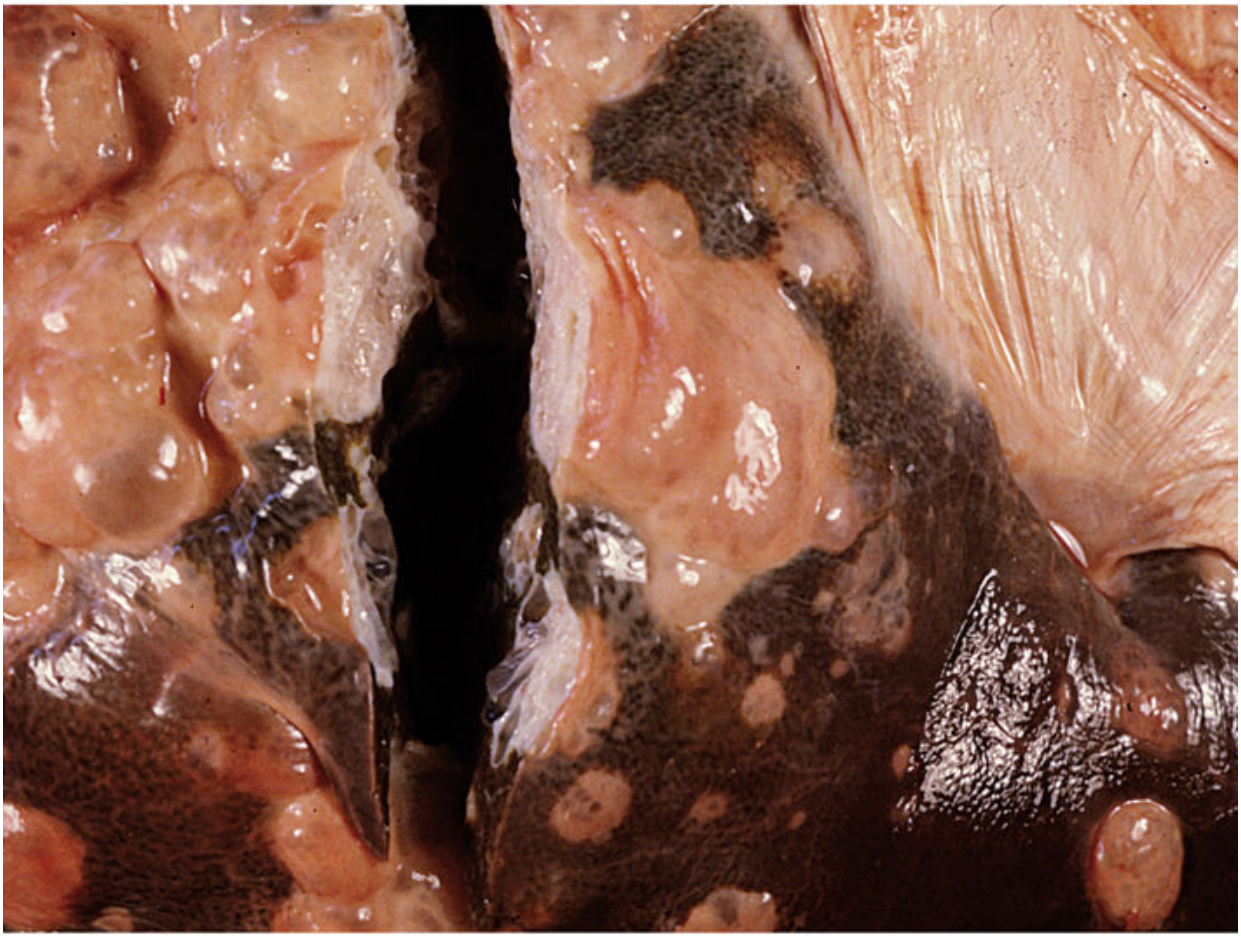
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35 Are hydatid cysts of horses the same as those found in other species?

During a routine inspection in a slaughterhouse, several horse livers are condemned owing to the presence of cestode larvae, and are sent to a veterinary faculty for use in practical classes.

1. What is equine metacestodosis?
2. Is equine hydatidosis a common parasitic disease?
3. Are *Echinococcus equinus* infections considered zoonoses?
4. Why are organs with hydatid cysts confiscated?

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1 What is equine metacestodosis?

Metacestodes are the larval stages of certain cestodes, such as those found include hydatid cysts. They are referred to using binomial nomenclature. The term hydatidosis is also used to describe the zoonosis caused by cestodes of the genus *Echinococcus* .

The only metacestode that can develop in equids is that found in hydatid cysts. There are no records of the presence of coenuri or cysticerci in equids. In equids, hydatid cysts are located mainly in the liver and lung. These small cysts are caused by a strain of *Echinococcus granulosus* that is now considered a species in its own right: *Echinococcus equinus* .

Hydatid cysts are typically unilocular and subspherical, and grow expansively and slowly, reaching a variable size (several cm) within less than a year. In horses, they can persist for up to 16 years.

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2 Is equine hydatidosis a common parasitic disease?

The prevalence of hydatidosis has decreased in many parts of the world thanks to the establishment of campaigns to combat this parasitic disease and the obligation to declare diagnosed cases, among other factors. Spain is no exception: data, although scarce, show that prevalence is continually decreasing. Between 1982 and 1984, 6.9 % of livers and 1.7 % of lungs from horses were condemned due to the presence of hydatid cysts. Figures recorded in other countries vary considerably. For example, studies have reported the detection of hydatid cysts in 1 % of horses in Sicily (Italy) and in 10 % of 160 donkeys in Egypt.

3 Are *Echinococcus equinus* infections considered zoonoses?

Almost four decades of field work and laboratory experience has revealed marked phenotypic variation in isolates of *Echinococcus* recovered from different intermediate hosts.

Molecular techniques have demonstrated several genetic differences within the genus *Echinococcus* , enabling identification of several genotypes. Based on the differences observed within the genus, the equine and bovine “strains” *Echinococcus equinus* and *Echinococcus ortleppi* have been raised to the species level.

E. equinus , which affects donkeys and horses, does not infect humans.

4 Why are organs with hydatid cysts condemned?

The parasitic species of equids (*E. equinus)* lacks the ability to infect humans. However, even if this were not the case, affected organs are condemned for aesthetic and sanitary reasons. These organs have an off-putting appearance that precludes their sale, at least in developed countries. Nonetheless, the ingestion of protoscoleces by an intermediate host (IH), such as a human, has no deleterious effects; the biological cycle simply ceases.

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Echinococcus granulossus. The rostellum consists of multiple rows of hooks, which anchor the parasite to the intestinal crypts while the suckers attach to the villi. This species rarely has more than three or four proglottides, since the last gravid proglottis is released once it is full of eggs.



Taenidae egg. Eggs of the family *Taenidae* are easily identified by the presence of marked peripheral striations and three pairs of hooks. Eggs of *Echinococcus* are indistinguishable from those of *Taenia* . Horses are parasitised while grazing in pastures contaminated with the faeces of infected dogs.

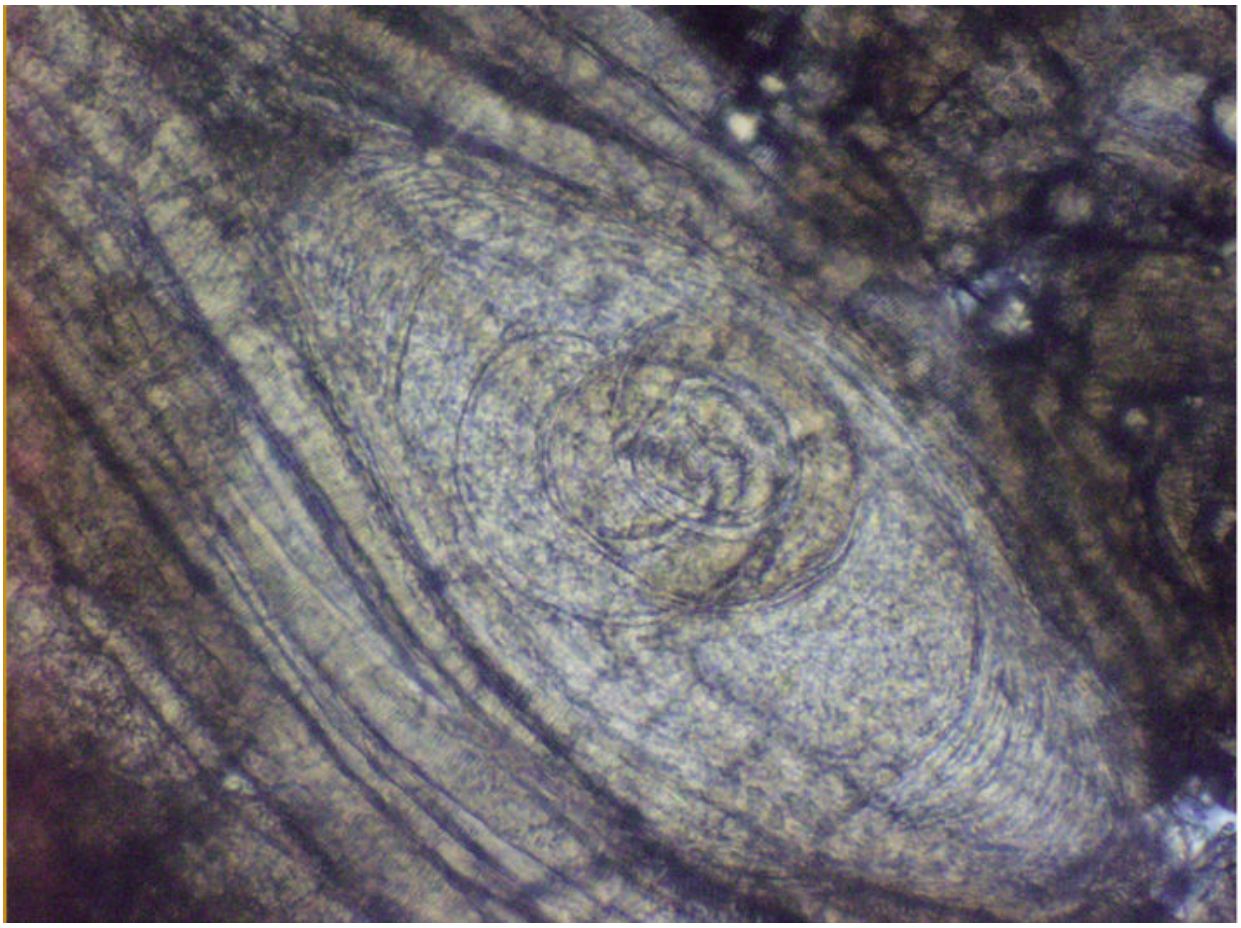
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36 Can horses transmit trichinellosis?

A veterinary surgeon in a slaughterhouse is asked to test for trichinella in five horse carcasses destined for sale. The techniques required to perform this test in pigs are well known by all inspectors of swine slaughterhouses. However, when asked to perform this analysis in a horse carcass, it is important to select the appropriate technique.

1. Is *Trichinella* infection common in horses?
2. Equids are herbivores. How can they become infected?
3. What method should be used for the diagnosis of infections caused by *Trichinella* ?

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1 Is *Trichinella* infection common in horses?

In the last 25 years, there has been an increase (in some European countries) in the number of cases of trichinosis caused by the consumption of horsemeat. The increase in international trade poses a risk in practically all countries. Therefore, the EU has established standards for the control of pork and horsemeat traded between member countries.

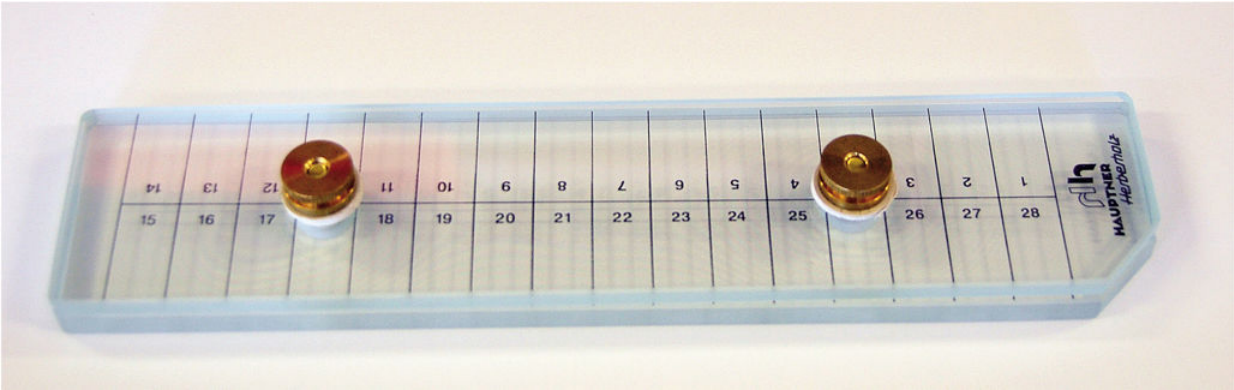
With the increase in international trade, and the consumption of pork products in fast food, the risk of outbreaks of trichinosis has increased in almost all countries. Consequently, some countries have introduced specific rules regulating the importation of pork or pork products.

Trichinosis is a serious disease caused by several species of *Trichinella,* and can give rise to acute and chronic processes in affected individuals that cause diffuse weakness or even death. This disease has both psychological and physical consequences. In recent decades, the majority of trichinosis

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outbreaks in developed countries have been the result of the consumption of meat from wild animals infected mainly by *Trichinella nativa* , *Trichinella murrelli,* or *Trichinella* T6. In some Western European countries, the large increase in trichinosis cases in the last 25 years can be mainly attributed to the consumption of horsemeat imported from Eastern European countries.

Factors implicated in these outbreaks include specific human eating habits as well as certain culinary specialties (e.g. *steak tartar*) made with raw horsemeat. The outbreaks of the 1990s led to several changes, including the substitution of horsemeat with other types of meat (e.g. beef).



Compressorium for the inspection of meat for *Trichinella* . It is uncommon to find *Trichinella* cysts in the muscle tissue of equids. For these to develop horses must ingest meat or dead rodents from the bottom of the feeder, or carrion in the field.

Trichinellosis is distributed worldwide, with the exception of Antarctica. This cosmopolitan distribution, together with certain gastronomic habits, are the main factors that favour human infections, both in industrialised and less developed countries. The prevalence of the disease is due to a lack of veterinary control and an increase in the hunting and consumption of wild animals that act as reservoirs of *Trichinella* .

While over a dozen species have been described, the following are the most important members of the genus in terms of their impact in horses:

- ***Trichinella spiralis*** is a cosmopolitan species found especially in temperate and equatorial zones. It has spread across many continents owing to its high infective capacity in pigs and in certain synanthropic animals such as the rat. It preferentially infects domestic and wild boar (*Sus scrofa*), synanthropic animals such as rats, armadillos, cats, and dogs, and many wild carnivores. Given their herbivorous diet, horses are not considered

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common hosts. However, the potential of *Trichinella* to infect horses has significant implications for human health, especially in the EU.

- ***Trichinella britovi* and *Trichinella* T8** . *T. britovi* is responsible for infections in wild carnivores in temperate areas of the Palearctic region, from the Iberian Peninsula to Kazakhstan, Iran, and Turkey. Epidemiological and molecular studies have demonstrated the presence of this species in wildlife in several African countries along the Mediterranean coast. Human *T. britovi* infections, caused by the consumption of pork, game, and horsemeat, have been reported.
- ***Trichinella murrelli*** is responsible for infections in wild carnivores in the Nearctic region. It has little infective capacity in pigs and rats. In 1985, importation of contaminated horsemeat from Connecticut (USA) to France resulted in an outbreak of human trichinellosis that caused two deaths.

2 Equids are herbivores. How can they become infected?

Despite epidemiological studies of the thousands of cases of human trichinosis acquired through the consumption of horsemeat in France (2,296 cases in 8 outbreaks) and Italy (1,031 cases in 6 outbreaks), it remains unclear how the horses became infected in natural conditions. However, data suggest a relationship between the infection of horses and the infection of swine. In the three documented cases in which *Trichinella* species normally found in wildlife (*T. britovi* and *T. murrelli*) have been detected in horses, a link to infected wild animals or fur animals has been postulated.

Feeding of horses with products of animal origin is a common practice in many countries, including those in which the infection has been diagnosed in equids, such as Poland, Romania, and Serbia. The increase in outbreaks of human trichinellosis in France and Italy in the 1990s and the detection in slaughterhouses of horses infected with *Trichinella* coincided with a peak in *Trichinella* infections in domestic pigs.

To date only 18 horses infected with *Trichinella* have been detected in Spanish slaughterhouses.

The freezing of horsemeat was considered an acceptable means of destroying *Trichinella* larvae present in muscle tissue, but recent

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experimental studies of ponies infected with *T. spiralis, T. britovi* , or *T. pseudospiralis* suggest that *Trichinella* larvae survive freezing for several weeks and retain their infective capacity. However, more research is clearly needed, as other studies have shown that the larvae of *T. spiralis* present in naturally infected horses do not survive freezing for 24 hours.

3 What method should be used for the diagnosis of infections caused by *Trichinella* ?

There are several means of diagnosing infections caused by *Trichinella* spp., which are summarised below:

- A trichinelloscope can be used to rapidly demonstrate the presence of muscular cysts of *Trichinella* spp. Oat-flake-sized samples are collected along the length of the muscle fibres of different muscle bundles (the pillars of the diaphragm and the masseters). These samples are then placed between two compression plates and observed under a stereomicroscope (50–80× magnification).
- Acid-pepsin digestion releases *Trichinella* larvae from muscle tissue. Pieces of muscle from selected areas are macerated and 5 g of muscle tissue is mixed with a digestive solution consisting of 200 ml of water at 40 °C, 2 g of pepsin, 1.8 g of crystallised NaCl, and 1.4 ml of concentrated HCl. The mixture is incubated at 37–40 °C, with agitation, for approximately 1 hour, filtered through a double filter (pore diameters of 0.125 mm and 0.063 mm), and the *Trichinella* spp. larvae collected in the second filter. This second mesh is then washed and the larvae collected in a petri dish. Specific regulations for the control of *Trichinella* in meat are stipulated in the Official Journal of the European Communities (OJEC) No. 2075/2005.

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Muscle samples from several animals can be digested together using devices that automatically administer the necessary amounts of digestive solution at the appropriate temperature to digest muscle tissue and release larvae from their cysts.

Larvae of *Trichinella* spp. obtained by artificial digestion.

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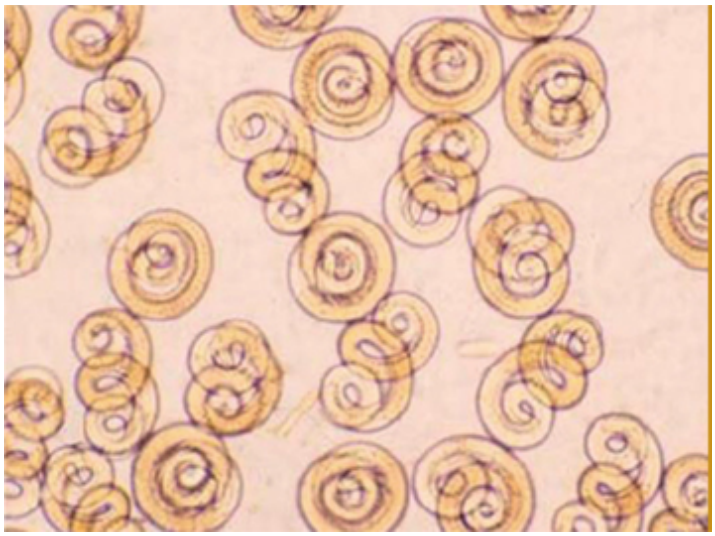


Image courtesy of Dr A. Martínez.

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37 What is dourine?

A “special certificate” from the Government of Spain is required to export purebred Andalusian horses to the USA. In the case of males of over 731 days of age reared in Spain, official veterinary services must certify that the animal has not been in any location where any one of several specified diseases, including dourine, has been detected in the 60 days prior to export. Although dourine has been eradicated in Spain and does not pose a problem, cases do occur in very nearby countries, including Morocco.

1.What is the aetiological agent and how is it transmitted?
2.How are animals with dourine identified?
3.How is a dourine outbreak confirmed?
4.How was this disease eradicated in Spain?

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1 What is the aetiological agent and how is it transmitted?

Dourine is produced by a flagellate called *Trypanosoma equiperdum* . Strains of variable pathogenicity have been described, although some hypotheses suggest that *T. equiperdum* does not exist as a species and that dourine is the result of the specific immune response to either *Trypanosoma brucei equiperdum* or *Trypanosoma evansi* . Recent studies have proposed that *T. equiperdum* and *T. evansi* are subspecies of *T. brucei* .

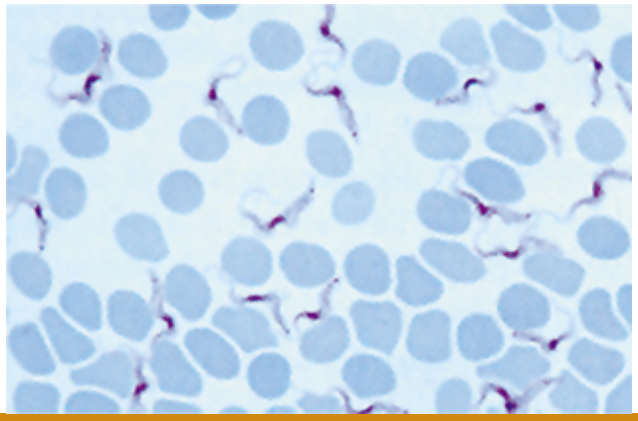
Infection of geldings or foals is attributed to the workers who care for them or the material they use (e.g. cleaning sponges). Infection of suckling foals has been linked to the presence of trypanosomes in milk.

This parasite is a fusiform protozoan of 16–35 µm, with a slightly off-centre nucleus that occupies the entire width of the cytoplasm. Volutin

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granules in the cytoplasm lend it a grainy appearance. The wide undulating membrane extends almost 7 µm beyond the anterior end.

Dourine can affect other animals such as dogs, rabbits, and rodents.



Trypanosoma equiperdum. The aetiological agent of dourine is morphologically similar to other equine trypanosomes that are considered highly pathogenic, including *Trypanosoma brucei* in Africa and *Trypanosoma evansi* and *Trypanosoma equinum* in Asia, North Africa, and South America. These are slender parasites with a partially attached flagellum. *Trypanosoma brucei* displays some degree of pleomorphism, and has a more rounded shape, measuring about 25 µm, while *Trypanosoma equinum* lacks a kinetoplast.

2 How are animals with dourine identified?

Clinical signs appear a few weeks or even months after infection. They vary in intensity, and relapse can result in death.

Dourine is characterised mainly by inflammation of the genitals, the formation of cutaneous plaques, and nervous signs. Intensity varies depending on the strain responsible for the infection, the nutritional status of the animal, and the presence of various stressors.

The first signs that appear are mucopurulent discharge and oedema of the genitals that can extend to the perineum, abdomen, and mammary glands. Mares frequently exhibit vulvitis, vaginitis with polyuria, and signs of discomfort and depigmentation in the genital region, perineum, and udders. In males mucopurulent discharge is observed in the urethra, as well as paraphimosis and preputial oedema that can extend to the scrotum, perineum, abdomen, and thorax.

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In both mares and stallions oedema of the genitals can disappear and reappear. Thickening of the affected area is exacerbated with each relapse. Vesicles and ulcers appear, and heal leaving white scars known as “toad spots”. Infections caused by highly virulent strains can result in abortions.

Skin lesions resembling oedematous “patches”, wheals, or plaques (silver dollar plaques) measuring up to 10 cm in diameter and 1 cm thick, may be observed, particularly on the flanks. These wheals remain for 3 to 7 days and are pathognomonic, although they are not produced by all strains of *T. equiperdum* .

Nervous signs may be observed shortly after the appearance of genital signs, or several months later. Affected animals are restless, increasingly weak, and show signs of incoordination and eventual paralysis. In some equids, facial paralysis is unilateral.

Conjunctivitis and keratitis are common, and are sometimes the first signs detected. Other clinical signs include anaemia, intermittent fever, and progressive organ deterioration that predisposes affected animals to other conditions.

The course of the disease is variable. Some strains give rise to mild, chronic clinical presentations that last for years, while others produce acute presentations of 1 or 2 months’ duration, although in some cases death can occur in just over a week.

3 How is a dourine outbreak confirmed?

The key clinical signs of suspected dourine are genital and neurological. The presence of wheals or plaques is pathognomonic of this disease. Clinical diagnosis is difficult in the initial stages of the disease.

Although there is the possibility of establishing a clinical diagnosis, other infections such as coital exanthema, surra, anthrax, equine infectious anaemia, equine viral arteritis, and purulent forms of endometritis such as contagious equine metritis must be ruled out.

Definitive diagnosis of dourine is established based on the clinical signs, the serological test results, and identification of the parasite responsible for the process.

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4 How was this disease eradicated in Spain?

Control of this trypanosomosis is based on clinical examination, specific serological tests, and the euthanasia of sick/infected animals.

Efforts made since the last decades of the 19th century in multiple European countries in response to dourine outbreaks have enabled tight control of the disease. The only official, internationally recognised test to establish diagnosis is the complement fixation test, although there has been some disagreement regarding the results produced. While this test provides very good results and has facilitated the eradication of dourine, it can produce false positives in mules and donkeys. Other techniques used include ELISA, counter-immunoelectrophoresis, agar immunodiffusion, and agglutination. Cross-reactions can occur with “old world” trypanosomes (*T. brucei* and *T. evansi*). Immunoblotting allows differentiation between piroplasmosis, dourine, and glanders.

Definitive confirmation of the diagnosis requires identification of the parasite, although *T. equiperdum* is quite difficult to observe. The presence of trypanosomes can be demonstrated in smears of lymph node aspirate, vaginal mucus (obtained 4–5 days post-infection), or fluid obtained from cutaneous plaques or wheals immediately after their appearance. In exceptional cases, parasitic forms may appear transiently in the blood. In all cases, the number of trypanosomes is always quite low.

It should be noted that *T. equiperdum* cannot be distinguished morphologically from *T. evansi* . However, owing to its speed and safety, microscopic examination is recommended. According to some publications, microscopy allows positive diagnosis in 60.7 % of cases.

The successful application of detection methods, together with strict sanitary measures and adequate government investment, encouraged owners to voluntarily euthanise seropositive animals, enabling eradication of this disease in Spain by the middle of the 20th century.

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Dourine is caused by a flagellated protozoan, *Trypanosoma equiperdum,* which is transmitted during copulation without the need for an arthropod vector. It therefore affects adult stallions and breeding mares, particularly those that breed naturally.

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38 Can horses transmit toxoplasmosis?

Diagnosed encephalomyelitis in equids is included in the list of notifiable diseases in the most recent bulletin of the World Organisation for Animal Health (OIE). During postmortem examination of an animal with motor incoordination and nonsymmetric muscular atrophy of the posterior third, examination of the bone marrow reveals the presence of a large number of small organisms that resemble the tissue phases of *Toxoplasma gondii.*

1.How do horses become infected with toxoplasmas?
2.Can horses transmit infection to humans?
3.How does *Toxoplasma* differ from *Neospora* ?
4.How is the aetiological agent confirmed?

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1 How do horses become infected with toxoplasmas?

Infection with *Toxoplasma gondii* is very prevalent in humans and in some animals around the world. Although the consequences of parasitisation were well known, before the discovery of the coccidian cycle of *T. gondii* it was assumed that coccidia had a narrow host specificity and specific localisation, and were transmitted via the faeco-oral route.

However, it is now known that transmission occurs via several routes. Congenital transmission is not sufficiently frequent to account for the widespread distribution of the infection. For over half a century it was thought that transmission occurred via the ingestion of undercooked meat. The fact that cysts of *T. gondii* can resist the action of proteolytic enzymes supported this view. However, neither congenital transmission nor cannibalism or predation account for the infection of herbivorous animals or vegetarians.

The first case of fatal acute visceral toxoplasmosis was diagnosed in a 4-month-old dog at the beginning of the 20th century. *Toxoplasma* was not

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isolated from livestock until the middle of the 20th century. Following the discovery of two new parasites, *Neospora caninum* and *Sarcocystis neurona* , which were previously confused with *T. gondii,* the distribution of *T. gondii* in different hosts became better understood. We now know that cattle and horses are resistant to clinical infection by *T. gondii* , that *N. caninum* is one of the most common causes of abortions in cattle worldwide, and that *S. neurona* causes fatal encephalomyelitis in horses in the Americas. The low prevalence of *T. gondii* infection in horses (in some studies only 1 % of sera samples have tested positive) suggests that horses are more resistant than sheep and pigs, or are less frequently exposed to infection.

2 Can horses transmit infection to humans?

Meat-producing animals are an important source of *T. gondii* infections for humans, owing to the presence of cysts in muscle tissue. Tissue cysts develop as early as 6 or 7 days after contact with the parasite, regardless of whether it has been induced by oocysts or tissue cysts, and generally persists for the lifespan of the infected animal. However, the location and number of tissue cysts that form in the host vary depending on the species of intermediate host (IH). Tissue cysts are very common in some species, but less so in others, such as horses.

Horses are one of the intermediate hosts of *T. gondii* . There is evidence that this parasite produces clinical signs in infected animals. However, *T. gondii* has also been isolated from healthy animals, indicating that the consumption of horsemeat constitutes a potential risk to humans, although compared with other animal species few serological studies of horses have been performed.

3 How does *Toxoplasma* differ from *Neospora* ?

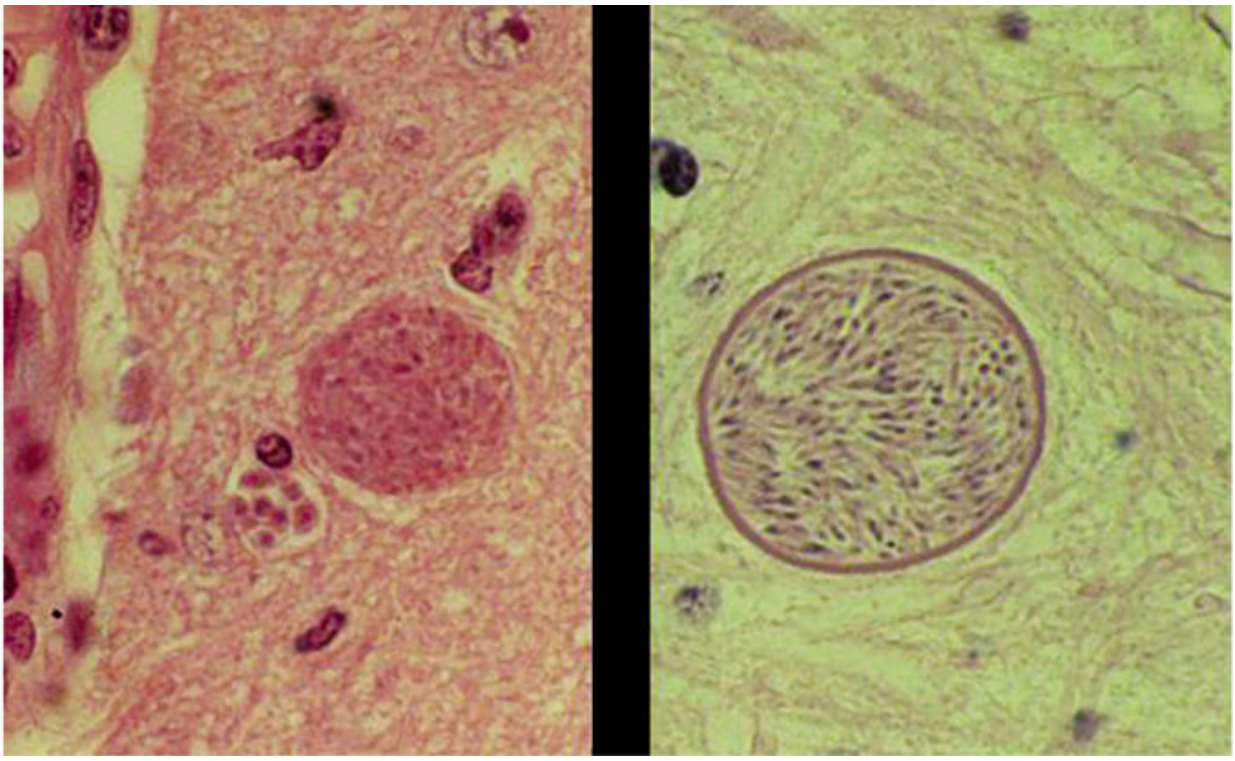
It was recently demonstrated that dogs are the definitive hosts of *N. caninum* , and that numerous animal species, including horses, act as intermediate hosts. In contrast to *T. gondii* infections, clinical signs, including neurological manifestations and abortions, have been described in horses infected with *Neospora* . However, few serological studies have been carried

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out to estimate the prevalence of *N. caninum* and *Neospora hughesi* infections in equids.

4 How is the aetiological agent confirmed?

Serology was first used to demonstrate *N. caninum* infection in horses in 1990. A few years later, another *Neospora* species (*N. hughesi*) was isolated from a horse in the USA. Cross reactions between the two species prevent differentiation between *N. caninum* and *N. hughesi* using routine serological methods. Definitive diagnosis is established by postmortem analysis of samples using molecular techniques.



Morphological characteristics of *Toxoplasma* (left) and *Neospora* (right) cysts. Rounded pseudocysts have been observed in the nervous tissues of animals with neurological processes characterised by motor incoordination, limb paralysis, and behavioural alterations such as circular movements or constant nodding of the head.

Image courtesy of Dr J. Dubey.

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4

Q & A
Parasitic infections
of the skin

Chapter 1

Q & A Endoparasites of the digestive system

Introduction

The diseases discussed in this chapter are, obviously, parasitic in nature. From the aetiological point of view, they can be grouped into two main categories: those caused by arthropods and those caused by helminths (more specifically nematodes).

However, many clinical pictures are aetiologically multifactorial, involving the participation of other pathogens (most importantly dermatophyte fungi).

Generally speaking, mange is one of the more familiar parasitic cutaneous processes, although several forms of mange are relatively unknown. These include processes that can be difficult to resolve using currently available drugs.

Four clinical cases of mange in horses are presented here. First, we discuss the mite species that produce mange in horses. Some of these

arthropod parasites are not exclusive to equids and can also affect other animals. Humans are not free of risk either, and can contract mange from equids.

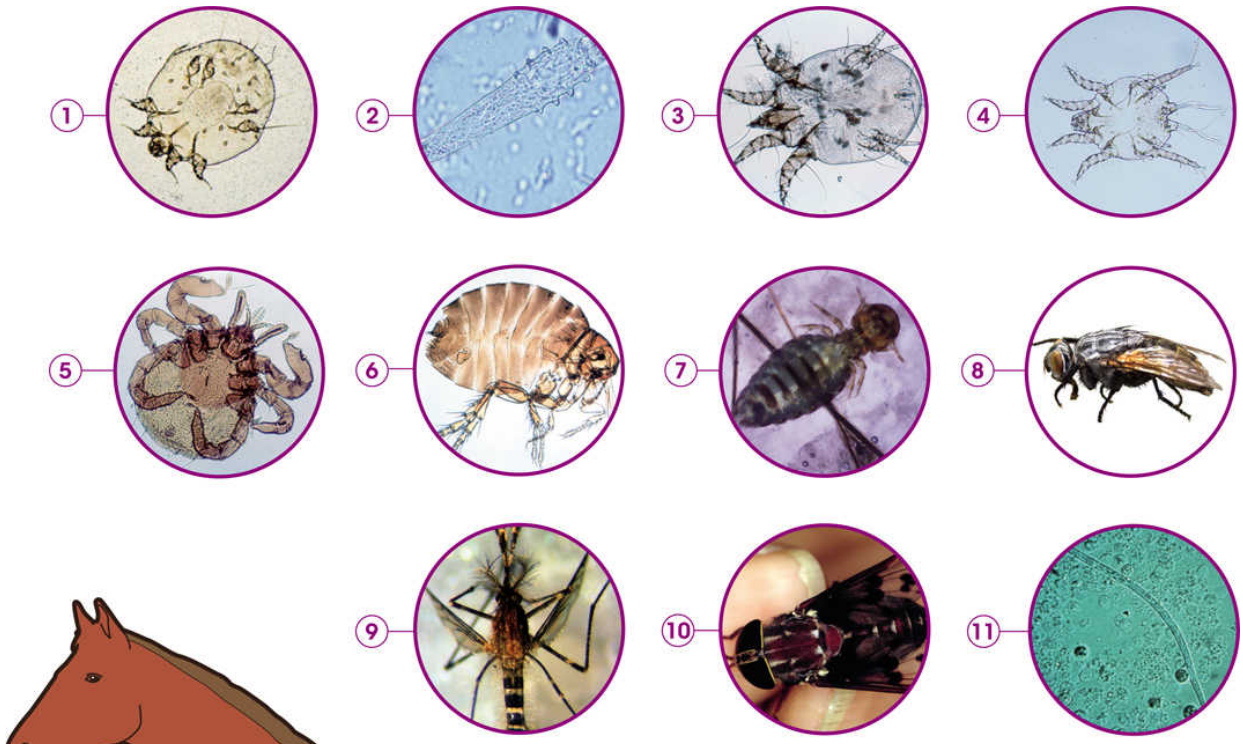
From a practical point of view, it is convenient to be familiar with the methods and techniques used to demonstrate the presence of mites in skin scrapings and to confirm suspected clinical cases. These classic techniques are not sophisticated, and can be performed easily and quickly, even with limited resources, allowing rapid establishment of an accurate diagnosis.

The vectorial role of arthropods is very important. Many viral and bacterial pathogens, among others, require arthropods (usually hematophagous species) in order to pass from one animal to another. Owing to the capacity of mosquitoes, ticks, fleas, and flies to transmit disease, arthropods play prominent roles in numerous pathologies of humans and animals. Moreover, climate change-associated habitat modification favours the geographical expansion of many of these diseases, which until recently were limited to tropical and subtropical areas. In addition to their role in the transmission of infections and/or diseases, some arthropods give rise to important clinical conditions caused by allergic, toxic, and other reactions.

The nematodes are the second group of parasites discussed in this chapter. These parasites, some of which are relatively unknown, have complex biological cycles and produce skin lesions that, a priori, are not associated with nematodes. The infections caused by these parasites are often incorrectly diagnosed, owing to their scarce clinical repercussions, seasonal nature, and poor response to treatment. Therefore, it is important to understand the true origin of these “diseases”.

In Chapter 4 we present 12 clinical cases: 4 dedicated to mange; 10 to the role of vectors and the pathogenic effects of mosquitoes and ticks; and 5 to nematode-specific cutaneous processes that are of particular importance in certain areas and at certain times of the year (e.g. habronemosis, filariosis).

Agents that cause parasitic skin diseases in equids



1.*Sarcoptes*
2.*Demodex*
3.*Psoroptes*
4.*Chorioptes*
5.Tick
6.*Echinodfaga*
7.*Werneckiella*
8.*Wohlfahrtia*

9.Culicid
10. Horse-fly
11. Microfilarias

39 What types of mites parasitise equids?

A 6-year-old male gelding arrives in Madrid in November from a stud located in the Unstrut river basin in Germany. The animal has numerous small, reddish-orange warts around the lips and eyes that were not evident when he was purchased 2 months previously. He is restless and rubs against any available surface, creating small wounds. The German veterinary surgeon is consulted and recommends ruling out trombiculosis, based on the origin of the horse and the time of year he was transferred.

1. What is trombiculosis?
2. What is it caused by and why was it suspected by the veterinary surgeon?
3. What other types of mites cause dermatitis in equids?





1 What is trombiculosis?

Trombiculosis is caused by larvae of species belonging to the family Trombiculidae. These larvae are obligate parasites, and depend on the ingestion of serous fluid, unlike the nymphal and adult forms, which are free-living and feed on decomposing vegetable or organic matter.

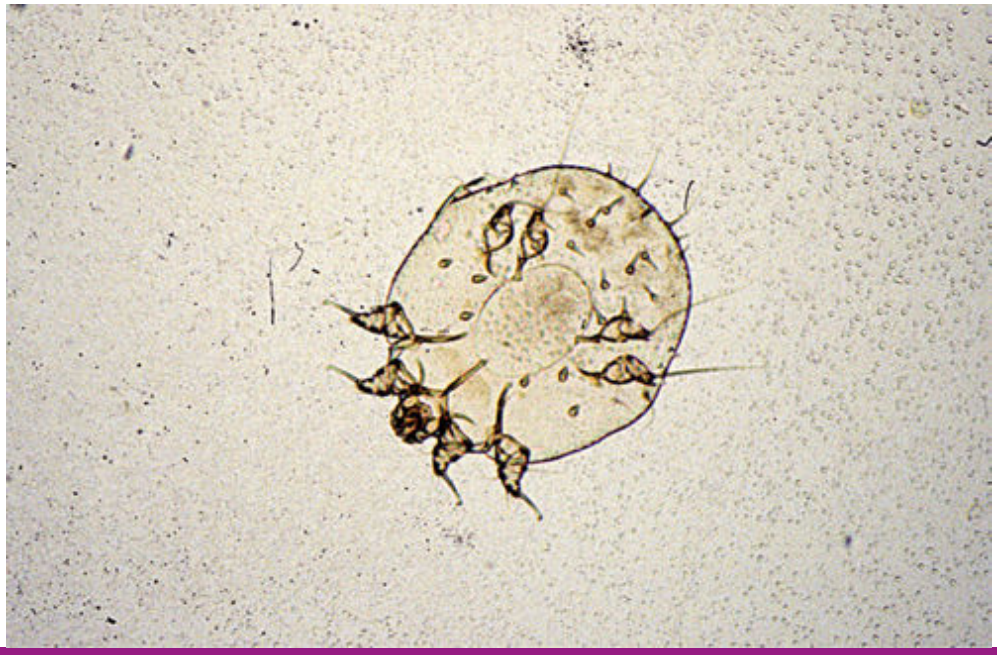
2 What is it caused by and why was it suspected by the veterinary surgeon?

The “warts” observed on the imported horse were in fact trombiculid larvae. These orange-red, ovoid larvae are about 200 µm long and have three pairs of legs and well developed mouthparts. In Germany this species is known as the autumnal, birch, or berry mite, after the time of year and places in which it thrives. Areas of elder and birch trees along the banks of the Unstrut river are known to veterinary surgeons as a source of trombiculosis.

Trombiculosis occurs when the larvae hatch from the eggs at the end of summer and climb the vegetation almost to knee-height, lending the foliage a reddish appearance when thousands of larvae are present. They preferably attach to areas of thin skin such as the perilabial or periocular region, the pinnae, or the fold of the pastern. In 3 to 8 days they double or triple in size and detach to continue development on the ground as free-living nymphs and adults. They have a peculiar feeding strategy: using the tips of the chelicerae (nail-shaped chitinous cutting structures), they cut through the stratum corneum, releasing a salivary secretion that reaches and dissolves the basal stratum. At the same time, a mobile tubular sucking organ is inserted into the tube they have created. This organ can exceed the total body length of the mite itself, and allows the saliva to penetrate deeper. Subsequent sclerosis around the tube created by the mite leads to the formation of a channel into which the tubular organ is inserted and through which the mite sucks.

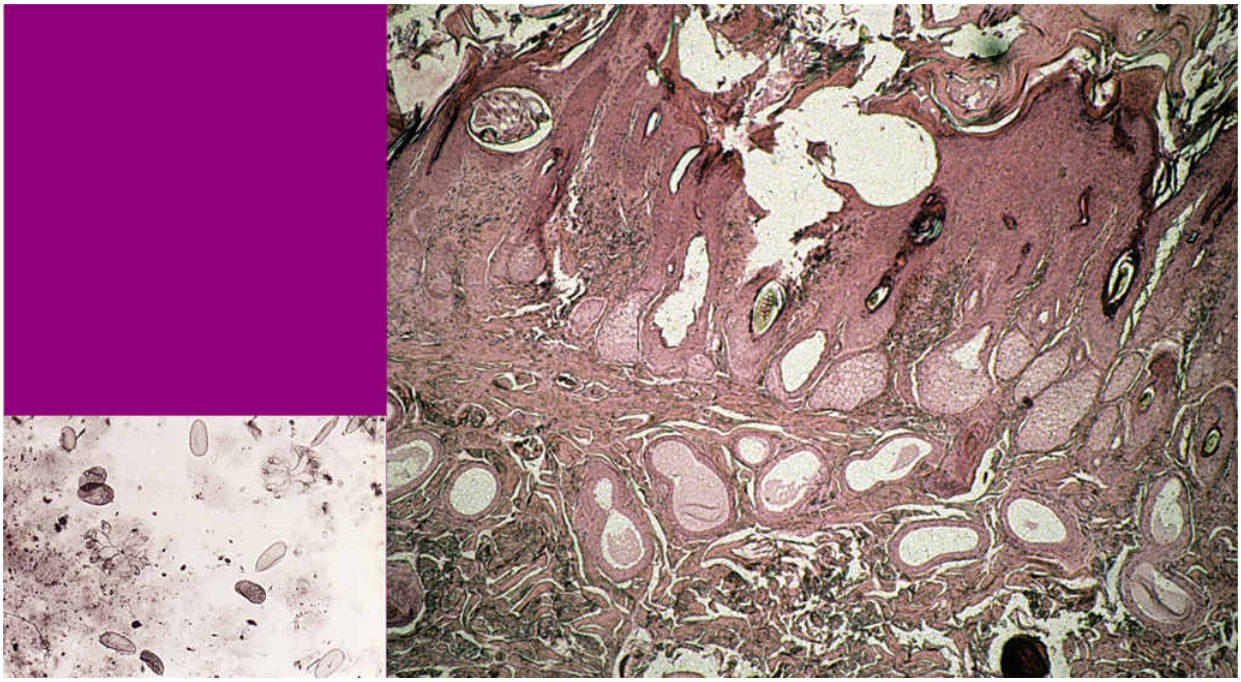
3 What other types of mites cause dermatosis in equids?

Mites are a group of animals belonging to the Arachnida class. They are not clearly segmented, and resemble miniature turtles with eight (adults) or six (larvae) legs. Instead of a head, they have very specialised mouth parts. Most, such as dust mites and grass mites, are free-living species. Others have undergone adaptations that allow them to live on host animals, feeding on secretions or exudates.



Sarcoptes . These are small, ovoid, whitish or greyish-white mites (females, 0.4 mm; males, 0.2 mm). The cuticle is covered in fine striations, triangular scales, and taxonomically specific spines. The well-developed first and second pairs of legs are more developed and are oriented forwards. By contrast, the less developed third and fourth pairs do not extend beyond the surface of the body and are oriented backwards. All tarsi end with a nail and a non-articulated bristle. The bristles are found on the first two pairs of tarsi of all stages and on the fourth pair in males. The ovoid eggs are large (160 µm), with a transparent cuticle through which the embryo is visible. The abundance of mites found in a skin scraping of a lesion provides an indication of the evolution of the process.

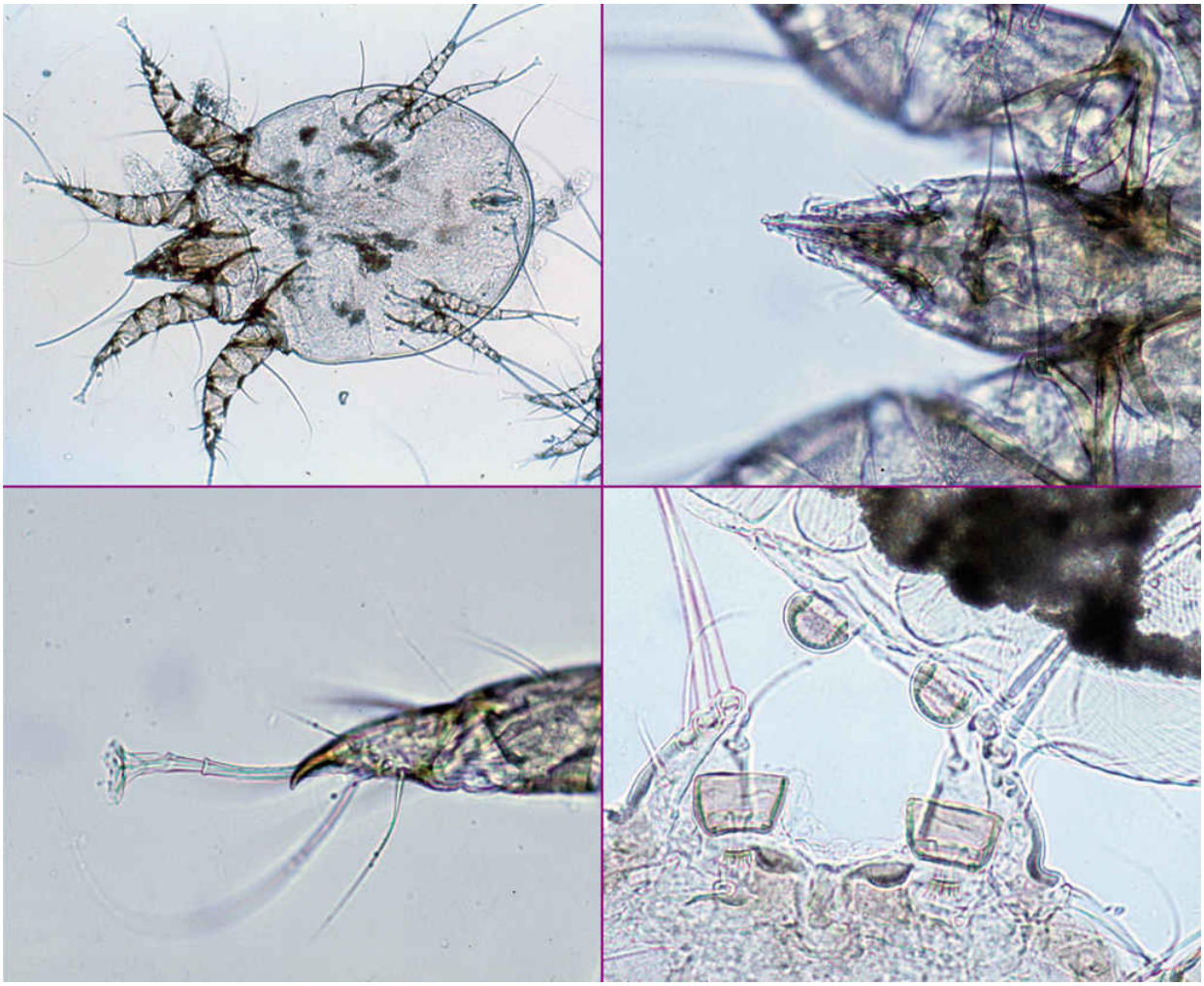
Sarcoptes. The presence of adult mites and eggs in a skin scraping is indicative of active parasitisation.



Some relationships between mites and animals are accidental, but do not necessarily go unnoticed. For example, infestations caused by free-living forage mites of the Tyroglyphidae family cause dermatitis in animals and humans, resulting in the formation of pruritic papules and crusts at the sites of attachment.

Horses can be parasitised by chance by *Dermanyssus gallinae,* the "red mite" of poultry, named after the colour it acquires when full of blood (unfed mites are greyish-white). They live hidden during the day in the cracks and crevices of the doors, windows, and walls of poultry houses, and come out at night to briefly feed on blood, after which they return to their refuge, pausing to defecate before entering. Their activity is closely dependent on the ambient temperature.

During their biological cycle they lay half a dozen eggs 6 to 8 times in the cracks and crevices in which they live. Their biological cycle lasts 7–10 days, and multiple generations are produced throughout the year. They can tolerate fasting for at least 5 months, and although they feed primarily on birds they can also feed on rabbits, dogs, cats, goats, cows, horses, and humans. Therefore, even in the absence of birds for some time, a residual mite population can persist for several months. In horses this mite causes intense dermatitis, with itching and eczematous signs and a mangy appearance.



Mites of the genus *Psoroptes* are relatively large and are easily observed moving across the body surface. They have an ovoid body and an elongated, conical rostrum. The legs end in a long triarticulated peduncle with suckers and/or long bristles. The smaller males (0.6 mm) have suckers on the first, second, and the third, well-developed pairs of legs. The posterior end extends into two mammillations, where the copulatory suckers are located. The fertilised females are 0.85 mm long and have suckers on the first, second, and fourth pairs of legs. They are semi-transparent; eggs inside the body are sometimes visible.

Other mites such as those of the genera *Psoroptes* and *Chorioptes* are found continuously attached to the body surface, while females of *Sarcoptes* burrow tunnels in the skin in which they lay their eggs, and *Demodex* live permanently inside a hair follicle. Pruritus is a clinical sign common to all these parasites and most of the associated pathological alterations are the result of hypersensitivity reactions to the products secreted or excreted by the mites and the damage caused by self-trauma. One exception is *Demodex* , which tends not to produce clinical signs.

Different types of mange can be distinguished based on their aetiology and clinical signs:

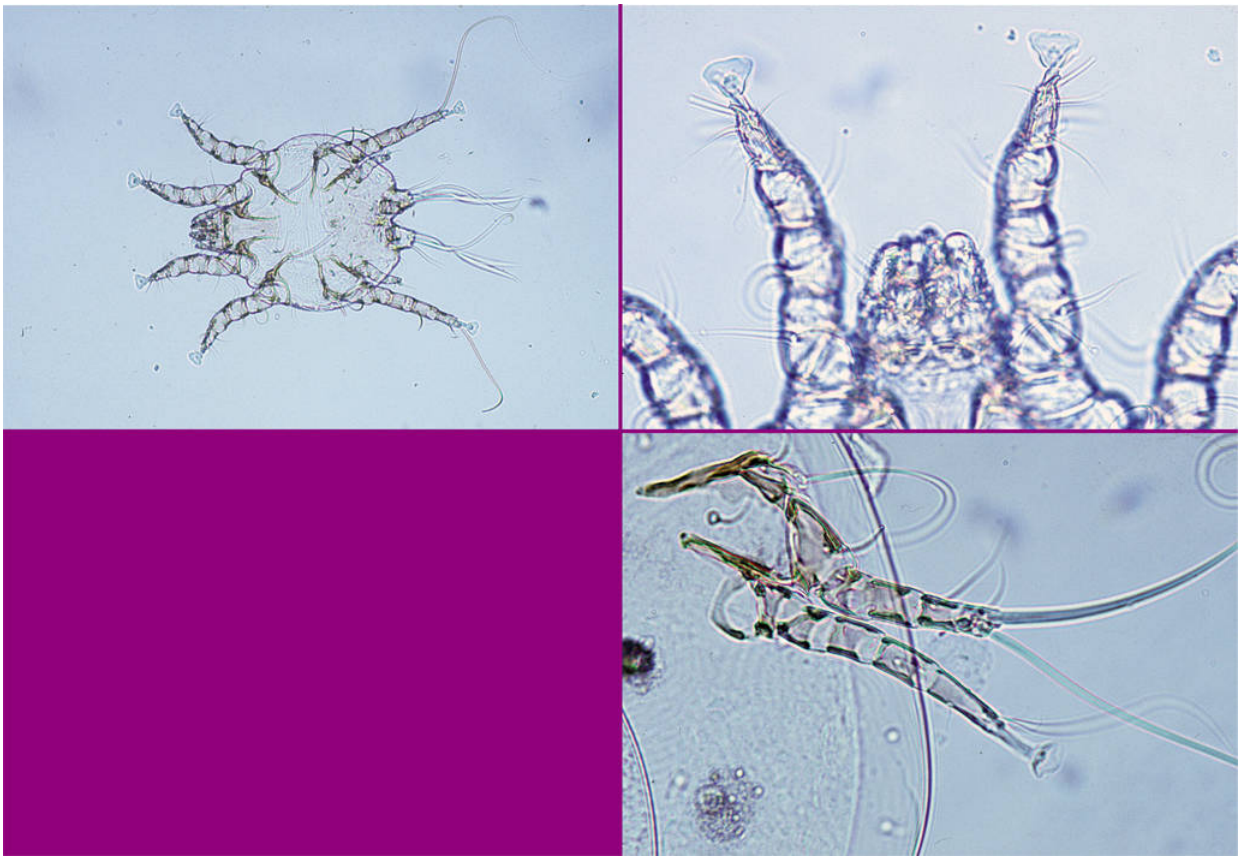
Sarcoptic mange , caused by *Sarcoptes scabiei equi* , affects areas devoid of hair, particularly the head.

Psoroptic mange , caused by *Psoroptes equi equi* , affects areas with abundant wool or hair, as well as the back and flanks. Mange caused by *Psoroptes cuniculi* is limited to the pinnae, and **chorioptic mange** , caused by *Chorioptes bovis equi* , affects the limbs and tail.

The biological cycle of these mites is direct and lasts less than 1 month. All phases of the biological cycle take place on the skin of the host, and transmission is facilitated by direct contact between animals.

These days, mange is relatively sporadic in most developed countries, given that the majority of horses are used for recreational or sporting activities, and therefore receive considerable health care and attention. The appearance of mange is closely related to inadequate management and is more frequent in colder months of the year.

With an elongated body and a rostrum that is wider than it is long, *Chorioptes* can be identified by its short, unjointed pedicles. The males measure about 0.3 mm and have suckers on all tarsi. The last pair of legs is poorly developed and the abdominal lobes (where the copulatory suckers are located), are elongated and end in four long flattened bristles. The fertilised females are about 0.4 mm long and lack suckers on the third pair of legs.





Demodex . These vermiform mites have transverse striations that do not correspond to any internal metamerisation. They have a broad rostrum, with a pair of stylet-like chelicerae and adjacent palps. The legs, which are very underdeveloped, are attached to the anterior part of the body and end in spines. The female measures about 0.25 mm, and has a ventrally located genital pore. The male measures 0.2 mm. Its penis is located in the dorsal portion of the cephalothorax.

Dermanyssus gallinae. These are elongated, ovoid mites of 0.6 mm (males) to 0.75 mm (females), with four pairs of highly developed legs. The chelicerae of the female are particularly long and stylet-like, and are visible from the dorsal aspect. They do not live on their hosts, and only accidently feed on horses that share habitats with galliformes.



40 Are mites easily visualised?

The owner of three horses in Almonte (Huelva) purchases a female mule, which he stables with the horses. Three days after her arrival, the mule appears to be very restless, rubbing and scratching herself against the wooden poles and the walls of her shelter. This rubbing behaviour persists during a visit by a veterinary surgeon, who observes alopecia at the base of the ears, the upper part of the neck, the scapular area, and the base of the tail. Excoriations are evident, but no maculopapular eruptions, nodules, or vesicles. After careful examination of the other animals, lesions are detected on the pinnal margins, face, and lower jaw of one of the mares, who shows no signs of pruritus.

1.What type of mange do these clinical signs suggest?
2.What could account for such a pruriginous reaction in the mule?
3.How is diagnosis confirmed?
4.Can carriers be asymptomatic?



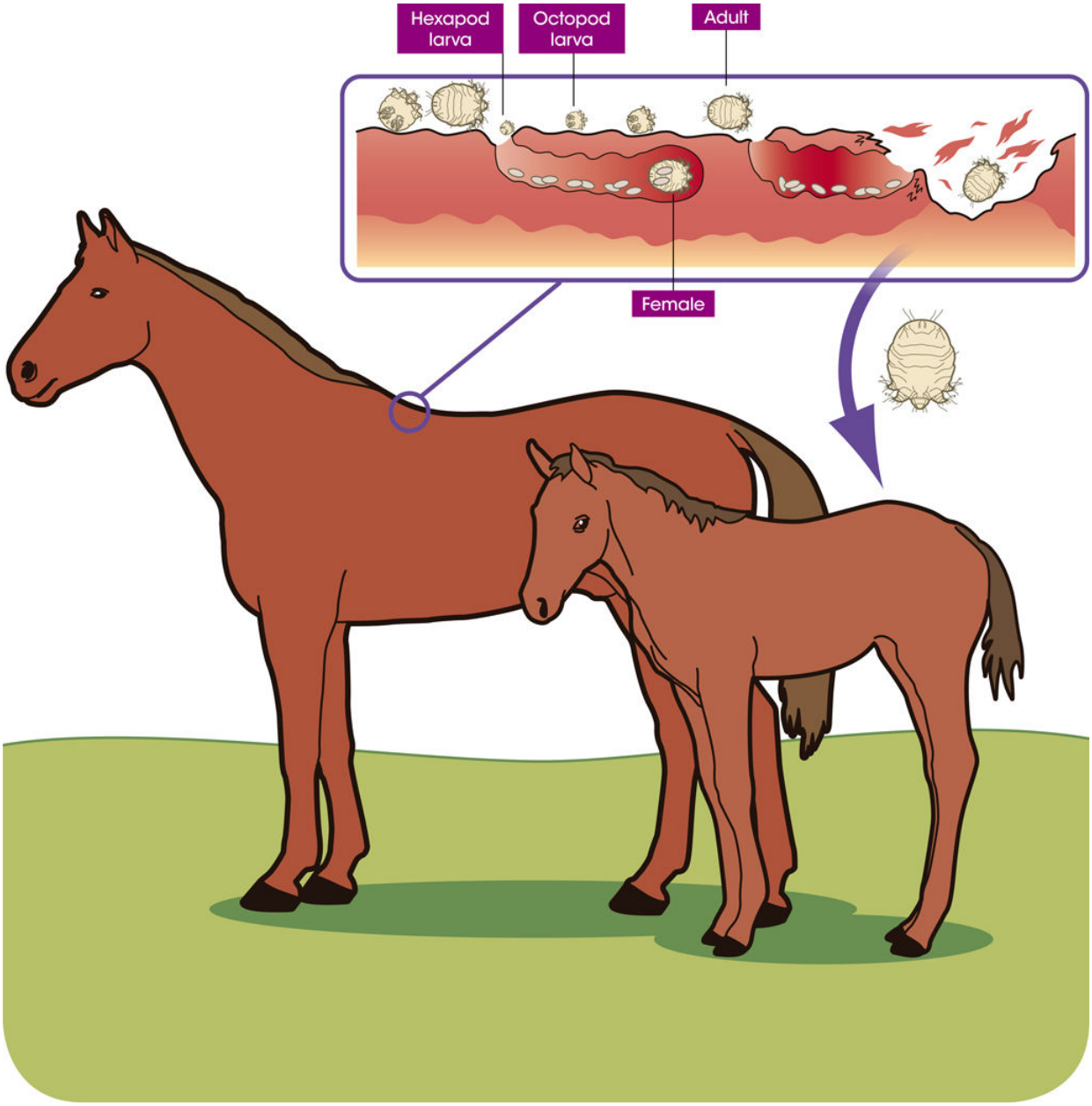
1 What type of mange do these clinical signs suggest?

Intense pruritus that induces obvious self-trauma caused by scratching suggests possible sarcoptic mange. This type of mange is caused by small mites (0.6 mm) of the genus *Sarcoptes* . The species found in equids is *Sarcoptes scabiei equi* . Although this species can cause infections in humans, these infections usually resolve spontaneously in immunocompetent individuals. *Sarcoptes scabiei* var. *hominis* is the agent of human mange or scabies, a term derived from the Latin *scabere* (“to scratch”). This dermatosis has been known since antiquity. The first association between mites and scabies lesions was reported in 1647 by Bonomo and Cestón.

Females of *Sarcoptes* dig burrows or tunnels in the skin and feed on lymph and epidermal cells. They live for about 30 days and, once fertilised, lay 3–5 eggs per day in the tunnels they create. The eggs contain a hexapod larva that hatches in 3–5 days. Some larvae move to the skin surface and die, others migrate to hair follicles or pre-existing tunnels, and yet others create

new burrows and after 10–12 days moult into the octopod nymph stage. After two nymphal stages, sexual differentiation occurs. Immature females begin to dig tunnels before copulation. After 4–5 days, egg laying begins, ending the biological cycle. The larvae, nymphs, and immature females are the stages responsible for the spread of mange, but are very fragile outside the host.

Life cycle of *Sarcoptes*





Mites exert an irritant effect on the skin as a consequence of their perforating action, sucking, and the constant movement of their bristles, scales, and spines. The allergic reaction, combined with nervous stimulation induced by the mites' activity, produces a stinging sensation to which the animals respond by biting or rubbing.



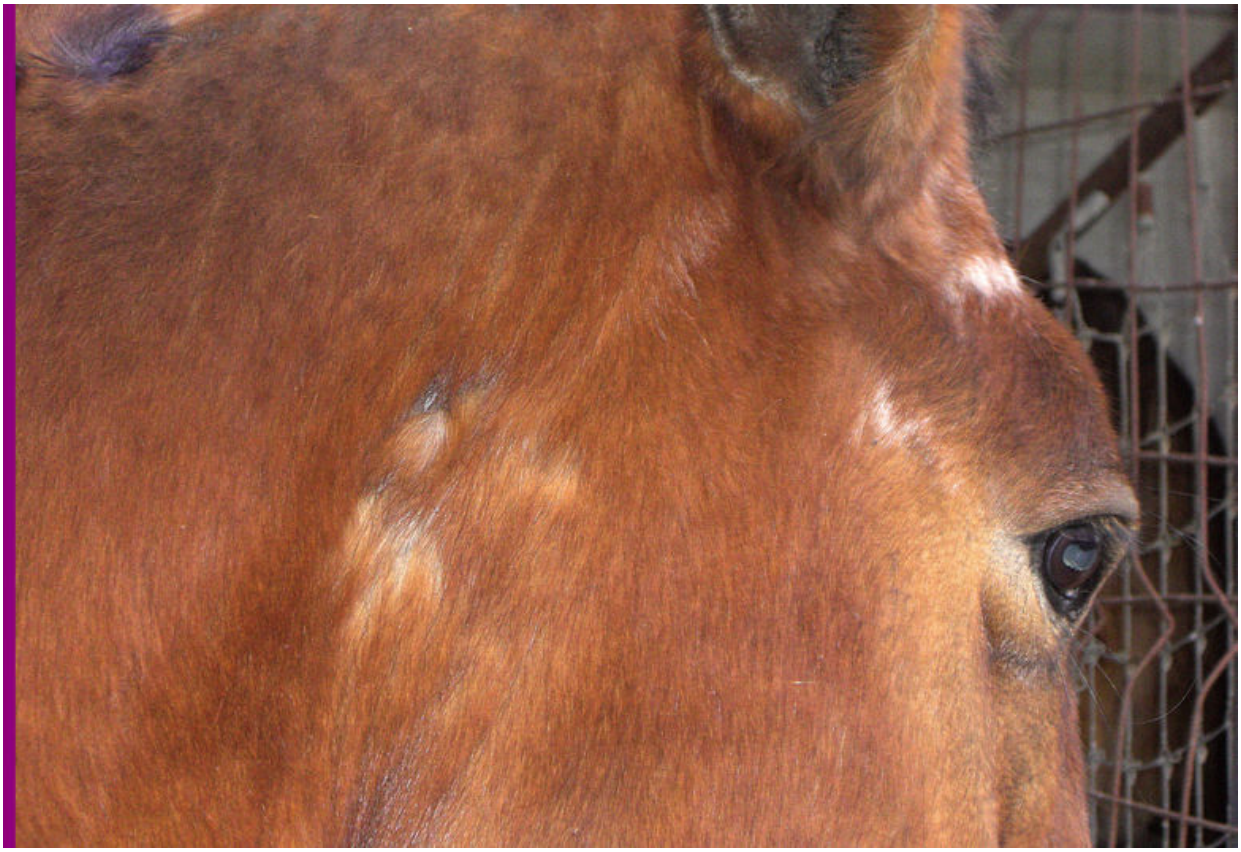
Mange is transmitted by direct contact or via fomites. The sharing of brushes, blankets, or saddles between animals facilitates transmission.

The first signs of parasitic invasion are diffuse alopecia with highly pruritic papular/crusting eruptions on the skin of the affected areas. Scratching of these lesions gives rise to intense excoriation in areas adjacent to the entry point of the parasites, and the formation of yellowish-grey crusts due to secondary bacterial infections. Over time, the process becomes chronic, the skin thickens, and wrinkles, folds, fissures, and cracks (hyperkeratosis) appear. In very chronic cases areas of hyperpigmentation appear and the skin acquires a thickened appearance (lichenification), resembling that of an elephant.

2 What could account for such a pruriginous reaction in the mule?

The mechanisms underlying the pathogenesis of mange are twofold: local action of mites on the skin resulting in mechanical irritation of the epidermis (which they perforate during feeding and/or reproduction) or dermis (in which they dig tunnels); and a hypersensitivity reaction to allergenic agents produced by the parasite. The activity of mites in the skin of the host causes intense pruritus as well as skin lesions of variable severity.

In the mare in which the infection originated the skin of the pinna is thickened and alopecic.



As a consequence of the loss of continuity of the skin caused by the invasion of mites, coinfections with bacterial and fungal agents (e.g. *Trichophyton*) are common.



Allergic hypersensitisation is induced by the mites' digging activity, feeding habits, and excretions, and by the absorption of agents contained in their buccal secretions and the remains of dead *Sarcoptes* mites. An increase in immunoglobulin-secreting cells in the pinnal area has been observed 2–5 weeks post-infection. These reactions result in severe pruritus, to which the affected animal reacts by scratching against hard surfaces, giving rise to traumatic lesions. Exudate fills and blocks the tunnels excavated in the dermis, and coagulates forming crusts. At the same time, the germinative epithelium responds to the aggression with an increase in cell multiplication, leading to thickening of the skin and hyperkeratosis.

3 How is diagnosis confirmed?

Sarcoptes scabiei is difficult to detect. Multiple skin scrapings, many of which prove negative, are usually required to detect this mite. Ideally skin scrapings should be obtained from areas containing recent, erythematous lesions, from which it may be easier to remove mites. Skin scraping of chronic lesions with hyperpigmentation and hyperkeratosis is ineffective. The treatment of skin samples with hot 10 % KOH solution dissolves the crusts and facilitates detection of mites.

In cases involving a negative aetiological diagnosis, but evident clinical manifestations (pruritus and compatible lesion distribution) and epidemiological data indicating possible contact with sick animals, animals can be treated with acaricides and diagnosis established based on the treatment response.

4 Can carriers be asymptomatic?

The allergic component of mange is very important. In mares, the presence of chronic lesions on the pinna that resolve after acaricide treatment indicates an attenuated response to the presence of mites and more slowly progressing parasitation. A similar phenomenon is observed in one clinical form of human scabies (Norwegian scabies): the appearance of excrescences on the body and limbs of those affected appears to be the result of the host response.

41 Can rabbits transmit mange to horses?

A veterinary surgeon receives a call from an animal shelter requesting treatment of an animal with mange. The patient is an older horse that is no longer used for riding classes, and arrived at the shelter the previous week. Lesions are widely distributed around the mane area. Staff at the shelter mention that this problem was already evident when the horse arrived. They say that they have had previous cases of mange in other animals, such as goats and rabbits, but only affecting the ears and never the neck.

1.What type of mange is compatible with this lesion distribution?
2.Are any forms of mange common to horses, goats, and rabbits?



1 What type of mange is compatible with this lesion distribution?

This parasitosis is caused by species of the genus *Psoroptes* (scab mites), which measure 0.4–0.8 mm in diameter and do not dig tunnels. The biological cycle lasts 10–12 days. The females live 30–40 days and lay 5 eggs per day. They can survive outside the host for up to 2 weeks. They feed on blood and produce a local inflammatory reaction of the skin.

In horses, *Psoroptes equi* is generally found in areas covered with dense hair such as the mane, forehead, and the base of the tail. It can extend to the inner aspect of the thighs, the prepuce, and the rest of the body. The skin's reaction to the bites of the mite results in the formation of nodules. These nodules exude a lymph-like fluid that forms crusts when it dries. Alopecia and increased epidermal desquamation are also observed. The crusts and scales can be up to 1 cm thick. The pruritus is quite severe, and provokes rubbing behaviour that gives rise to severe deep inflammation with suppuration.

These two forms of mange can be distinguished by detecting the causative mite. *Psoroptes equi* is easier to detect than the mite responsible for sarcoptic mange. Moreover, this mite shows a predilection for protected areas of the body and tends to form circumscribed foci that spread slowly. However, in cases of generalised distribution, the clinical pictures of both forms of mange can be difficult to differentiate. It is not uncommon to find mixed forms on the same horse (up to 20 % of cases), which are more resistant to treatment. Because the parasites are located in the most superficial layers of the skin, psoroptic mange can be cured rapidly and easily. However, this type of mange can be more difficult to cure if large areas of the body are affected.

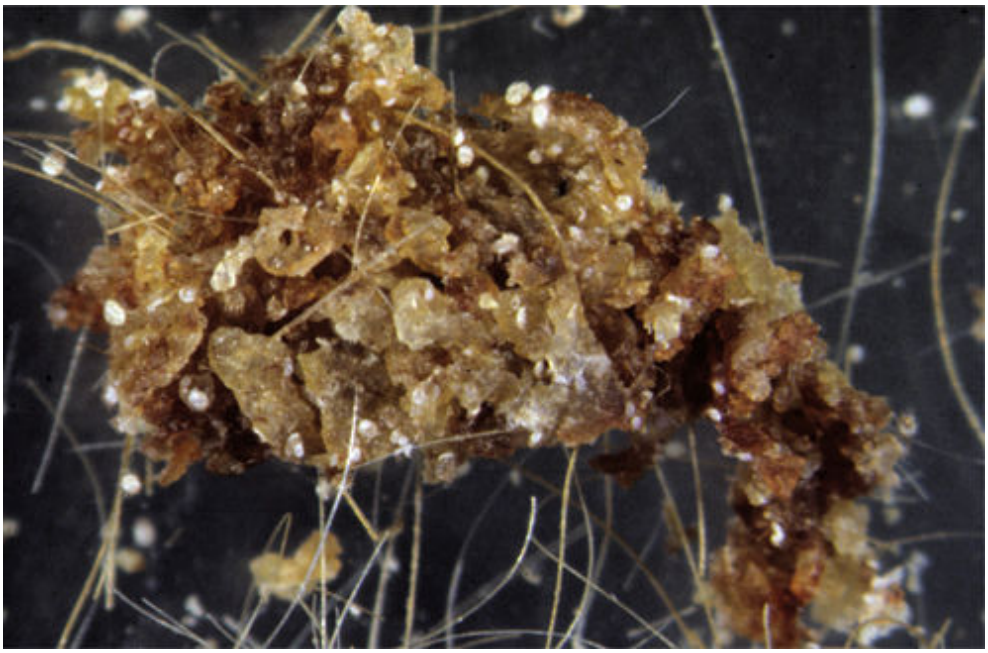
2 Are any forms of mange common to horses, goats, and rabbits?

Psoroptes equi cuniculi can cause mange on the ears of horses and goats. In natural conditions, some animals may exhibit no clinical signs, while in others head shaking or external otitis may be observed. Common signs include large accumulations of cerumen, as well as crusting and even alopecia at the base of the ears. This large, oval-shaped mite has a striated back and lacks spines. It closely resembles *P. equi* . The male measures about 0.45 mm and the female 0.7 mm. The articulated peduncles contain

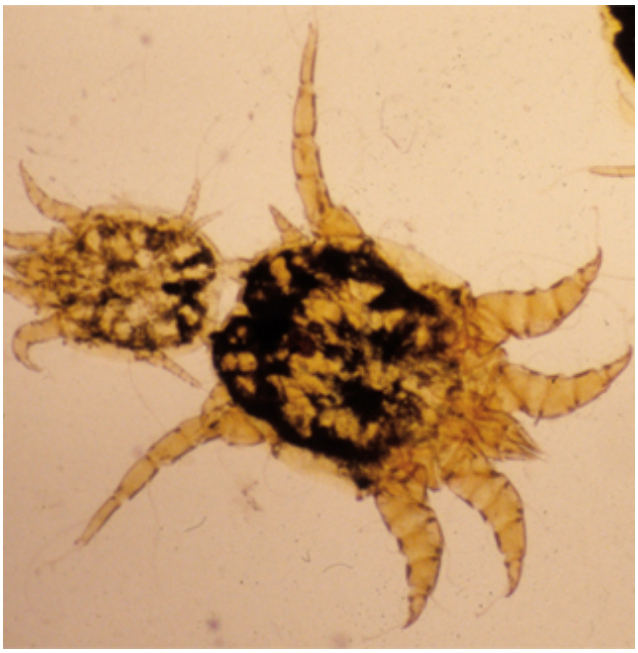
suckers, a characteristic feature of this genus. This mite is found inside the pinna, where the biological cycle occurs over a period of 2–3 weeks, producing up to 10,000 mites per cycle. Psoroptic mange is characterised by external otitis with serous exudate and the formation of foul smelling crusts.



Auricular mange caused by *Psoroptes cuniculi* can be shared by horses, goats, and rabbits. In severe cases affected animals become highly irritated, shaking their heads and in some cases striking their heads against nearby objects, resulting in bruising around the base of the ears.



Using an otoscope, mites can be easily observed as small whitish mobile dots that stand out against the crusty exudate inside the pinna.



Morphologically, *Psoroptes cuniculi* (above) is almost indistinguishable from *Psoroptes equi.*

Some rabbits infected with *P. cuniculi* in natural conditions exhibit no clinical signs, while in others intense head-shaking is accompanied by the accumulation of cerumen and the formation of crusts inside the ears. Mites induce an inflammatory reaction in the ear canal, with hyperaemia and the production of serous exudate. These yellowish-brown crusts become thickened and stratified in severe cases.

Mites are commonly found in the ear canal of sheep and goats. In sheep, both *P. cuniculi* and *Psoroptes ovis* have been detected in the pinna. According to some authors, mites that cause psoroptic mange of the body may remain inside the ear canal during the summer months, when conditions are not conducive to development, thereby acting as a reservoir of infection. In goats, clinical signs may be absent in some affected animals (carrier animals). In others accumulations of dense brownish cerumen in the external auditory canal, crusts on the pinna, and areas of alopecia are accompanied by clear signs of irritation, including intense head-shaking.

42 How is leg mange diagnosed?

A Friesian horse is brought from Navarra in northern Spain to a riding school near Madrid. A caretaker notices that the animal moves continuously and stamps his hooves on the ground throughout the night. The horse also scratches and bites his pasterns. Careful examination reveals mild epidermal desquamation with hair loss and the formation of crusts, folds, and cracks in the central area of the pastern; the skin is dry, rough, and thickened, with horny papillary proliferations.

1. What parasite causes lesions compatible with those of this horse?
2. How is this form of mange distinguished from others? Can it affect humans?
3. Can it be transmitted to horses by any farm animals?



Image courtesy of Dr D. Rendle

1 What parasite causes lesions compatible with those of this horse?

Species of the genus *Chorioptes* are typically found around the pasterns. These mites are morphologically very similar to those of the genus *Psoroptes* .

The species in question in this case is *Chorioptes bovis equi* , which lives on the surface of the skin of the limbs, usually on the pasterns of the hindlimbs, and feeds on epidermal detritus. Its biological cycle is completed within 2–3 weeks. For a long time it was thought that mites had poor survival capabilities outside the host. However, recent studies in cattle in varied environmental conditions have shown that these mites can survive for up to 69 days. Therefore, a quarantine period of 2.5 months is recommended.

During cold and humid periods, mites are more numerous and the associated clinical signs more frequent. In summer, the process disappears spontaneously, the number of mites decreases considerably, and the small numbers of surviving parasites are limited to the pastern and interdigital cleft.

2 How is this form of mange distinguished from others? Can it affect humans?

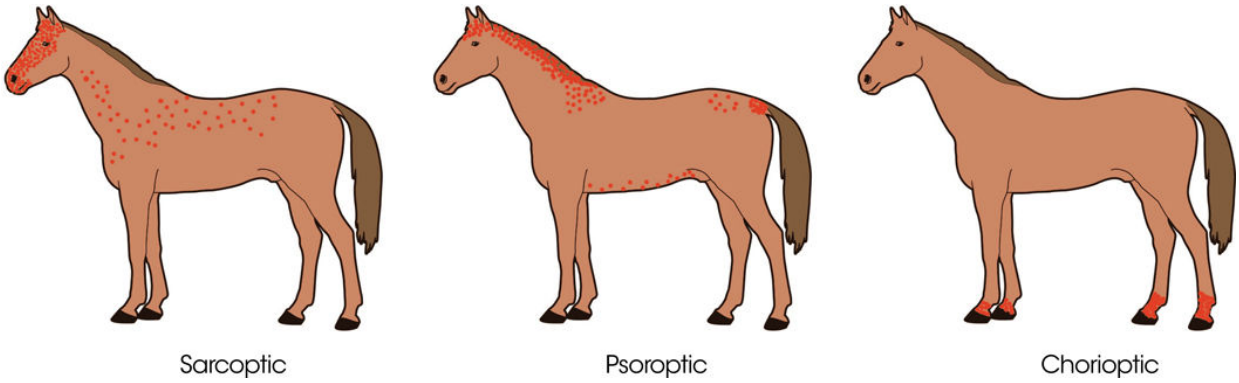
Chorioptic mange is also known as leg mange, since it usually begins in the hindlimb pasterns, from where it spreads, particularly in foals, to affect the entire body.

It is one of the least contagious forms of mange (**it does not affect humans**) and progresses slowly, in many cases affecting only a single limb over the course of several months. Chorioptic mange induces marked pruritus, to which horses react by continuously stamping and biting the pasterns.

Initially, chorioptic mange can be identified by the presence of scaling, which results in shedding of the epidermis, gradually leading to the formation of scaly crusts and the appearance of transverse ulcers. It is more common during winter and predominantly affects the palmar/plantar aspect of the pastern, from where it can progress to the fetlock, the front and rear aspects of the cannon bone, the hock, carpal joint, and elbow. In rare cases, it

can extend to the back, neck, leg, and thigh. However, its progression is so slow that changes are barely noticeable until several months have passed. In summer, chorioptic mange spreads much more slowly than in winter. In some cases only a single limb is affected.

Distribution of mange in equids: main areas



- **Sarcoptic mange** is caused by *Sarcoptes scabiei equi* . It is a very contagious process that **can transiently infect humans** who have contact with parasitised animals. In the superficial layers of the epidermis the mite digs tunnels in which it deposits its eggs, initially causing intense pruritus. Secondary infections induced by scratching are very common. An automatic scratching reflex can be induced by rubbing a hand on the affected area of skin, blowing against the skin, or even by making loud noises and exaggerated movements of the lips. A positive reflex is reason to suspect mange. Depending on the animal's general condition, nodules and small vesicles initially form on the head and withers, gradually extending to the back and the lateral chest walls, in some cases reaching the back, flanks, and hindlimbs. In severe cases animals develop cachexia and die. Sarcoptic mange is highly contagious to humans, although it usually follows a benign course and heals spontaneously in 2–8 weeks.
- **Psoroptic mange** is relatively rare. It is a highly contagious process that **does not affect humans** and is transmitted by direct contact or by exposure to fomites. Mites do not burrow in the dermis, but live on the skin surface and feed on exudate and cell debris. In horses, *Psoroptes equi equi* produces lesions in areas covered with hair, such as the tail, mane, and ears. It can extend to affect the entire body, beginning with the inner aspect of the thighs and the prepuce. The characteristic lesions are well

circumscribed patches that appear in protected areas and those covered with long hair. Initially, the cutaneous response to mite bites gives rise to nodules that exude fluid, which then dries and forms crusts. Epidermal scaling and alopecia are observed as the process progresses. The crusts and scales can be up to 1 cm thick. The skin gradually thickens, forming folds and acquiring a leathery appearance. The pruritus can be quite intense, provoking rubbing behaviour that gives rise to severe deep inflammation with suppuration.

Because the parasites are located in the most superficial layers of the skin, psoroptic mange can be cured rapidly and easily. However, if large areas of the body are affected, it can be more difficult to cure.

3 Can it be transmitted to horses by any farm animals?

Transmission can occur between cattle and equids, which share common mite species. This transmission was demonstrated in the 1950s, as well as the mite's ability to survive for weeks outside the host, provided they have access to epidermal tissue.



Images courtesy of Dr J. Littlewood.

43 Are males required in order for lice to multiply?

A farrier who routinely attends to a group of donkeys used for fair rides mentions to the owner that their general condition has deteriorated; they have lost weight and developed numerous plaques all over their bodies. A veterinary surgeon who inspects the donkeys confirms that their diet is inadequate, and collects more than 100 lice, all females of the species *Werneckiella equi*.

1. What type of lice affect equids?
2. How do males differ from females?
3. What are the clinical signs of pediculosis?
4. What diseases do lice transmit?



1 What type of lice affect equids?

Infestations by lice (order Phthiraptera) in horses and donkeys are caused by a species of the suborder Mallophaga, *Werneckiella equi,* and by another of the order Anoplura, *Haematopinus asini.*

These are permanent parasites. They measure a few millimetres in length, have a flattened body, and lack wings. The head, which is narrow in Anoplura and broad in Malophaga, has well-developed antennae but lacks eyes. On each thoracic segment is a pair of powerful legs, at the end of which is a peculiar anchoring system that prevents detachment of the louse caused by the host's movements. On the last segment is a curved claw that opposes a small protuberance on the penultimate segment, allowing the louse to grasp the hair shaft. Thus, fixed at six points, this louse is not easily detached from the host.

The eggs or nits are firmly attached to the base of the hair, where they are deposited by females that lay up to 15 eggs per day. The larvae inside resemble the adult form, and require three moults to reach the adult stage.

- ***H. asini*** *:* the life cycle of this louse lasts about 4 weeks. The egg incubation period is 4–14 days, and the nymphs require 11–12 days to develop and transform to the adult stage. Females can deposit 50–100 eggs over their lifespan of 4–5 weeks. This louse can survive outside the body for an average of 2–3 days. The male to female ratio is usually 1:1.
- ***W. equi:*** the life cycle of this louse lasts 27–30 days. The egg incubation period is approximately 7 days. The males are extremely rare, suggesting that females reproduce by parthenogenesis.

In equids, intense Phthiraptera infestations usually occur during winter, when the conditions for their multiplication are particularly favourable owing to the thickness of the host's coat and, in certain cases, the stabling of animals in unhygienic and overcrowded conditions. Lice pass from one animal to another by direct contact, and can be found on neonates as little as 10 hours after birth. In adults, lice are more common among young animals. Away from the animal, lice survive for only 3 days, and eggs that detach from equids die in the soil. Utensils such as brushes, blankets, and harnesses can promote the transmission of parasites.



In infected animals it is easier to detect nits, which resemble strings of small pearls firmly attached to each hair, than adult forms. The number of nits attached to a hair provides an indication of when the parasitisation began.



Upon separation of the hairs in affected areas, the deterioration of the hair coat and epidermis can be appreciated. Alopecic areas with dermal scaling and small lesions are indicative of pediculosis.

2 How do males differ from females?

The abdomen consists of nine segments. The sting-like male copulatory organ and the female genital orifice are located on the last segment (which in females appears to be divided in two).

3 What are the clinical signs of pediculosis?

Pediculosis technically refers to an infestation caused by species of the genus *Pediculus* , which parasitise humans and primates. However, this term is also extended to any infestation caused by Anoplura or Mallophaga.

The masticatory activity of the Mallophaga causes severe pruritus and irritation in parasitised animals, with consequent effects on nutrition and rest. Similarly, the loss of blood caused by Anoplura infestation can result in weight loss and anaemia in cases of massive infestations, during harsh winter months, or in animals with concomitant diseases.



The clinical signs of pediculosis are more evident in winter when the haircoat is thicker and animals are stabled and in closer contact with one another.

For all these reasons, Mallophaga infestation results in localised hair loss, lesions, and areas of ulceration caused by both the intense activity of the louse and by self-trauma. Anoplura cause more damage as a consequence of their blood sucking activity, resulting in weakness in infected animals, stunting in young animals, and deterioration and loss of hair in adults.

Small numbers of lice can be tolerated without the appearance of clinical signs. However, the pain caused by bites and the frequent movement of the parasites contribute to the development of dermatitis and pruritus. This in turn causes restlessness and provokes constant scratching, giving rise to skin lesions, scaling, and eczema. Hairless areas and severe skin lesions can be observed in animals that continuously rub themselves against the ground, posts, or trees.

While diagnosis is relatively straightforward, in some cases lice are not detected despite a clinical picture compatible with pediculosis. In the absence of adults, the detection of eggs or nits attached to the hair is sufficient to establish diagnosis; the number of eggs attached to a single hair can provide an indication of the duration of the infestation, since females always lay their eggs at the base of the hair.

4 What diseases do lice transmit?

Lice of equids *(H. asini* and *W. equi*) are capable of transmitting equine infectious anaemia virus.
Phthiraptera also appear to play a role in the transmission of *Trichophyton* dermatomycoses.

The economic effects of these parasitoses are related to the activity of the lice, which affect host vitality and productivity, as well as their role as reservoirs and transmitters of diseases.



Lice spend their entire life on the host. Their biological cycle, from egg to adult, lasts approximately 1 month, and involves three intermediate stages. Transmission occurs directly via contact between animals or via fomites (e.g. brushes, blankets, saddles).



Werneckiella equi : males, 1.6 mm; females, 1.8 mm. These lice are yellowish with rusty reddish brown stripes, and have a head almost as long as it is wide. The thoracic segments are of equal width and each abdominal segment has a single spot.

Haematopinus asini : males, 2.6 mm; females, 3.6 mm. These lice have an elongated head, a narrow yellowish grey body, and long, hairy antennae. The trapezoidal thorax is brown with dark spots and the abdomen is yellowish and oval shaped.

44 Why are ticks so abundant?

During a horseback tour through a farm in the province of Ciudad Real, a group of riders stops at noon for lunch, and several of them dismount and walk around. By the time the riders return home, ticks have ascended their boots and several have attached themselves to the skin, especially around the waist area. The weather is especially warm and it has not rained in several days. There are no water sources nearby.

1.How do ticks survive in conditions of extreme drought?
2.Why do the eggs not die on the ground?
3.What is diapause?



1 How do ticks survive in conditions of extreme drought?

Ticks are considered highly conserved organisms. Despite their evolutionary age (it is thought that they first appeared about 200 million years ago, at the end of the Paleozoic Era), their morphology and biology are very uniform, and deviate little from that of the ancestral forms from which they are descended.

Argasids always live in microhabitats that are protected from the elements (e.g. burrows, pigsties, caves, birds' nests) and allow immediate contact with hosts.

By contrast, the Ixodidae family includes species that have undergone ecological and biological adaptations that allow them to settle inside burrows or kennels and to also live in open fields, where they can parasitise other hosts. The greatest challenge for these species is making contact with a host; for this to happen, the host must pass through the tick's specific location. If this does not happen, the ticks die relatively quickly as a result of direct exposure to the elements. By keeping livestock, and maintaining large numbers of animals in confined spaces, humans greatly facilitated contact between this type of tick and potential hosts. Tick species with large wild herbivore hosts benefitted most. Their original wild hosts were substituted by more accessible and numerous domestic animals that remained in the same fields as the ticks.

The temperature and humidity thresholds that influence tick feeding and activity vary between genera, and in turn affect their distribution. Generally, ticks are more active in warmer seasons, provided that rainfall is sufficient. One of the key variables affecting tick survival is maintenance of the water/ion balance.



The slow insertion of the buccal apparatus into the epidermis causes no pain, thanks to the release by the tick of anaesthetic substances. The chelicerae pierce the skin, to which the hypostome is subsequently anchored.

While ingesting blood, the tick regurgitates excess liquid, thereby maintaining the water balance, and inoculates the host with substances that facilitate feeding and regulate the immune response.



While feeding on the host, ticks accumulate an excess of both water and ions. To solve this problem, they regurgitate surplus water and ions in saliva. However, during the long free-living phase, ticks face the opposite problem, and avoid dehydration thanks to the lipid composition of their external tegument and their ability to close their respiratory stigmas. Some species can even absorb water without being saturated. To avoid dehydration, ticks modify their behaviour accordingly throughout the day: when the relative humidity is very high, fasting ticks ascend to the tops of the blades of grass; when the relative humidity is low, they descend to the ground, where the humidity is greater.

2 Why do the eggs not die on the ground?

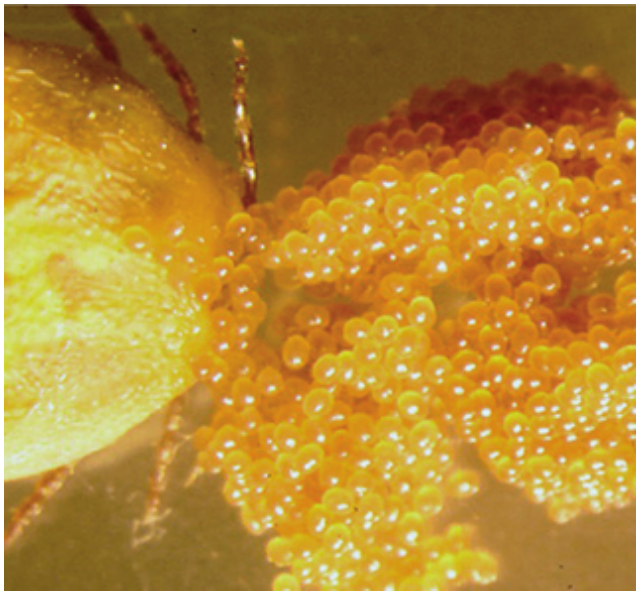
To protect eggs during periods of drought, hard ticks cover them with a substance secreted by Gené's organ or lay eggs en masse. Alternatively, the

mother may carry the eggs on her body. The production of such an abundance of eggs (up to 15,000) is a survival mechanism. Eggs deposited on the outer surface may not survive, especially in dry environments, but will serve to protect other eggs inside the mass. The mother strategically selects well protected, slightly damp areas between dead leaves in which to lay her eggs.

3 What is diapause?

Diapause is a mechanism of adaptation to adverse environmental conditions. Normally, this phenomenon is determined by the photoperiod (the number of hours of light per day), but it is also influenced by humidity and temperature.

Ticks that have not fed reduce their energetic requirements to the minimum while awaiting favourable conditions and stop searching for a host (behavioural diapause), sated larvae and nymphs delay moulting, and gravid females delay oviposition (morphogenetic diapause).



After continuously feeding for several days, during which time the ovaries mature and the female is fertilised, female hard ticks fall to the ground and lay their eggs. For several days, the female lays eggs one by one to produce a mass of several thousand, and subsequently dies.



Once they have fed, depending on whether the tick species has one, two, or three hosts, the larvae can either remain on the animal or detach and fall to the ground. For some time they remain immobile. Then, the posterior area of the cuticle is torn open and a new specimen with a larger buccal apparatus and four pairs of legs emerges.

45 What diseases do ticks transmit?

On the bulletin board of the riding school where a group of friends stable their horses, a sign appears warning of a particular type of tick. It recommends that anyone bitten should attend the local medical centre for removal and conservation of the tick, which can then be studied if the person becomes ill.

1.Can humans become infected with diseases of equids via tick bite?
2.How do ticks feed?
3.How are ticks infected?
4.How should ticks be removed?



1 Can humans become infected with diseases of equids via tick bite?

Hard or ixodid ticks are efficient vectors of diseases of humans and animals, second only to mosquitoes. They can transmit protozoan parasites *(Babesia* and *Theileria*), viruses, bacteria, and rickettsia to equids. Their efficacy as a vector is due, in part, to their hematophagous nature and their long feeding periods, which favour the ingestion of pathogens from the host. Furthermore, depending on the tick species, all three of the parasitic stages (larvae, nymphs, and adults) may feed on the same or different hosts. Many pathogens can withstand the great changes involved in tick moulting and be transmitted to the next stage (**transstadial transmission**), or can infect the oocytes, thereby passing on to the next generation of ticks, which will in turn transmit the agent when feeding (**transovarial transmission**). The high degree of adaptation of organisms and their vectors allows infectious agents to remain in their vector for long periods of time, or even pass to the vector's offspring after it has already died.

2 How do ticks feed?

Hard ticks alternate parasitic feeding periods with non-parasitic periods (moulting, oviposition, hatching, cuticle hardening, and searching for a new host). During non-parasitic periods, ticks are especially sensitive to environmental conditions, in response to which they can inhibit their activity for weeks or even months if necessary. Excluding the egg, ticks undergo three stages of development: larva, nymph, and adult (male and female). To pass from one stage to the next they must feed and moult.

Once the tick makes contact with a suitable host, it searches for and attaches itself to areas of thin skin that are inaccessible to the host. First, it pierces the skin of the host with its chelicerae, and then introduces the hypostome. Next, cement is secreted to ensure more solid attachment of the tick. Feeding involves a phase of slow and progressive ingestion, during which the females are fertilised, and another phase of rapid ingestion, during which the female and nymph undergo a marked increase in size over a short period of time. Pathogens are usually inoculated at the end of the fast feeding phase, in part because the salivary glands are very active at this time.

After a single bloodmeal lasting several days, during which the ovaries mature and the females are fertilised, female hard ticks fall to the ground where they lay their eggs. For several days, the female lays eggs individually to produce a mass of several thousand, and subsequently dies. After a variable period of time, depending on the species and environmental conditions, the eggs hatch and the larvae search for a host on which to feed. After feeding, the larvae can remain on the animal (1- or 2-host ticks) or detach and fall to the ground (3-host ticks). Next, the larvae moult into nymphs. For some time the fed larvae remain immobile. Next, the posterior area of the cuticle is torn open and a new specimen with a larger buccal apparatus and four pairs of legs emerges. After ingesting blood for several days the nymphs moult into adults. In the case of 1-host ticks, the tick remains on the animal on which it has fed and moults there. In 2- and 3-host ticks, moulting to adults occurs in the soil. The newly emerged adults must feed again to complete the biological cycle.

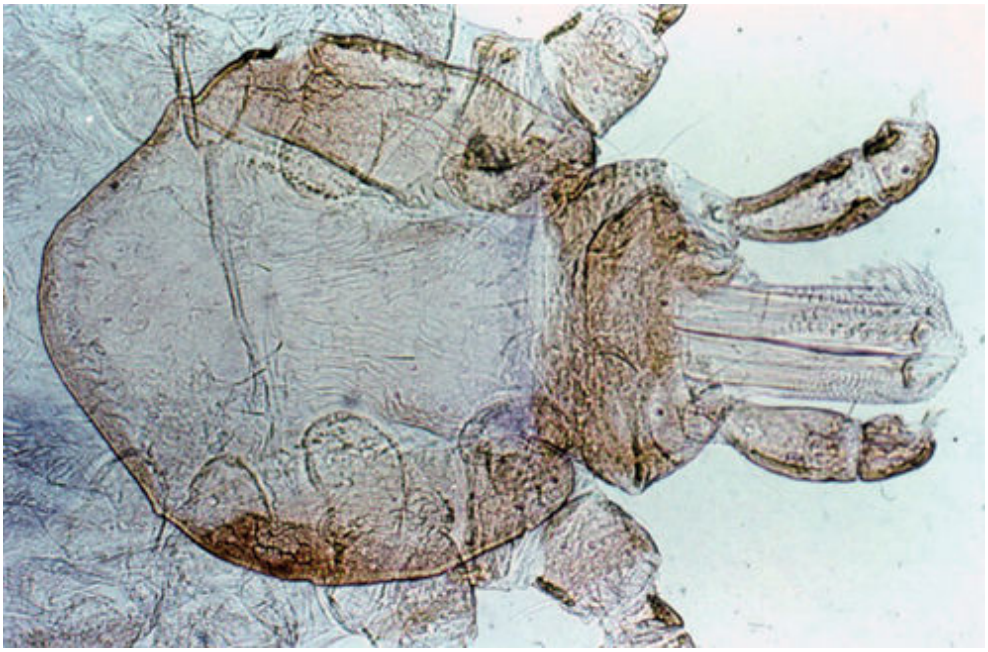
3 How are ticks infected?

In most cases arthropods are infected by parasitic invasion of the epithelial cells of the midgut during feeding. After infection, the parasitic organism must be able to remain viable until the tick or its offspring come into contact with a new susceptible host, which may take years. Transmission of the agent to vertebrates usually occurs during vector feeding. The most common route of transmission is inoculation via the saliva of the ticks. However, this is not the only possible route; infection can occur by contamination of the bite with faeces or coxal fluid (soft ticks) or by accidental ingestion of the tick (e.g. *Hepatozoon canis* in dogs).

The risk of transmission of agents of disease increases with the length of time the tick remains attached.

The distal segment of the chelicerae is used to perforate the skin. According to some authors, this process is also facilitated by the secretion of enzymes by the salivary glands. As the chelicerae tear the skin, the hypostome is inserted. In a few species of ixodids, the hypostome and the chelicerae make direct contact with the tissues. In others, approximately 10 minutes postfixation the salivary glands begin to secrete cement (a whitish liquid consisting mainly of lipoproteins), which hardens forming a tube around the appendages. The tube is open on either end. In some tick genera,

this tube widens with proximity to the skin, forming a cone of about 2–3 mm in diameter. This is usually easily removed by plucking the tick from the host. It acts as an anchoring device, isolating the mouthparts from the tissues.



At the base of the capitulum are 2 protective palps, 2 chelicerae, which are used to pierce the tissue, and the hypostome, a single, club-shaped organ with backward-facing denticles (which serve to anchor and prevent removal of the hypostome once it is embedded).

Feeding occurs in two phases. During the slow feeding phase, the tick's fasting weight increases by about 10 fold. In the subsequent fast feeding phase, the tick's weight increases by another 10 fold during its last 12–24 hours on the host. This is also the period of greatest transmission risk. Ticks that are found should be removed as soon as possible.

4 How should ticks be removed?

Generally, diagnosis is established by direct visualisation of the parasite on the animal. Tick extraction is simple, but it is important to fully remove the tick, avoiding any movements that leave the hypostome inside the dermis. Early removal of the tick (within 24 hours) is the surest way to prevent the transmission of pathogens.

To ensure complete removal of the tick, it is recommended to grasp it as close as possible to the host's skin using angulated forceps, and to then exert gentle but continuous traction perpendicular to the skin until the tick is removed. Care should be taken not to crush the tick during removal, as this can result in the inoculation of substances and pathogens from the body of the tick into the host. Once extracted, after disinfecting one's hands and the wound created by the tick, the tick is conserved in 70 % alcohol.



A fine forceps is used to grasp the buccal apparatus (pressure should never be applied to the body of the tick). The tick is removed by applying decisive and continuous traction perpendicular to the skin, avoiding sudden movements.

A disadvantage of the direct extraction method is that, if not performed properly, buccal parts can be left inside the skin, giving rise to foreign-body cysts.

The impregnation of ticks with certain substances (e.g. mineral oils, petroleum, Vaseline) is not advised; these prevent respiration of the parasite,

which in turn makes more powerful suction movements, regurgitating and inoculating more pathogens.

It is also counterproductive to burn the tick with hot objects; upon sensing the heat, the tick contracts and regurgitates the contents of its intestine (including pathogens).

46 Are ticks found during winter?

When brushing the horses at a riding club in the month of January, granulomas of 1 or 2 cm, each containing a tick in the centre, are observed at the base of the mane. After collection and transport to a laboratory, they are identified as *Dermacentor marginatus*.

1.What are the most common tick species in Spain?
2.Is it common to find ticks in winter?



1 What are the most common tick species in Spain?

Over millennia, ticks have adapted to the different climatic conditions of the planet, and are found from Arctic to desert regions. However, they have retained the general morphological characteristics of the obligate hematophagous ectoparasites of subclass Acarina, to which they belong.

Many tick species can parasitise multiple host species. For some, horses are the main hosts. Others can be found on horses but prefer to parasitise other hosts. The main species of horse ticks are *Rhipicephalus bursa* , *Hyalomma marginatum, and Dermacentor marginatus* . However, a knowledge of the other species, which can cause a variety of disorders, is also important.

- Ticks of **the genus *Dermacentor*** are easily identified by their 2-coloured scutum and whitish spots. Their distribution varies; some species are particularly abundant in areas in which others are scarce. *D. reticulatus* is widely distributed in the north of Spain, while *D. marginatus* is found in large numbers in the centre and south of the Iberian Peninsula. Both species have similar host preferences: larvae and nymphs generally parasitise small mammals, while adults feed on large mammals. The adults of both species are active in the autumn-winter period. However, the immature stages of *D. marginatus* are most active in spring, while those of *D. reticulatus* are mainly active in autumn. Both species are usually found on the heads and necks of their hosts.
- ***R. bursa*** is a medium-sized tick typically found on ruminants and equids. It has been identified throughout almost the entire Iberian Peninsula. During its life cycle it alternates between two hosts, one on which the immature forms feed, and another on which adults feed. Both hosts are usually mammals. The site of attachment of the immature stages varies considerably; they can be found in practically any area of the body, but are often found homogeneously distributed on the sides of the neck. Adults tend to attach themselves to the inguinal and perineal areas. The periods of activity are autumn and winter for larvae and nymphs and spring and autumn for adults.
- Tick species of the **genus *Hyalomma*** *(H. lusitanicum* and *H. marginatum)* are identifiable by their large size. The immature stages of both species preferably select small vertebrates (micromammals, birds) as hosts, while adult stages parasitise large mammals. They are adapted to hot and dry climates, and are therefore highly abundant in the centre and south of the

Iberian Peninsula from spring to autumn, depending on the specific conditions of each geographical area.

- ***Ixodes ricinus*** is a 3-host tick of humid, temperate/cold climates. The main factor limiting its survival in Spain is relative humidity; this species survives only a few days if the relative humidity is less than 80 %. Its distribution is therefore limited to the northern regions of the Iberian Peninsula, with residual populations found in specific habitats throughout the rest of Spain. In wetter parts of Spain, *I. ricinus* can remain active throughout the year. In other areas its activity is completely conditioned by humidity. Thus, adults can be detected throughout the year but their activity peaks during the autumn and winter months. Nymphs are active in spring and larvae about 1 month later, depending on the specific conditions of each season. Owing to these restrictions, the biological cycle of *I. ricinus* can last for several years. Normally, immature stages prefer micromammals, birds, and even lizards, and typically attach to the areas around the eyes and ears, and in some cases the tail. Adults preferentially attach themselves to the inguinal area and the medial aspect of the limbs of large mammals.
- ***Haemaphysalis punctata*** like *I. ricinus* , is found mainly in humid habitats in the north of Spain, and sporadically throughout the rest of the Iberian Peninsula. This is a 3-host tick of low specificity, and is predominantly active from autumn to spring. The immature stages feed around the ears and eyes of birds and reptiles, while the adults are found across the body surface of large mammals.
- ***Rhipicephalus (Boophilus) annulatus*** is a single-host tick found in open spaces, and has a limited distribution in Spain. It has been identified in the Balearic Islands, Galicia, and Doñana, mainly during warm, humid months. It is quite specific in terms of its choice of host, and displays a preference for domestic and wild ruminants. It can be differentiated from ticks of other genera by its short palps, which do not cover the other mouth parts.

2 Is it common to find ticks in winter?

In humid areas of Spain the most abundant ticks are adult forms of *R. bursa* in summer and immature forms of the same species in autumn and winter; *H.*

punctata in autumn; *D. reticulatus* in autumn and winter; and *I. ricinus* throughout the year.
In dry areas, ticks of the genera *Hyalomma* and *D. marginatus* are the most abundant, especially in the spring/summer months and during the winter, respectively. *R. bursa* is also common in the summer.



Dermacentor species have a highly characteristic 2-coloured scutum with whitish spots, club-shaped palps, and festoons on the posterodorsal edge of the scutum.



The buccal apparatus of *Rhipicephalus* species contains short, triangular palps, which are attached to the base of the hexagonal capitulum. In the males, large, triangular anal plates are located ventrally.

47 Can severe allergic reactions be caused by mosquitoes?

Hypersensitivity to insects is a common cause of skin conditions in horses. Mosquitoes of the genera *Culicoides* (midges) and *Simulium* (black flies), species of *Tabanus* , *Stomoxys* (stable fly), *Haematobia* (horn fly), and *Musca* (domestic fly), as well as bees and even wasps, can cause evident skin lesions in horses.

1. What type of lesions do they produce?
2. What are midges?
3. What is a black fly attack?
4. What are culicids?



1 What types of lesions do they produce?

Dermal lesions caused by Diptera include pruritus, alopecia, excoriations, and lichenification, although a generalised papular/crusting eruption may be observed in some cases. Diagnosis is based on a compatible clinical history, physical findings, and the exclusion of other causes of pruritus in horses. Skin biopsies may reveal the presence of superficial or deep perivascular eosinophilic dermatitis, intercellular oedema of the epidermis, and necrotic areas.

2 What are midges?

A midge is a female of the genus *Culicoides* , of which more than 50 fly species have been described in the Iberian Peninsula. In horses, they can

cause skin disorders known by a variety of names (e.g. Queensland itch, summer itching). The location in which *Culicoides* feeds varies depending on the species. While some prefer the dorsal surface of the body (mane and tail), face, and ears, other species prefer the lower body areas (underside of the neck, trunk, and limbs). *Culicoides nubeculosus* is one of the most important species in Spain and Portugal. These midges are active in the early hours of the morning, at twilight, during the summer in temperate areas, and throughout the year in tropical regions. The disease usually affects the ears, mane, dorsal midline, and tail of animals over 3 years of age, and is characterised by intense pruritus; the formation of small papules causes the animal to scratch violently, resulting in alopecia. The pathogenic basis of the disease is an immediate hypersensitivity reaction to the salivary antigens of midges.



Midges (*Culicoides*) are small insects that can cause allergic processes in some equids as an immediate reaction to their saliva. A hereditary component has been described, and the affected body areas correspond to the habits of the causative species (*C. pulicaris* , areas of dense hair; *C. nubeculosus* , lower abdomen).

Image courtesy of Dr J. Butler.



Black flies (*Simulium*) gather in swarms and attack at sunset, targeting the head, ears, and the ventral part of the abdomen.



Culicids are mosquitoes. Females must ingest blood to obtain sufficient protein to produce eggs and successfully reproduce. They attack during the coolest hours of the day, at sunset and at dawn.

Image courtesy of Dr A. Encinas.

Hypersensitivity reactions are clearly seasonal in cold areas, but not always in areas with temperate climates, and can affect animals of any age, sex, or breed.

The development of *Culicoides* species occurs in humid wetlands or on decomposing plant material near water. The incubation period ranges from 2

to 9 days depending on the species and temperature. In some species, the egg phase lasts throughout the winter.

3 What is a black fly attack?

Simulids can be found in abundant numbers in wooded areas, near streams of running water. They are diurnal and their radius of activity can extend 50 km from location in which they hatch. When climatic conditions are favourable (warm temperatures and decreased water flow in streams and lagoons), adults hatch en masse and swarms of females attack the same animal. They cause rapidly developing petechiae and oedema of the bitten area, particularly in the region between the chest and the udders or male genitalia, as well as small-to-medium sized urticarial papular formations. The area of the bite is covered with a drop of blood when the fly leaves the animal. Histology reveals perivascular dermatitis and eosinophilic infiltration.

Simulids require a source of clean water for development, and lay their eggs on partially submerged stones or in aquatic vegetation. Incubation occurs over several days in hot weather, but can take months in colder weather, and in some species hibernation occurs during the egg phase.



Stable flies (*Stomoxys*) prefer the lower limbs, ventral area of the abdomen, thorax, and back. They are easily differentiated from *Musca domestica* by their buccal apparatus, a proboscis covered with small teeth, which lacerate the tissues to create a small wound from which the blood is sucked.

Parasitosis is seasonal (from late spring to autumn, depending on the geographical area). Of the many species of simulids (black flies) found in the Iberian Peninsula, those that specifically target equids are *Simulium reptans* (two generations per year) and *Simulium equinum* (hibernates during the larval state). The main result of attacks by these flies is simuliotoxicosis, a syndrome characterised by generalised petechial haemorrhages, particularly in areas of thin skin, together with oedema of the larynx and abdominal wall, sometimes accompanied by tachypnoea and tachycardia. This clinical picture is less common in equids than ruminants, and is caused by the presence in the fly's saliva of a toxin that acts on the heart and blood vessels.

The flies usually feed on the legs, abdomen, head, and ears, and most species are particularly active during the morning and night in warm, cloudy

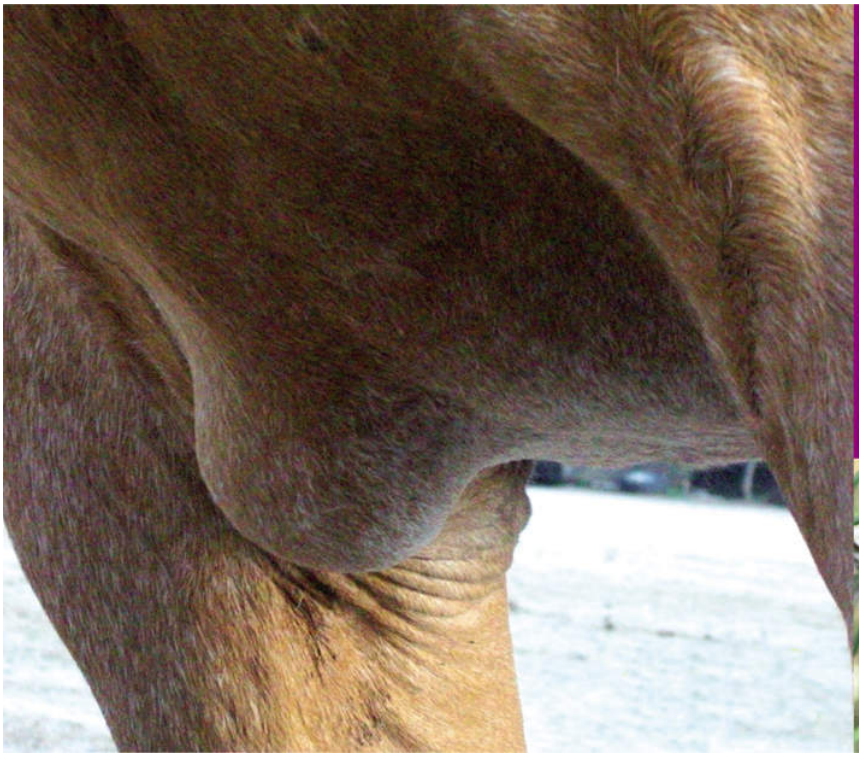
weather. Horses are bitten inside the ears.

Although adults can be found all year round, in the tropics their numbers usually increase during the rainy season. In temperate and arctic regions, adults usually die in the autumn, and new generations appear in the spring and summer. The painful stinging caused by the syntrophium of *Simulium* is irritating, sometimes causing bitten animals to stop eating and leave the pasture to seek shelter in wooded areas.

4 What are culicids?

Culicidae is a dipteran family known colloquially as mosquitoes, and includes, among others, the genera *Anopheles* , *Culex* , and *Aedes* . These are flying insects with a thin body and elongated legs. The size of the adults varies depending on the species, but they rarely exceed 15 mm. Larvae and pupae develop in water. Culicid larvae can be found from the Equator almost to the Arctic Circle in almost any body of water (however small) that remains stagnant for at least a week. In most cases, water 1-cm deep is sufficient for completion of the larval phase. Most larvae breathe via a pair of spiracles located on the penultimate abdominal segment, but in *Culex* these structures are located at the end of a small tube called a respiratory siphon. Larval maturation occurs over a few weeks or several months. In temperate climates, some species hibernate during the larval phase. Larvae are found in tremendously varied habitats, ranging from small temporary collections of water to larger bodies of water (e.g. swamps).

Attacks on grazing equids by swarms of bees can be fatal if palliative treatment is not quickly administered.





They are not normally found in large bodies of water such as lakes, or in fast flowing rivers. Adults can only fly a few hundred metres, but can be dispersed over long distances by the wind, and can even travel internationally in airplanes.

Most mosquito species feed at night, and their bites can cause considerable discomfort. The long length of their mouthparts allows them to bite humans through clothing.

48 What diseases do flies and mosquitoes transmit?

In the summer of 1987 in Spain an outbreak of African horse sickness resulted in the death of approximately 250 animals in several provinces. The initial focus was an animal reserve to which zebras and donkeys had arrived from the African continent the previous month. Within 1.5 months all equids in the reserve had died or been euthanised. The disease spread following the course of nearby rivers, and new cases were detected within several kilometres of the reserve. Mass vaccination, disinsection, and the immobilisation of animals ensured complete elimination of the focus. The climatic conditions during autumn of that year also facilitated elimination of vectors.

1. What was the role of mosquitoes in the diffusion of the disease?
2. What action should be taken in response to a localised outbreak of African horse sickness?
3. What other pathogens do mosquitoes transmit?
4. What measures are effective against Diptera in general?



1 What was the role of mosquitoes in the diffusion of the disease?

African horse sickness was limited to the African continent, south of the Sahara, until the mid-20th century, when it was demonstrated that mosquito vectors could travel by plane (resulting in outbreaks in Turkey and Cyprus in 1960). Horses are the most susceptible equids, with mortality approaching 90 % (mortality in donkeys and mules is approximately 50 %), and exhibit viraemia only during the first 2 weeks after infection. Once they overcome the disease, they do not remain as carriers, unlike zebras and African donkeys, in which the virus can be isolated in blood or tissue up to 40 days after infection in the absence of evident clinical signs. This difference is the result of the close contact maintained between the latter group of animals and the virus. There are records of a similar disease in Africa dating back to the 2nd century BC.

African horse sickness is not transmitted directly. Sick and healthy animals can be grouped together without risk. Transmission occurs via the bites of blood-sucking *Culicoides* or by inoculation of contaminated blood (0.0001 ml is sufficient).

Despite a lack of scientific evidence, imported animals were assumed to be carriers, and both *Culicoides* and the first horses to die were considered agents of transmission. The timing of outbreaks correlated with the biological cycles of both the mosquito and the virus. Fourteen days after the death of the first two horses, the rate of outbreaks increased exponentially. After ingestion of infected blood by mosquitoes, a period of 8–10 days at 25 °C is required for the virus to reach the salivary glands and replicate (producing 10,000 viral forms for every 1 ingested). In infected animals the incubation lasts the next 7 days.

2 What action should be taken in response to a localised outbreak of African horse sickness?

Importation of animals from endemic areas to disease-free zones should be restricted. In situations in which this does occur, a quarantine period of 60 days is essential given the possibility of subclinical pathologies and the potential role of native animals as possible carriers for up to 40 days.

In case of a suspected epidemic outbreak, prophylactic measures should be taken to prevent transmission by vectors while at the same time conducting the necessary studies to identify the agent of transmission.

- The first measure should be to delimit the area in which drastic measures are required, taking topography into account (mountains, lakes, and rivers can act as natural barriers).
- The next step is to immobilise the affected species within the delimited area and prevent the entry of other animals. Equids should be housed from dusk until morning in facilities designed to prevent the entry of vectors. The use of repellents and insecticides is recommended.
- Areas that favour vector multiplication should be eliminated: fields should be tilled, pastures drained, and ponds and standing water eliminated.
- To ensure early detection of affected horses, rectal temperature should be recorded daily upon stabling in the evening and before release in the morning.

- Sick animals should be completely isolated or euthanised.
- The entire equine population should be vaccinated en masse with a polyvalent vaccine until the agent has been identified, after which the appropriate monovalent vaccine can be administered and will induce an effective and lasting response within 3 weeks.

3 What other pathogens do mosquitoes transmit?

It is necessary to differentiate mechanical from biological transmission. In the latter, multiplication of the infectious or parasitic agent within the vector enhances the infective capacity of the virus.



In enzootic zones, indigenous equids carrying infectious agents in their blood can act as disease carriers when they develop subclinical pictures with few evident manifestations.

Culicoides mainly transmit *Onchocerca* spp. and arboviruses (ARthropod–Borne viruses), such as the agent of African horse sickness, to horses. *Simulium* can transmit filarias such as *Onchocerca* spp.

Culicidae (not to be confused with the genus *Culicoides*) are known mainly as vectors of malaria (*Plasmodium* spp.) in humans. The most

common species of the genera *Anopheles* , *Culex,* and *Aedes* are important vectors of several arboviruses, including those that cause Eastern, Western, and Venezuelan encephalitis in horses. Culicids can transmit *Setaria cervi* to horses.

In addition, flies, especially biting flies, can mechanically transmit parasitic agents (e.g. *Trypanosoma evansi* , which can be transmitted by *Stomoxys calcitrans*) and infectious agents (equine anaemia virus). Some Muscidae species act as intermediate hosts of equine helminths (*Musca domestica* is implicated in the biological cycle of *Habronema muscae* and *Draschia megastoma* , while *S. calcitrans* is an intermediate host of *Habronema microstoma*).

4 What measures are effective against Diptera in general?

Knowledge of the biological requirements of the species greatly aids the establishment of control programmes. Species of the genus *Culicoides* tend to propagate in stagnant waters, while *Simulium* species and horse-flies do so in moving water (rivers and streams). Most common and stable flies require decomposing organic matter for reproduction, and thus are more abundant in stables with suboptimal hygiene/sanitation.

The time of day at which insect attacks peak also differs between species. Swarms of *Simulium* and stable flies tend to be more active during daylight hours, while mosquitoes (*Culicoides* and Culicidae) prefer to feed at dusk or just before sunset. Based on this knowledge, animals with proven hypersensitivity to a particular insect can be stabled at the appropriate time.

Ideally, animals should be stabled during the hours of maximum risk, windows covered with a fine mesh, and a residual insecticide applied using an atomiser.

Stagnant water and faecal matter, in which larval stages of flies can feed, should be removed from the bedding.

Many equids are sensitive to petrolatum-based products, which can cause skin erythema and hair loss in the areas to which insecticide ointments are applied. Application of these products when the skin is cold and dry appears to minimise the risk of sensitivity reactions. A patch test should always be performed before applying a new product to the entire body surface.

In some countries, Simuliidae attacks force the stabling of animals during periods of massive swarms. If this is not possible, it is essential to mechanically protect animals using masks, covers, or blankets, as well as insecticide-impregnated strips or bands attached to the mane, tail, or harness.

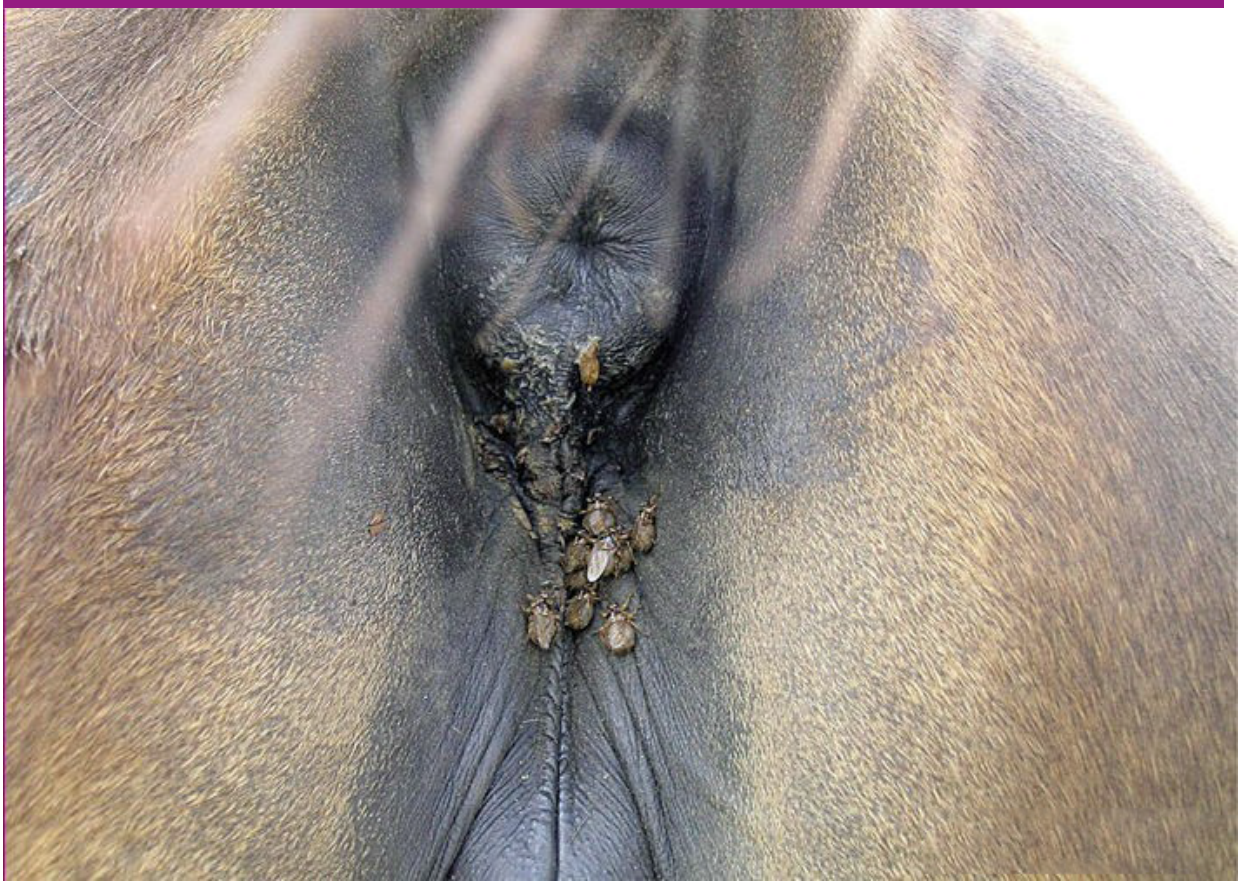
Main arthropod vectors that attack equids.

Arthropod vectors	Infectious agents that can be transmitted
Cyclorrhapha (flies)	
Stomoxys (stable fly)	*Trypanosoma, Habronema, Setaria,* bacteria
Musca (common fly)	*Habronema,* viruses
Hippobosca (forest fly)	Bacteria, viruses
Nematocera (thread horns)	
Culicoides (midges)	*Onchocerca,* viruses
Simulium (black flies)	*Onchocerca*
Culicids (mosquitos)	*Setaria,* viruses
Brachycera (horse-flies)	
Tabanus, Haematopota (clegs) *Chrysops* (deer fly)	*Trypanosoma,* bacteria, viruses

49 Why does the forest fly not fly?

A visit to Doñana in the month of June coincides with an annual local event known as “La Saca”. Riders on horseback enter the marshes to round up mares and foals, which they then bring to the esplanade in front of the village of El Rocío. The horses are then transferred to an enclosure for identification and possible sale. Large numbers of ectoparasites are observed on the animals. Strikingly, the flies do not appear to fly around the horses.

1.What is *Hippobosca equina* ?
2.What pathogens can it transmit?



1 What is *Hippobosca equina* ?

Hippoboscidae are flies of low pathogenic potential with a thin, flattened, segmented and generally soft and leathery abdomen. The two large eyes are well separated on the head. They feed on blood using beak-like mouthparts, and adhere to the hairs of animals with strong nails on their legs. The abdomen can be very dilated following ingestion of blood or when the female is gravid.

The forest fly *(H. equina*) has a worldwide distribution and is easily identified. The adults are approximately 1 cm long and are usually pale brown with yellow dots on the abdomen, the segmentation of which is relatively inapparent. They have large, reddish wings that extend past the edges of a round, dorsoventrally flattened abdomen.

This same family includes *Melophagus ovinus* , a parasite of sheep that has lost its wings and halteres since adapting to parasitism. Unlike *Hippobosca* , this species cannot fly. It behaves like a louse, and is transmitted by direct contact.

Members of this family are classified together with *Glossina* (the African tsetse fly) in a separate suborder, Pupipara, because the females produce mature larvae that rapidly transform into pupae. The female leaves her host and deposits a mature larva on dry soil or humus. Each female can produce 5–6 larvae during her lifetime. These larvae pupate immediately and turn from yellow to black. Maturation of the adults depends on the temperature. In temperate areas adult forms are more abundant in the summer months.

Forest flies remain on their host for long periods of time, mainly on the perineum and the backs of the legs.

Some horses appear to become accustomed to attacks by these flies, several hundred of which can swarm around the perineum without causing significant discomfort. However, they can cause great irritation in animals unaccustomed to these attacks. They cluster in groups under the tail, around the perineum and genitals, on the inner aspect of the thighs, and even the lower abdomen and other areas of thin skin, where they can cause mild cutaneous lesions and extract small amounts of blood (6–30 ml) over about 15 minutes. They move with great agility, marching backwards and sideways, and rarely fly.

2 What pathogens can it transmit?

Because they pierce the skin to suck blood, they can act as vectors of blood parasites. They are known to act as vectors of equine infectious anaemia virus and bacteria, including *Clostridium perfringens* . They can also bite cattle, dogs, and humans, giving rise to a hard, pruritic papule that persists for about 2 weeks.



Hippobosca equina is a blood sucking Diptera species that produces larvae capable of pupating immediately in the soil or in crevices. It displays several adaptations that facilitate its parasitic life cycle; it has a flattened body and small claws at the end of each leg with which it attaches to the host.

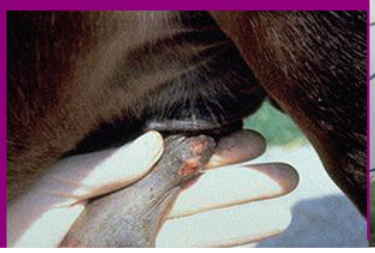


Melophagus ovinus. This hippoboscid spends its entire life on the host, and has lost the ability to fly (i.e. it is an apterous Diptera species).

50 Which parasite impairs covering in stallions?

A stud in the town of Jerez de la Frontera purchases a new stallion of almost 5 years of age from Cádiz, in the south of Spain. When covering the mare, the stallion appears not to adapt to the new environment, systematically refusing to cover, despite having done so previously without problems. After a period of adaptation and multiple failures, a veterinary surgeon is called, who, upon retracting the foreskin to the base of the penis, detects a painful, granulomatous lesion of several centimetres that bleeds easily.

1.What parasite can cause this type of lesion? How is it transmitted?
2.How is this lesion caused?
3.Can it be found in other areas of the body?





1 What parasite can cause this type of lesion? How is it transmitted?

This is a parasitic infection caused by larvae of the nematodes *Habronema* and *Draschia* . These are not fly larvae. Adults are found in the stomachs of equids, but rarely cause gastric problems; the most important clinical consequence is a cutaneous alteration known colloquially as “cutaneous habronemosis” or more commonly “summer sores”. These parasitoses are distributed worldwide, and are more common in warm climates where the flies that act as intermediate hosts are more prevalent. Incidence is seasonal, peaking in warm months and disappearing during the winter.

2 How does this lesion occur?

In skin wounds, *Draschia* is the most common of the two species. Larvae deposited in the wound remain in it, feeding and migrating, and exert an irritant action that prevents normal wound healing. In the early stages, inflammation causes intense pain and itching in the affected area. It is common for horses to exacerbate the process by biting or rubbing against objects, often resulting in secondary bacterial and/or fungal invasion. The lesions are characterised by a very intense granulomatous reaction, and can increase in size to up to 8 cm. It is assumed that the pruritus is secondary to a hypersensitivity reaction, which ranges from mild to severe. The lesions are frequently ulcerated, with yellowish granules of approximately 1 mm in which no branching is observed, like in pitiosis or zygomycosis.

The lesion begins with the formation of small papules that erode and increase in size, reaching up to 30 cm. The central part of the lesion is slightly depressed and consists of granulation tissue covered by a greyish necrotic membrane. If left unattended, the lesion becomes chronic, proliferative, granulomatous, and bleeds easily.

Initially, the pruritus is intense, causing animals to scratch and rub and resulting in abrasions. The prominent reddish granuloma can become inactive and fibrotic, but it does not heal until the next cold season, when the vectors disappear.

If the larvae are deposited in the conjunctival sac, their irritant action produces granular conjunctivitis with the development of polypoid nodules that tend to form yellowish plaques in the palpebral and vulvar conjunctiva. Blepharitis and palpebral granulomas develop in some cases. Larvae deposited in the nostrils can ascend through the nasal mucosa and reach the

lungs, giving rise to peribronchial nodules of about 0.5 cm in which the larvae die.

3 Can it be found in other areas of the body?

Cutaneous habronemosis lesions appear seasonally, depending on the time of year during which vectors are active. They are known as “summer sores”, since they are most prevalent during the summer. They heal spontaneously at the end of autumn, during the colder months, when the flies are inactive and the larvae die in the wounds. These are recurrent processes that reappear in the hotter months of the summer. They are more frequent in areas of the body that are easily injured by exercise or the use of equipment such as harnesses and saddles (e.g. the inner aspect of the limbs, coronary band, back, and face). The medial canthi of the eyes, the third eyelid, and the genitals of the males are other commonly affected locations.



Nematodes and flies evolve synchronously: the fly larva ingests the eggs in the faeces and by the time the adult fly emerges from the puparium it carries the third-stage larva inside it. When the fly begins to

feed, the larva migrates to the proboscis, from where it invades the tissues of the host while the fly feeds.

When a wound is contaminated with nematode larvae (*Habronema* or *Draschia*) deposited by flies, the granulomatous reaction prevents healing.



Image courtesy of Gabriel Mansó.