Question Bank

ILLUSTRATED OBJECTIVE TOXICOLOGY FOR VETERINARY EXAMS

As per lastest VCI MSVE 2008 syllabus

Dr. ALPHA RAJ.M

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Price: Free

DEDICATION

Dedicated to my beloved parents

Late. Sri. M. Deva Rajulu & Smt. N.G.Nirmala Devi

And my doctoral guide

Prof.A.Gopala Reddy

University Head of Pharmacology & Toxicology

Sri Venkateswara Veterinary University

PREFACE

"Freely you have received; freely give". Mathew 10:8.

Illustrated objective toxicology for veterinary exams is an attempt to provide comprehensive quick reference for various examinations. However, it should be noted that this e-book serves only as a supplement and not as a replacement for text book and class room learning.

This e-book covers all the aspects of veterinary toxicology prescribed by latest Veterinary Council of India (VCI) minimum standards of veterinary education (MSVE) 2008. It is a robust question bank comprising of over 777 objective questions covering 32 lecture outlines. A chapter is devoted on residue toxicology owing to contemporary importance of the topic. Over 108 illustrations were used at appropriate places to improve the lucidity and understanding of the student. Appropriate type of objective questions were used to fit the information being conveyed. Further, the objective type questions are so framed to convey maximum information possible. Explanations and additional information are provided where ever deemed necessary.

As this e-book is mainly meant to be read on a computer, laptop, mobile or tablet, navigation is provided in every page to reach appropriate locations elsewhere in the book. Any corrections and suggestions for improvement of this e-book are most welcome.

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CHAPTER I

FUNDAMENTALS OF TOXICOLOGY

1.1 Introduction

1. The branch of science which deals with the harmful effects of physical and chemical agents on human and animal life is TOXICOLOGY.

2. In the term toxicology, the word '*toxicon'* (Greek) means POISON.

3. The branch of toxicology, which deals with diagnosis, treatment and management of toxic substances, is known as CLINICAL TOXICOLOGY.

4. The branch of toxicology, which is involved in establishing safety limits for chemical exposure is REGULATORY TOXICOLOGY.

5. Investigating and controlling the toxic effects of various substances on the community is dealt by TOXICOVIGILANCE.

6. The branch of toxicology, which investigates vetero-legal cases of death, poisoning and drug abuse is FORENSIC TOXICOLOGY.

7. The study of toxicity produced by substances of plant, animal and microbial origin is termed as TOXINOLOGY.

8. What is the difference between 'Poison', 'Toxicant' and 'Toxin'?
   Ans: Poison is a broader term which includes any / every substance causing harmful effects to living beings. Toxicant is a synonym for poison.Toxins are a sub-category of poisons, which could be of plant (Phytotoxins), fungal (Mycotoxins), animal (Zootoxins) or bacteria (Bacteriotoxins – Endo & Exotoxins) origin.

9. A foreign chemical substance, which is not normally produced in the body and which may form a part of the food, is known as XENOBIOTIC.

10. How 'Toxicosis' differs from 'Toxicity'?
    Ans: Toxicosis and Toxicity as related as 'effect' and 'degree/extent of the effect'. Toxicosis is the disease or condition (effect) which results due to exposure to a poison, whereas, 'toxicity' is the degree of the disease or condition. However, the term toxicity is also used to mean adverse effects of a poison.

11. Which of the following toxicity can occur due to single exposure?
a. Acute toxicity b. Sub-acute toxicity c. Sub-chronic toxicity d. Chronic toxicity
Ans: a. Acute toxicity; Acute toxicity also occurs due to multiple exposures within 24h. The other types of toxicities definitely need multiple/repeated exposures.

12. Toxicity occurring due to repeated exposure within 30 DAYS or LESS is termed as sub-acute toxicity, whereas, within 1 TO 3 MONTHS is termed as sub-chronic toxicity.

13. If the period of exposure of a toxicant is more than 3 months, the type of toxicity is termed as CHRONIC TOXICITY.

14. Repeated oral toxicity of a compound is tested in rats for a period of 28-days. What kind of toxicity is being studied here? (You need to answer this question!!)

15. The type of toxicity which results due to *progressive* accumulation of a toxicant in the body is known as CUMULATIVE TOXICITY.
(Several toxicants such as heavy metals, alcohol, DDT etc cause cumulative toxicity.)

16. The amount of toxicant in food and water, which can be consumed daily over a life time without any significant health risk, is known as ACCEPTABLE DAILY INTAKE (ADI).

17. What is the difference between Lethal Dose ($LD_{50 \text{ or } 99}$) and Lethal Concentration (LC)?
Ans: Both LD and LC causes death in exposed population. However, LD refers to the 'lowest dose' of the toxicant administered directly to the animal in any route. LC refers to the 'lowest concentration' of toxicant present in feed and water. (In LD, the compound is administered directly to the animal on mg/kg bd wt basis, whereas, in LC the toxicant is present in feed or water (mg/kg of feed or Liter of water).

18. What is the meaning of the subscripts in the terms $LD_{50 \text{ or } 99}$ and LC_{50}?
Ans: The subscripts indicate the % of mortality (deaths) in exposed population. In 1927, J.W.Trevan introduced LD_{50} to determine the toxic potential of various compounds.

19. For a given compound, between NOEL and NOAEL, which one will be higher in dose?
Ans: NOEL (No observed effect level) is the highest dose, which will not produce any effect, whereas, NOAEL (No observed adverse effect level) is the highest concentration, which will not produce any adverse effect. Since, the development of adverse effect needs higher dose than minor effects, NOAEL is higher than NOEL.

20. Maximum acceptable/ permitted amount of a drug present in feed and foods is known as MAXIMUM RESIDUE LEVEL (MRL). (For pesticides – MAXIMUM RESIDUE LIMIT).

21. The highest dose of a compound, which produces adverse effects but no mortality is called MAXIMUM TOLERATED DOSE (MTD).
(MTD is also referred to as $LD_{0\ (Zero)}$ as it will cause adverse effects but no mortality).

22. For a given compound, between MTD and NOAEL, which one will be higher?
Ans: NOAEL is the highest dose which will not cause any adverse effect whereas MTD refers to the highest dose which will cause adverse effects. Hence, MTD is higher.

23. What is the relationship between the various toxicity doses with respect to a given compound?
Ans: *LD > LC > MTD > NOAEL > NOEL > ADI > MRL*

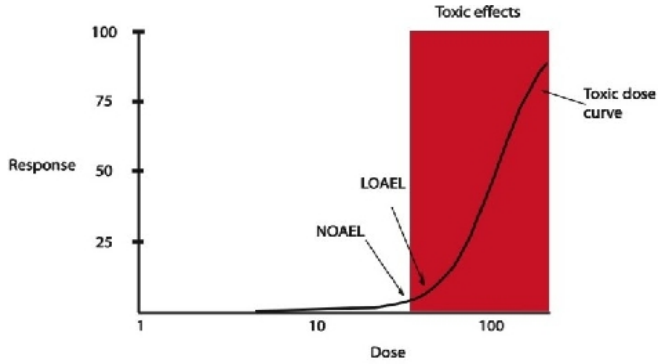


Fig.1 No-observed adverse effect level [1]

24. The source of adverse effect/ damage is known as HAZARD.
(Eg: Water containing Fluoride; Here fluoride is the hazard and fluorosis is the adverse effect. Hazard is independent of dose or exposure i.e.,Fluoride is present whether someone drinks the water or not).

25. The likelihood or probability of adverse effect upon exposure to a hazard is known as RISK.
(Eg: While drinking water containing fluoride, the chances of getting fluorosis is called risk. Risk can range from 0 to 100% depending on dose and exposure i.e., one should be exposed to a hazard to calculate the risk).

26. The statement, “all substances are poisons; the dose differentiates poison from a remedy” is associate with PARACELSUS.

Fig: 2 Theophrastus Paracelsus Bombastus Von Honheim (1493-1541) [2]

27. The scientist, who is referred to as 'Father of Toxicology' is M.J.B. ORFILA.
(He established toxicology as a separate discipline; defined the term toxicology; advocated the use of chemical analysis in forensic toxicology).

Fig:3 Father of Toxicology - Mathieu Joseph Bonaventure Orfila (1787-1853) [3]

28. DDT (Dichloro diphenyl trichloro ethane), which is used to control malaria and typhus was discovered by PAUL MULLER.

Fig:4 Paul Muller (1899-1969) – Synthesized DDT [4-5]

29. The scientist, who is known as 'Father of nerve agents' is GERHARD SCHRADER.
(Nerve gases such as Sarin, Tabun, Soman, Mustard gas etc were used in World War II and by Iraqi troops on Kurds as a part of chemical warfare).

30. The author of the book 'Silent Spring' in which the detrimental effects of DDT and other pesticides on environment – particularly on birds was documented - is RACHEL CARSON.
(Springs are pleasant with the noises of birds. But due to the use pesticides, the bird population is considerably reduced. Hence, the springs are becoming silent. This book created a silent campaign against the use of pesticides and led to a ban on DDT worldwide; US Environmental agency was setup in this context.)

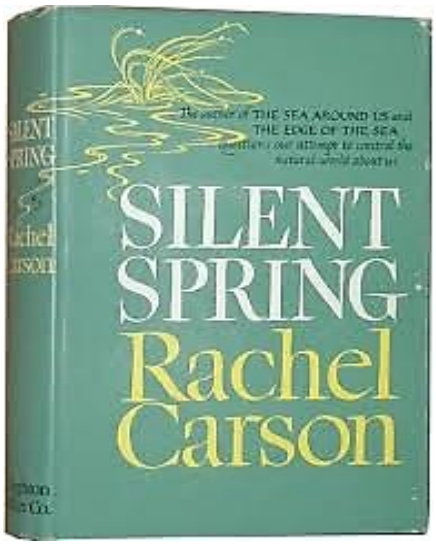


Fig:5 Rachel Carson (1907-1964)–Author of Silent Spring–Campaign against DDT [6-7]

31. Bhopal gas tragedy, which is considered as the world's worst industrial disaster, was caused by the leakage of METHYL ISO-CYANATE (MIC) gas from Union Carbide fertilizer company.

32. Use of thalidomide in pregnant women for treating morning sickness caused PHOCOMELIA condition in newborn infants.
(Phocomelia is a condition in which an infant is born with malformation of limbs. Thalidomide was not properly tested for safety and hence caused thalidomide disaster worldwide affecting almost 10,000 children)

Fig: 6 Thalidomide tragedy – Phocomelia in children[8]

33. The type of necrosis, which is commonly seen in liver due to various toxicants is CENTRILOBULAR NECROSIS.
(Centrilobular region around the central vein (zone 3) has poor oxygenation and is rich in P450 cytochrome (responsible for metabolism of various drugs – needs more oxygen). Hence, this region is more prone to damage than peri-portal region (around portal triad - zone 1) which is more oxygenated.)

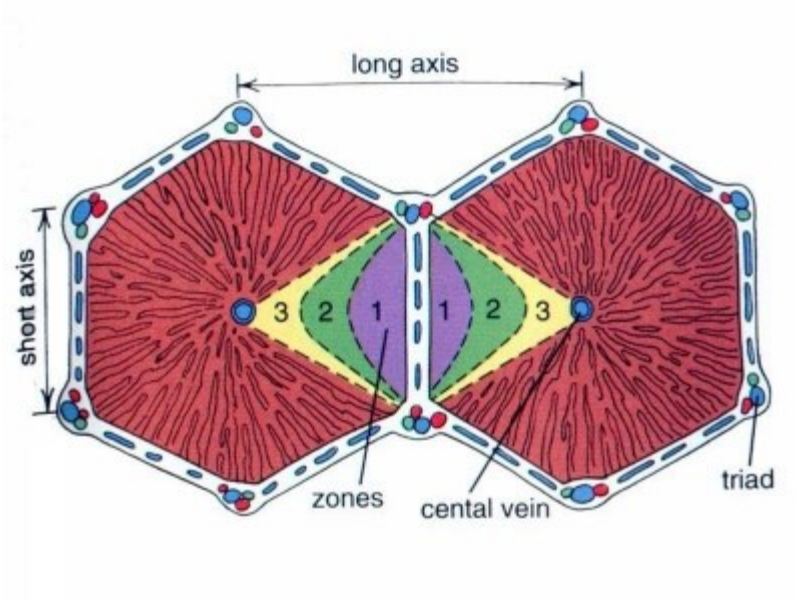


Fig:7 Structure of Liver lobule. Zone 3 around central vein is less oxygenated [9]

1.2 Metabolism, Mechanisms and Factors Affecting Toxicity

1. Unlawful or criminal killing of animals through administration of poisons is known as MALICIOUS POISONING.

2. Unintentional addition of toxicants or contaminants to feed and water is known as ACCIDENTAL POISONING.

3. Man-made sources of toxicants are referred to as ANTHROPOGENIC sources.

4. Genetically determined abnormal reactivity of an individual to a chemical is known as IDIOSYNCRACY.

5. Failure to elicit a response to an ordinary dose of a substance due prior exposure is known as TOLERANCE.
   (*Tolerance is generally caused due to the induction of metabolizing enzymes in liver. However, in case of chronic alcoholism, pseudo-tolerance is observed, due to thickened GIT mucosa, which decreases absorption*).

6. The phenomenon in which toxic substances elicits beneficial effects at low doses is known as HORMESIS.

7. For which of the following route of exposure, pre-systemic elimination is possible?
   a. Oral b. Inhalation c. Intramuscular d. Intravenous
   Ans: a. Oral; GIT and liver are responsible for elimination prior to entry into circulation.

8. In the event of irreparable injury, the cell undergoes a process of programmed cell death known as APOPTOSIS.
   (Apoptosis is also involved in number of physiological process such as embryogenesis, ageing, cancer prevention etc)

9. A substance is classified as extremely toxic if the lethal dose (LD) is LESS THAN 1mg/kg and as practically non-toxic if the LD is 5 to 15 g/kg.
   (It should be remembered that more the LD of a compound, less is its toxicity. Highly toxic –1 to 50 mg/kg; Moderately toxic – 50 to 500 mg/kg; Slightly toxic – 0.5 to 5 g/kg).

10. The ability of a substance to induce cancer is known as CARCINOGENESIS.

11. The common process involved in the absorption of xenobiotics across the cell membrane is PASSIVE DIFFUSION.

12. In body, heavy metals such as mercury, lead, cadmium tend to accumulate in KIDNEY (organ of the body).
    (However, the major site for accumulation of lead in the body is bone and teeth).

13. Organo-chlorine insecticides such as DDT tend to accumulate in ADIPOSE TISSUE (organ of the body).

14. Arsenic tends to accumulate in HAIR and SKIN (organ of the body).
    (Hence, bacterial decomposition of carcass is absent resulting in preserved carcass)

15. Major route of excretion for xenobiotics is RENAL EXCRETION.

16. The process of chemical transformation (conversion from one form to another) occurring in the body is known as BIOTRANSFORMATION.

17. The major site for biotransformation of xenobiotics in body is LIVER.
    (Liver is the major site due to the presence of variety of metabolizing enzymes. Other important sites for biotransformation include lung, kidney and intestines)

18. In a hepatocyte, metabolism of xenobiotics takes place in ENDOPLASMIC RETICULUMN (ER) or MICROSOMES.

19. Most important microsomal enzymes involved in biotransformation are MONOOXYGENASES or MIXED FUNCTION OXIDASES (MFO).

20. Major biotransformation reaction occurring in Phase I is OXIDATION and in Phase II is CONJUGATION.

21. Phase I Oxidation reactions are mainly catalyzed by MICROSOMAL ENZYMES (MFO).

22. All Phase II conjugation reactions are catalyzed by non-microsomal enzymes except for GLUCURONIDE CONJUGATION, which is catalyzed by microsomal enzymes.

23. The ability of certain substances to increase the activity or synthesis of microsomal enzymes is known as INDUCTION.
(Non-microsomal enzymes which are present in cytosol are not inducible)

24. The metabolic reaction that is deficient in dogs is ACETYLATION; in cats is GLUCURONIDE CONJUGATION, and in pigs is SULFATION.
(Hence, sulphonamides which undergo acetylation cause toxicity in dogs and similarly, paracetamol, which undergoes glucuronide conjugation, causes hepatotoxicity in cats).

25. The process of conversion of non-toxic substance into a toxic metabolite through biotransformation is known as LETHAL SYNTHESIS.

26. Organo-chlorine insecticides such as DDT, BHC tend to be more toxic in oily vehicles due to INCREASED ABSORPTION.

27. Elemental mercury is toxic when exposed through INHALATION route.
(Ingested mercury is not likely to cause toxicity; But is toxic through inhalation route. Guess, which of the following situations is more dangerous: a child chewing on a thermometer and ingesting mercury or a thermometer falling on to the floor leading to evaporation of mercury?!).

28. If the action of one substance opposes or neutralizes the effect of another substance, the relationship is referred to as ANTAGONISM.
(All antidotes have antagonistic relationship with their respective toxicants)

29. Rodents are preferred for oral toxicity testing as they lack VOMITION reflex.

30. The species of animal that is resistant to the toxic effects of consuming *Atropa belladonna* (Belladonna) leaves is RABBIT.
(Rabbits contain the enzyme atropinase, which destroys atropine).

31. The breed of dog that is more susceptible to the toxic effects of ivermectin is COOLIE BREED.
(In coolies, ivermectin easily crosses blood brain barrier and causes neurological symptoms).

32. Asprin and sulphonamides cause hemolysis in individuals deficient in the metabolic enzyme GLUCOSE-6-PHOSPHATE DEHYDROGENASE (G6PD).

Fig: 8 Coolie Breed of Dog – More susceptible to ivermectin toxicity [10]

33. Why Greyhounds are more susceptible to the toxic effects of barbiturates (used for anesthesia)?
    Ans: Barbiturates mainly distribute to adipose tissue. Since, Greyhounds, have little body fat, higher circulating concentration of barbiturates causes toxicity.

Fig: 9 Greyhound Dog Breed – More susceptible to barbiturate toxicity [11]

1.3 Diagnosis & Treatment of Poisoning

1. The type of evidence obtained at the scene of poisoning is referred to as CIRCUMSTANTIAL EVIDENCE.

2. The most common feed contaminant that can be expected during improper storage is MYCOTOXINS (Aflatoxin).

3. Pink colouration of urine is suggestive of poisoning with PHENOTHIAZINES.

4. Phenols and cresols produce GREEN colouration of urine.

5. The symptom or lesion that is characteristic to a particular toxicant is known as PATHOGNOMONIC (symptom or lesion).

6. The evidence that is obtained during postmortem examination is known as PATHOLOGICAL evidence.

7. Bitter almond smell of ruminal contents is suggestive of CYANIDE POISONING.
(Bitter almond smell

8. Poisoning with phosphorus results in GARLIC-LIKE odour during postmortem examination.

9. Detection of toxic material in body using laboratory methods constitutes ANALYTICAL evidence.

10. The evidence that is obtained by feeding suspected material (feed) to healthy animals to ascertain the presence of a toxicant is known as EXPERIMENTAL EVIDENCE.

11. The aim of treatment during poisoning is to INCREASE the threshold of the toxicant.
(Measures are directed to create a situation where more toxicant is required to produce the toxicity)

12. The time versus concentration curve of toxicant in the body is BELL OR INVERTED 'U' shaped.

13. The ascending phase of the time-concentration curve of the toxicant represents ABSORPTION.

14. The descending phase of the time-concentration curve of the toxicant represents EXCRETION.

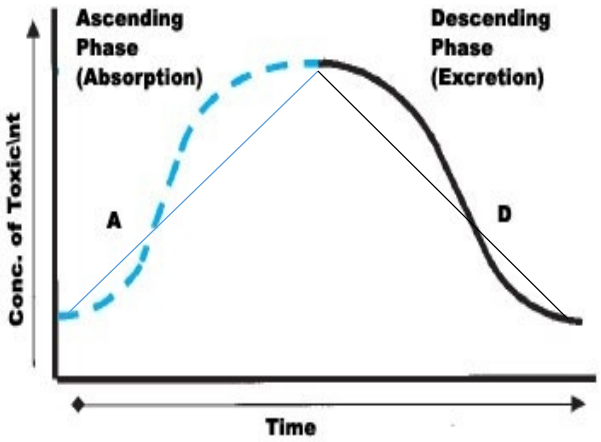


Fig: 10 Time vs. Concentration curve of toxicant in body during poisoning [12]

15. The following is the aim of treatment in poisoning
    a. Increasing the slope of ascending phase (AP) and decreasing slope of descending phase (DP).
    b. Decreasing the slope of AP and increasing slope of DP.
    c. Increasing the slope of AP and increasing slope of DP.
    d. Decreasing the slope of AP and decreasing slope of DP.

Ans: b. Decreasing the slope of AP and increasing slope of DP. When the slope of AP is decreased, it takes longer time for the toxicant to be absorbed. And increasing the slope of DP causes rapid elimination in a short time.(Fig:3)

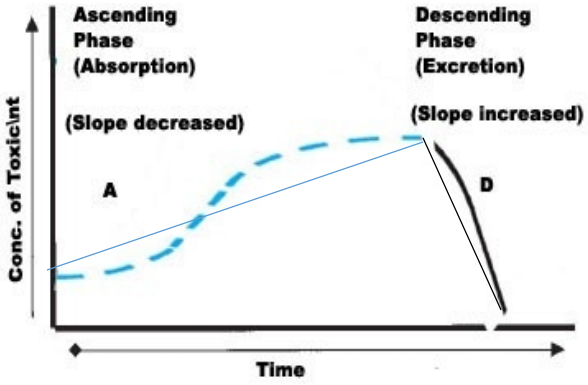


Fig: 11 Time vs. Concentration curve of toxicant in body during treatment. [13]

16. Emesis is contraindicated in the following poisoning(s)
    a. Volatile compounds b. Corrosives c. Convulsants d. CNS stimulants
    Ans: All of the above. In addition, emesis is not indicated in unconscious patients, in cases of respiratory distress or debilitated animals.

17. The safest alternative when emesis is contraindicated is GASTRIC LAVAGE.
    (In case of ruminants, rumen lavage or rumenotomy can be performed)

18. The most commonly used adsorbing agent for binding toxicants in GIT is ACTIVATED CHARCOAL.
    (Charcoal should be activated through burning (oxidation) which increases the number of pores and thus the surface area).

19. The type of diuretics or purgatives that are preferred in cases of poisoning are OSMOTIC/SALINE TYPE.
    (Osmotic or saline type of agents are the only category which actually drag water from the tissues. However, for prompt action, furosemide, which is a loop diuretic is also used).

20. When large amounts of toxicant is absorbed or when renal failure ensues, the method of choice employed for elimination of the toxicant is DIALYSIS.

21. The mechanism involved in the enhanced elimination of acidic agents in alkalized urine and basic agents in acidified urine is ION TRAPPING.
(Ion trapping for basic drugs occurs in the acidic pH of rumen - due to the production of volatile fatty acids).

22. The substance that counteracts or neutralizes a toxicant is known as ANTIDOTE.

23. Ethanol acts as an antidote for methanol poisoning through COMPETITIVE INHIBITION (Mechanism).

24. The non-specific antidote for alkaloids is POTASSIUM PERMANGANATE.
($KMNO_4$ acts by oxidizing the alkaloids)

25. The antidote for paracetamol (acetaminophen) toxicity is N-ACETYL CYSTEINE.
(Paracetamol toxicity is common worldwide and is leading cause for acute liver failure in UK and US. Continuous use for a week is likely to cause hepatotoxicity. The toxicity is due to the depletion of liver stores of glutathione, which is required for conjugating the metabolite of paracetamol).

26. The agents that are used during CNS depression and respiratory arrest are called as ANALEPTICS. (Eg. Doxapram).

CHAPTER II

TOXICOLOGY OF METALS

2.1 Arsenic

1. The metallic poison, which is considered as 'king of poisons and poison of kings' is ARSENIC.
   (Arsenic (and Thallium) causes severe GIT irritation unparalled by any other poison. Hence, arsenic is considered 'King of poisons'. Since, arsenic was extensively used by kings to eliminate competitors, it is also known as 'Poison of kings'.

2. Which form of arsenic is more toxic? Why?
   Ans: Arsenites (As^{3+} or trivalent) are 5-10 times more toxic than Arsenates (As^{5+} or pentavalent) due to higher solubility.

3. Most toxic gaseous form of arsenic which is released during charging of storage batteries is ARSINE.

4. Least toxic form of arsenicals are ORGANIC ARSENICALS.

5. Drinking water containing >0.25% of arsenic is potentially toxic to large animals.

6. Arsenic contamination of ground water is endemic in WEST BENGAL state of India.
   (Arsenic contamination of ground water is also seen in 70 countries including USA).

7. Which arsenical compound is commonly employed for malicious poisoning?
   Ans: Arsenic trioxide

8. The managemental practice that can cause arsenic poisoning in sheep is DIPPING.
   (Generally, arsenic containing compounds [Eg. Sodium arsenite; Lead arsenate] are used for dipping).

9. The species that is more sensitive to arsenic poisoning is CAT.

10. Why arsenic tends to accumulate in keratin rich tissues such as hair and nails?
    Ans: Arsenic has high affinity for sulphydryl groups (-SH). Since, hair and nails contain –SH rich keratin, arsenic accumulates in them.

11. Arsenic undergoes METHYLATION biotransformation reaction in the body.

12. Is arsenic cumulative in animals?
    Ans: No; Arsenic is rapidly detoxified and is completely eliminated within few days.

13. The mechanism of toxicity of arsenic involves binding to SULPHYDRYL GROUPS (–SH) (Eg: Lipoic (thioctic) acid).

14. The primary symptom acute arsenic toxicity is GASTROENTERITIS.

15. The nature of diarrhoea in acute arsenic poisoning is described as RICE WATERY.

16. The characteristic coloration of mucosa in hronic arsenic poisoning is BRICK RED.

17. Why arsenic causes abortions but not nervous symptoms?
Ans: Arsenic can cross placental barrier. Hence can cross abortions but it cannot cross blood brain barrier (BBB), hence, is unable to cause nervous symptoms.

18. In which species, organic arsenicals cause nervous symptoms?
Ans: Swine. Nervous symptoms include ataxia, incoordination etc.

19. Which is the only arsenical that can cause blindness?
a. Arsenic trioxide b. Arsenic pentoxide c. Arsine d. Arsinilic acid
Ans: d. Arsinilic acid.

20. What are the prominent postmortem findings in arsenic poisoning?
Ans: Preserved carcass (absence of bacterial decomposition); Severe gastroenteritis;

21. Samples that should be collected in suspected cases of arsenic toxicity are HAIR and NAILS.

22. The level of arsenic in visceral organs that is indicative of arsenic poisoning is >3PPM.

23. How is arsenic poisoning differentiated from lead?
Ans: In arsenic poisoning, severe gastroenteritis alone is observed where as in lead poisoning, nervous symptoms predominate.

24. Specific antidote for arsenic poisoning is BRITISH ANTI-LEWISITE (BAL).
or DIMERCAPROL.

25. Superior water soluble derivatives of BAL are MESO-DIMERCAPRO-SUCCINIC ACID (MDSA) and DI-MERCAPTO-SUCCINIC ACID (DMSA).

26. In arsenic poisoning, why milk is considered unfit whereas meat is passed for human consumption?
Ans: Arsenic gets methylated and get rapidly excreted through urine, milk, sweat etc. Hence, milk is considered unfit. As arsenic tends to accumulate only in visceral organs and not in muscles, flesh of surviving animal is considered fit for human consumption).

2.2 Mercury

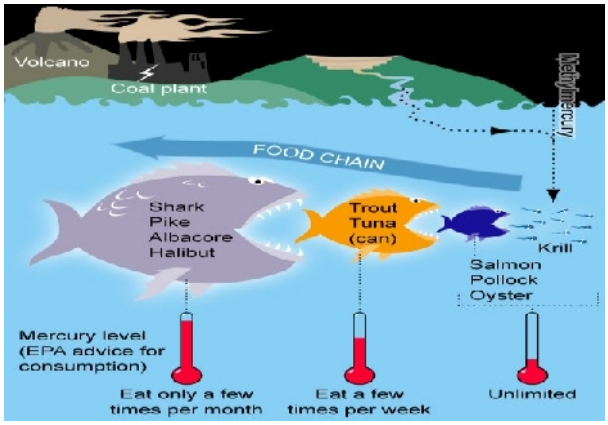
1. Give reasons as to why mercury poisoning is not common in animals?
   Ans: Hg compounds are completely replaced by better alternatives in medicinal, agricultural and industrial use. Hence, Hg poisoning is not common in animals.

2. Common source of mercury poisoning in animals is FOOD.
   (Predatory animals at the end of food chain are more likely to accumulate Hg).

3. Which of the following forms of Hg is more toxic?
   a. Elemental b. Monovalent c. Divalent d. Organic
   Ans: d. Organic form of Hg is more toxic than inorganic form due to higher lipid solubility.

4. Minamata disease in Japan was caused by the consumption of fish contaminated with METHYL MERCURY.
   (Observing affected cats, led to the identification of fish as the common source of food. Later, analysis of fish led to the detection of organic Hg as causative agent).

Cat affected by mercury poisoning



Mercury poisoning - Industrial pollution

Fig: 12 Minamata disease- organic methyl mercury poisoning through fish [14-15]

5. The species that is more sensitive to Hg poisoning is CATTLE. (Cow & Calves)

6. Why young animals are more susceptible for mercury poisoning?
   Ans: Developing nervous system is more susceptible to mercury. Hence young ones are more susceptible.

7. The process of accumulation of Hg in marine animals to a very high concentration over a period of time is known as BIO-ACCUMULATION or BIO-MAGNIFICATION.
   (Bioaccumulation can also occur in predatory animals at the end of food chain).

8. Mercury can cross the following barriers in the body
   a. Blood brain barrier (BBB) b. Placental barrier (PB) c. Both d. No barrier
   Ans: c. Both. As Hg can cross BBB causing neurological symptoms and crossing PB leads to accumulation in foetus and abortions.

9. The mechanism of toxicity of mercury involves binding with –SH, THIOL groups of proteins and enzymes.

10. Predominant symptoms of organic mercury poisoning are NEUROLOGICAL
   (Ataxia, incoordination, convulsions, abnormal behavior etc)

11. Predominant symptom in inorganic mercury poisoning is GASTROENTERITIS.

12. Predominant symptom in elemental mercury poisoning is PULMONARY SYMPTOMS.

13. Elemental mercury (Hg) is toxic only through INHALATION ROUTE of exposure.

14. The following properties can be attributed to Methyl mercury (Organic Hg)
   a. Mutagenic b. Carcinogenic c. Embryotoxic d. Teratogenic
   Ans: All of the above.

15. The sample of choice for detecting inorganic mercury poisoning is URINE.
   (Urinary concentration is a reliable indicator of inorganic Hg poisoning).

16. The sample of choice for detecting organic mercury poisoning is KIDNEY
   (Organic Hg tends to accumulate in visceral organs including brain. A concentration of 10 mg/kg in kidney is indicative of Hg poisoning)

17. Neurological and renal damage caused by mercury are IRREVERSIBLE (even with treatment).

18. The following chelating agent(s) that is (are) used for treating mercury poisoning
   a. Dimercaprol (BAL) b. D-Penicillamine c. DMSA (Succimer) d. Na-thiosulphate.
   Ans: All of the above. It should be noted that all these chelating agents are rich in –SH groups. As Hg has high affinity for –SH groups, it is easily removed by chelation.

19. Which of the following nutrient(s) can counteract toxicity of organic mercurial
   a. Vitamin A b. Vitamin D c. Vitamin E d. Selenium
   Ans: Vitamin E and Selenium. Mercury produces free radicals which are counter acted by Vit E, which is a free radical scavenger. Selenium and Vit E have interrelationship.

20. The carcass of animal affected with mercury poisoning is UNFIT for human consumption.
   (As HG accumulates in body)

2.3 Lead

1. Frequently encountered heavy metal poisoning in veterinary cases is LEAD POISONING or PLUMBISM

2. Why lead poisoning is more common in veterinary cases?
   Ans: Lead is ubiquitous in nature .Most of the animals live close to ground level and hence get more exposure. Further, habits like frequent digging of soil seen in dogs and cats increases exposure. Ultimately, increasing vehicular and industrial pollution is the major reason for lead toxicosis.

3. Animals with depraved appetite (pica) are more commonly affected with lead poisoning. Why?
   Ans: In pica, animals tends to lick walls, chew on dry peelings of paint, eat wall posters etc. Since, paints are lead based, the animals are affected with lead poisoning. Even children chewing on toys painted with cheap lead paints are also affected with lead toxicosis.

4. The lead compound that is added to petrol and gasoline as anti-knocking agent is TETRA ETHYL LEAD (TEL).
   (The tendency for fuels to auto-ignite and damage the engine is knows as knocking. Addition of TEL to petrol is banned in India from 1996).

5. The lead compound used for sweetening of wine in ancient days was LEAD ACETATE.
   (The above process along with usage of lead pipes for water supply, lead to the downfall of Roman empire due to lead toxicosis, which caused cognitive disorders and dementia).

6. The species that are most susceptible to lead poisoning are DOG, CATTLE and HORSES.
   (Dogs live close to soil are have the habit of frequent digging of soil; Cattle and horses tend to lick walls and chew on paints)

7. The species that is considered as indicator for lead in the environment is DOG.

8. The species that is very resistant to lead poisoning is SWINE.

9. The most common route of exposure to lead is ORAL.

10. Why acute lead toxicosis is not common?
    Ans: As >90% of ingested lead is eliminated from GIT without absorption and even after absorption, as only <1% of lead is in free form. Hence, acute lead toxicosis is not common.

11. The organ that is considered as sink for lead is BONE.
(About 95% of body's lead burden is found in bone. Lead toxicosis is not seen until bone is saturated).

12. Is it true that lead directly enters bone and get deposited?
Ans: NO. Initially, lead is distributed to various soft tissues and later gets re-distributed to bone from these soft tissues.

13. Lead can cross BLOOD BRAIN and PLACENTAL barriers in the body.

14. The major route of elimination of lead is BILIARY.

15. Why milk form lead affected animals is dangerous for young ones?
Ans: Considerable amount of lead is excreted in milk (about 5% of blood concentration). Since, young animals have greater capacity to absorb lead than adults, milk from lead affected animals is dangerous to young ones.

16. The element with which lead has major interaction in the body is CALCIUM.

17. Lead toxicity is a result of binding with –SH groups of proteins and enzymes.

18. Neurotoxicity in lead poisoning is a result of crossing BLOOD BRAIN barrier.

19. Central neurotransmitters that are affected by lead are GABA and DOPAMINE.

20. The system that is extremely sensitivity to lead toxicity is HAEMOPOETIC SYSTEM.

21. Lead decreases 'haeme' synthesis through the inhibition of the enzyme δ-AMINO LEVULINIC ACID SYNTHETASE (ALA-D synthase).

22. In lead poisoning, basophilic stipplings (BS) are commonly seen in this species
a. Cattle b. Sheep c. Dog d. Horse
Ans. c. Dog. BS are remnants of RNA seen in RBC which take up basophilic stain. But BS are not pathognomonic for lead.

23. The characteristic histological picture of lead poisoning in tubular cells of kidney is INTRANUCLEAR INCLUSION BODIES. (Eosinophilic)

24. Neurological symptoms accompanied by GIT symptoms possibly indicate LEAD poisoning.

25. The predominant symptoms of lead poisoning in cattle are NEUROLOGICAL SYMPTOMS.

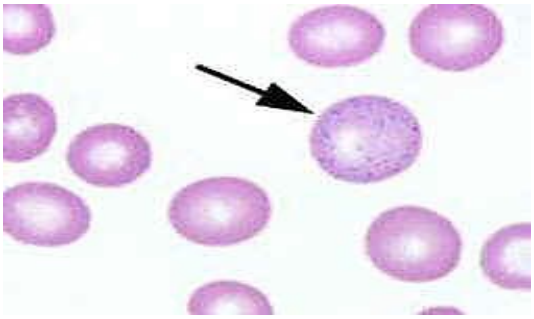


Fig:13 Basophilic Stipplings – RBC with remnants of RNA [16]

25. In dogs, the predominant symptoms of lead poisoning are GASTROINTESTINAL .

26. In horses, chronic lead poisoning manifests as paralysis of RESPIRATORY system.

27. In lead poisoning, roaring in horses is caused by RECURRENT LARYNGEAL nerve paralysis.

28. The detection of opaque lines in metaphyses of bones in an X-ray suggests LEAD poisoning.

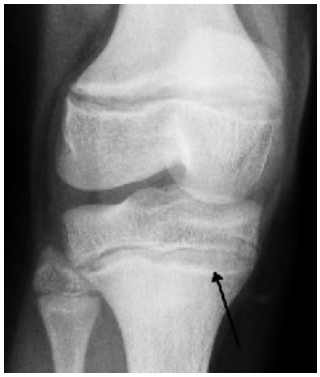


Fig:14 X-ray revealing opaque lead lines in metaphyses of bone [17]

29. In lead poisoning, estimation of ALA-D SYNTHASE enzyme in blood is of diagnostic value.

30. The content of lead in liver and kidney that is indicative of lead poisoning is > 4 PPM.

31. Why only whole blood is recommended for estimation of lead?
Ans: Majority of lead (90%) is bound to hemoglobin in RBC. Hence, plasma or serum samples are not appropriate as it is devoid of RBC.

32. Specific antidote for lead toxicosis is CALCIUM DISODIUM EDTA.

33. Meat from food animals recovered from lead poisoning is FIT for human consumption. *(However, bones should not be consumed as they store lead).*

2.4 Copper

1. Copper has inverse inter-relationship with the following element(s)
   a. Iron b. Molybdenum c. Sulphur d. Both b & c
   Ans: d. Both b & c. Molybdenum and Sulphur.

2. The species that is more susceptible of copper poisoning is SHEEP.

3. The species that is highly resistant to copper poisoning is CHICKEN.

4. The ideal ratio of copper to molybdenum in feeds should be 6:1.
   (A ratio of Cu: Mo of 10:1 can cause copper toxicosis).

5. The breed of dog that is highly susceptible to copper toxicosis due to genetic predisposition is BEDLINGTON TERRIER.
   (Autosomal recessive gene causes copper retention in liver as a result of failure of excretion).

Fig:15 Bedlington Terrier –Genetic predisposition for copper accumulation [18]

6. Deficiency of MOLYBDENUM micro mineral predisposes to copper toxicity.

7. The specific transport proteins for copper in the body are TRANSCUPERIN and CERULOPLASMIN.
   (Transcuperin and albumin transports Cu from blood to liver. However, Transcuperin is specific but is less abundant. Ceruloplasmin transports from liver to peripheral tissues. About 90% of Cu in circulation is in bound form with ceruloplasmin)

8. The primary organ for accumulation (storage) of copper is LIVER.

9. The major route of elimination for copper from body is BILIARY.

10. Molybdenum and Sulphur reduce toxicity of copper by enhancing EXCRETION.

11. The type of anemia seen in copper toxicosis is HEMOLYTIC.
(Hemolysis of RBC releases hemoglobin causing hemoglobinuria)

12. The characteristic appearance of kidneys in copper poisoning is GUNMETAL.
(Hemoglobin released during hemolysis clogs the renal tubules and leads to darkening and necrosis of kidneys. Hence, gunmetal-like kidneys are seen).

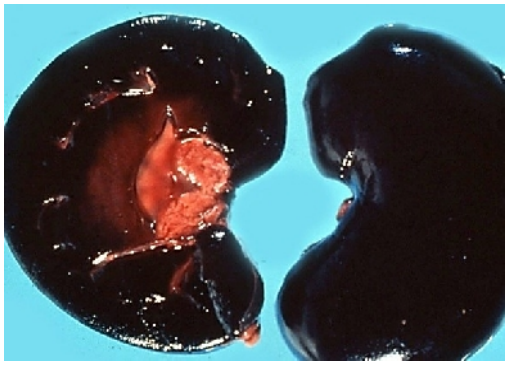


Fig:16 Gunmetal Kidneys – Copper Poisoning [19]

13. Major symptoms of copper toxicosis are HEPATOTOXICITY and HEMOLYTIC ANAEMIA.
(Hepatotoxicity precedes the development of hemolytic anemia. Once liver is damaged, Cu is released into blood causing rapid hemolysis).

14. In copper poisoning, elevation of liver marker enzymes (AST, SDH, and LDH) occurs prior to HEMOLYTIC CRISIS (about 3-6 weeks before).
(Though marker enzymes are elevated, the level of Cu in blood is not increased until a day or two before development of hemolysis).

15. In copper poisoning, after the development of hemolytic crisis, the prognosis is GRAVE.

16. Chelating agent used to treat copper poisoning is D-PENICILLAMINE.

17. Why molybdate salts are given as supportive therapy in copper poisoning?
Ans: Molybdenum has inverse relationship with copper, increasing its elimination. Hence, molybdate salts are used in copper poisoning.

18. How zinc administration prevents copper toxicity?
Ans: Zinc induces the synthesis of mucosal metallothionins in GIT, which bind to copper and prevent Cu absorption. Hence, zinc supplementation decreases the development of copper toxicity.

2.5 Molybdenum

1. The primary source of molybdenum poisoning in animals is GRAZING
   (Molybdenum poisoning is referred to as Molybdenosis or Teart. The term 'teart' refers to watery foul smelling diarrhea which is characteristic in molybdenosis).

2. The species that is more susceptible to molybdenum poisoning is CATTLE.

3. Molybdenum toxicosis occurs primarily in the deficiency of COPPER.

4. The ideal ratio of copper to molybdenum should be 6:1.
   (Ratio of <2:1 Cu to Mo can cause molybdenum toxicosis)

5. The level of molybdenum that causes toxicity, irrespective of copper content is >10 PPM.

6. Molybdenum is primarily excreted through URINE.
   (Biliary excretion accounts for about 20% of the excretion)

7. In molybdenum poisoning, deficiency of COPPER (mineral) is observed.
   (Hence, most of the symptoms in Mo poisoning resemble copper deficiency).

8. Peat scours or teart or shooting diarrhea is a characteristic symptom of MOLYBDENOSIS.
   (Peat scour or teart refers to foul smelling watery faeces with gas bubbles. Molybdenum complexes with catechols and inactivates them. Hence, the natural bacteriostatic activity of GIT is lost, which lead to infection and diarrhea).

9. Light coloured hair and depigmentation around eyes in molybdenum poisoning causes SPECTACLE-EYE appearance.
   (Due to Cu deficiency, melanin production is reduced due to decreased in activity of Cu containing enzyme, tyrosinase, which converts tyrosine to melanin. Spectacle-eye is prominently visible in buffaloes due to dark colouration of the animal).

Fig: 17 Spectacle-eye appearance – Molybdenum poisoning [20]

10. Molybdenosis in sheep is manifested as ENZOOTIC ATAXIA or SWAY BACK.
(Mo causes Cu deficiency. Cu dependent enzymes like cytochrome oxidase are necessary for the synthesis of phospholipids of myelin. Hence, in Cu deficiency, defective nerves are formed causing sway back).

11. The treatment of molybdenum poisoning involves administration of COPPER SALTS (Eg. Copper sulphate).

CHAPTER III

TOXICOLOGY OF NON-METALS

3.1 Fluoride

1. Why fluorine is not available in free form?
   Ans: Fluorine is the most reactive non-metal (due to high electronegativity) and hence is not available in free form. It is seen in combination with other elements as fluorides.

2. The chronic disease resulting from continuous ingestion of small amounts of fluoride is FLUOROSIS.
   (Fluorosis is endemic in at least 22 countries world-wide and in many areas of Andhra Pradesh and Telangana states).

Fig: 18 Worldwide distribution of endemic fluorosis – 22 counties are affected [21]

3. The part of the plant that does not accumulates fluoride is
   a. Seed b. Stem c. Leaf d. Flower
   Ans: Seed or grains.

4. Supplementation with ROCK PHOSPHATE mineral supplements can cause fluorosis.
   (The optimum ratio of fluoride to phosphorus in rock phosphates should be 1:100).

5. Fatal toxicosis that can occur from inhaling gases and dust from volcanic eruptions is FLUORINE INTOXICATION.

6. Acute fluoride poisoning is common in DOG, whereas, chronic poisoning is common in HERBIVORES.
   (Dogs are poisoned from fluoride containing pesticides whereas herbivores from eating contaminated pastures)

7. Maximum tolerable level of fluoride in forage for herbivorous animals is 40-50 PPM.
   (A level of 50 ppm should not be exceeded in the ration of animals)

8. The level of fluoride in drinking water that can cause fluorosis in animals is >2 PPM.
   (Dental defects are see at 5ppm, wear and tear at 10 ppm and systematic effects at 30 ppm).

9. Fluoride accumulates in BONE and TEETH in the body.
   (Bone acts as sink for fluoride similar to lead. However, accumulation of fluoride in teeth occurs only during formative stages i.e., young age only).

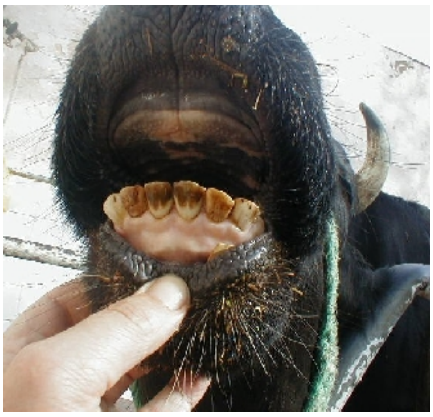
10. Fluoride is gradually excreted from the body through URINE.

11. Fluoride interferes with the following element(s) in the body
    a. Calcium b. Magnesium c. Manganese d. Phosphorus
    Ans: a, b & c: Ca, Mg and Mn. Interaction with calcium leads to hypocalcaemia and interference with magnesium causes hypomagnesaemia causing seizures.

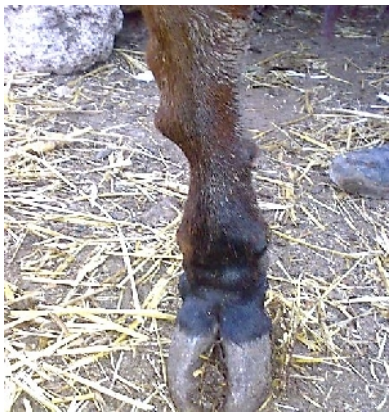
12. The corrosive effects of fluoride in GIT are due to the formation of HYDROFLUORIC ACID in the acidic medium of stomach.

13. Hyperkalemia in fluoride poisoning is a result of inhibition of Na^+-K^+ ATPase enzyme.

14. Brown or black discolouration of teeth in fluorosis is a result of oxidation of ENAMEL.
    (Fluoride is required for enamel formation but in it causes enamel damage due to oxidation).



Dental fluorosis - Mottling



Skeletal fluorosis - Periosteosis

Fig:19 Fluorosis in animals – Skeletal & Dental forms [22]

15. In fluorosis, defects in bones are a result of replacement of HYDROXYL groups with fluoride in hydroxyapatite structure.

16. Chronic fluorosis is manifested as SKELETAL and DENTAL forms.

17. The form of fluorosis observed in young animals exposed to fluoride during early stages of life is
    a. Skeletal b. Dental c. Both d. Not affected
    Ans: c. Both. If the animal is exposed in late stages of life, only skeletal form is observed but if exposure takes place in early stages of life, both forms are seen).

18. The samples of choice to be collected in suspected cases of fluoride poisoning are BONE and URINE.
    (Affected cattle have 3000 ppm and sheep have 5000 ppm of fluoride in bone against a normal value of 200 – 600 ppm. In urine >15 ppm is suggestive of fluorosis).

19. In fluorosis, the density of bones INCREASES.
    (Fluoride binds with Ca by replacing hydroxyl groups in bones causing an increased mineralization and bone density).

20. Soft tissue that accumulates highest amount of fluoride is PINEAL GLAND.

21. Death in fluoride toxicity is due to the development of HYPERKALEMIA and HYPOCALCAEMIA.

22. The salts of CALCIUM are employed in treatment of fluorosis.
    (Ca salts are mainly used as supportive therapy. However, there is no specific antidote for fluoride toxicity).

3.2 Phosphorus

1. Which of the following form(s) of phosphorus is(are) toxic?
   a. White b. Red c. Yellow d. Black
   Ans: a & c. White and yellow phosphorus are soluble and readily absorbed. Hence cause toxicity. Whereas red phosphorus is insoluble, hence is non-toxic.

2. Excess feeding of wheat bran rich in phosphorus causes BRAN DISEASE in horses.

3. Name the non-metal poisoning that can occur during Diwali and in war-zones?
   Ans: Phosphorus. Yellow phosphorus is used in the manufacture of fire crackers and military ammunition.

4. Garlic-like odour of breath and luminous stomach contents suggest PHOSPHORUS poisoning.

5. The routes of elimination for phosphorus are RENAL and PULMONARY.
(Hence the breath has garlic like odour).

6. Dermal exposure to white or yellow phosphorus leads to SKIN BURNS.

7. Why burns due to phosphorus causes higher mortality than other agents?
Ans: Absorption of phosphorus through raw burnt surface leads to multi-organ failure. Hence, burns due to phosphorus are more dangerous than other burns.

8. The immediate symptom upon oral ingestion of phosphorus is EMESIS (Hematemesis)
(Phosphorus is a strong irritant with corrosive properties. Hence it causes GIT irritation leading to vomition).

9. Major organs that are damaged in phosphorus poisoning are LIVER and KIDNEY.

10. Necrosis of jaw that is observed in chronic phosphorus poisoning is called as PHOSSY JAW.

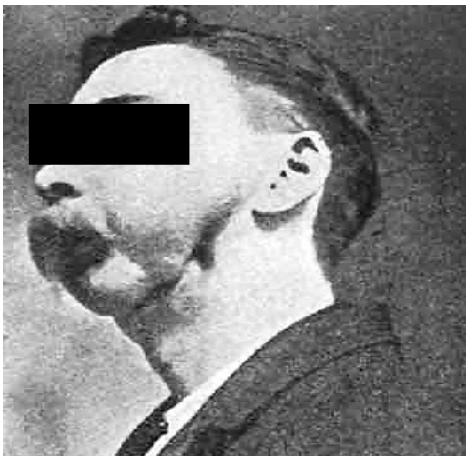



Fig:20 Phossy Jaw – Phosphorus poisoning – Degeneration of jaw bone [23-24]

11. Ideal sample material for the diagnosis of phosphorus poisoning is VOMITUS or STOMACH CONTENTS.

12. Why oily purgatives such as mineral oils are contraindicated in phosphorus poisoning?
Ans: Oils increase the absorption of phosphorus. Hence are contraindicated in phosphorus poisoning.

13. The prognosis in case of phosphorus poisoning is GUARDED to GRAVE.

3.3 Nitrate and Nitrite

1. Toxicity of nitrate is due to its conversion into NITRITE by rumen micro flora.
   (Nitrites [NO_2^-] are 10 times more toxic than nitrates [NO_3^-]).

2. The use of NITRATE fertilizers increases the concentration of nitrates in plants.

3. The herbicide that increases nitrates in plants is 2, 4-D.

4. The following plant(s) is/ are nitrate accumulators
   a. Cereal grasses b. Maize c. Sunflower d. Sorghum
   Ans: All.

5. The source of drinking water that is high in nitrates is DEEP-WELL.
   (Deep-well water contains around 1700 to 3000 ppm of nitrates due to seepage from surface soil).

6. The following method of storing forages reduces nitrate content
   a. Hay making b. Silage making c. Composting d. Straw making
   Ans: b. Silage making. The process of fermentation reduces nitrate content in forages.

7. The species that are more susceptible to nitrate poisoning are RUMINANTS.

8. Why cattle are more susceptible than sheep for nitrate poisoning?
   Ans: The rumen of sheep is effective in converting nitrites to ammonia, which is used for protein synthesis. But in cattle, the rumen is not as effective as sheep. Hence, in cattle are more susceptible.

9. Why non-ruminants are not affected by nitrates from plant sources?
   Ans: The conversion of nitrates to nitrites does not occur in non-ruminants (lack of rumen micro flora). Hence, non-ruminants are not affected. However, pigs are most sensitive to ingestion of pre-formed nitrites.

10. Why plants accumulate nitrates in toxic proportions?
    Ans: Plants absorbs nitrates from soil as a part of its physiology. However, any change in environmental conditions that affects the rate of utilization of nitrates leads to nitrate accumulation in toxic proportions. Eg: Lack of rainfall – no leaching of nitrates from soil; Low temperature – inhibits nitrate reductase activity; High temperature – excessive absorption from soil; etc.

11. The concentration of nitrates in plants that is toxic to animals is 1% or 10,000 PPM.
    (on dry matter basis, a concentration of 0.5% nitrates is toxic in plant material).

12. Drinking water containing more than 1500 PPM of nitrates can cause poisoning.

13. Maximum accumulation of nitrates is seen in the following part of the plant
    a. Tip b. Leaves c. Upper 1/3 of stem d. Lower 1/3 of stem
    Ans: d. Lower 1/3 of the stem. Parts closer to soil accumulate more nitrates.

14. The nitrate content in young plants is MORE than mature plants.

15. The presence of COLIFORM bacteria in water increases nitrate toxicity.

16. The growth of plants or algal blooms (eutrophication) in ponds leads to DECREASE in nitrate content of water.
    (Excessive growth of plants or algae in water bodies is due to eutrophication i.e., addition of nutrients (fertilizers) from run-off land water)

Fig: 21 Algal Bloom in a pond – Plants and algal blooms reduces nitrates in water [25]

17. The deficiency of molybdenum or sulphur or phosphorus in soil causes INCREASED nitrate accumulation in plants.

18. The deficiency of copper or cobalt or manganese in soil causes DECREASED nitrate accumulation in plants.

19. Addition of SOLUBLE CARBOHYDRATES (TDN) to feed improves tolerance to nitrate toxicity.
    (Carbohydrates are necessary for rumen micro flora for the conversion of nitrites into ammonia, which is utilized for protein synthesis)

20. The antibiotic used as feed additive that enhances conversion of nitrates to nitrites causing poisoning is MONENSIN.

21. Watering of animals immediately after consuming nitrate rich plants DECREASES the chances of nitrate poisoning.
    (As nitrates and nitrites are eliminated through urine. On the contrary, watering after consumption of cyanogenic plants increases cyanide toxicity).

22. Nitrite ion enters erythrocytes in exchange for CHLORIDE ion.

23. Nitrite combines with hemoglobin (in1:2 ratio) to form METHEMOGLOBIN.
(Ferrous (Fe^{2+}) is oxidized to ferric (Fe^{3+}) in met-hemoglobin, which decreases oxygen carrying capacity of blood).

24. The colour of blood in nitrate poisoning is CHOCOLATE BROWN.
(Due to methemoglobin formation).

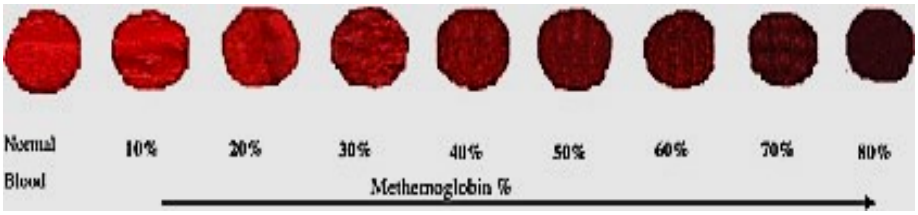


Fig:22 Methemoglobin – Brown coloured blood [26]

25. Physiologically formed methemoglobin (1 to 2 %) is converted back to hemoglobin by the enzymes DIAPHORASE – I & II.
(Diaphorase – I is NAD dependent whereas DIAPHORASE – II is NADP dependent).

26. Formation of 20 to 40% of methemoglobin produces symptoms of nitrate poisoning.

27. Vasodilation and hypotension observed in nitrate poisoning is due to smooth muscle relaxation caused by NITRIC OXIDE (NO).

28. The respiratiory rate in nitrate poisoning is INCREASED (RAPID).
(Rapid respiration is a prominent clinical sign in nitrate poisoning due to hypoxia).

29. Chronic nitrate poisoning leads to the development of GOITER in sheep.
(Nitrate interferes with iodine metabolism).

30. The preferred ante-mortem sample to be collected in nitrate poisoning is PLASMA.
(Serum cannot be used as nitrate are retained in the blood clot).

31. Specific treatment for nitrate poisoning is METHYLENE BLUE.
(Reducing agents such as ascorbic acid are also used).

32. Methylene blue is oxidized to LEUCOMETHYLENE BLUE during the conversion of met-hemoglobin to hemoglobin.

33. Methylene blue mediated conversion of methemoglobin to hemoglobin is dependent on the availability of $NADPH_2$.

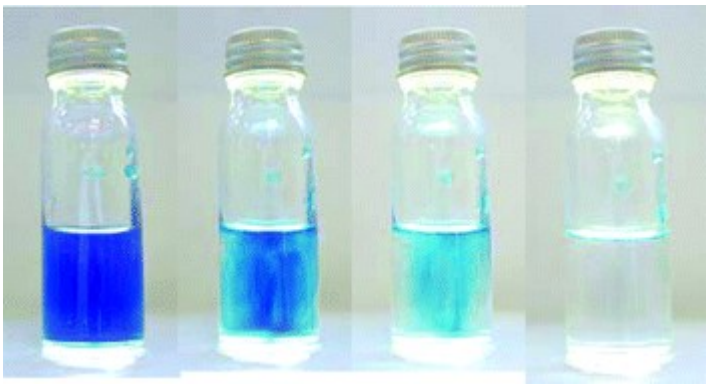


Fig:23 Methylene blue conversion to Leucomethylene blue [27]

34. The use of ADRENERGIC AGONIST agents to improve cardio-vascular function is contraindicated in nitrate toxicity.
(Adrenergic agonists increase oxygen demand but in nitrate poisoning, the oxygen carrying capacity of blood is already hampered. Hence, their use is contraindicated).

3.4 Cyanide

1. Cyanide from plant sources is present in the form of CYANOGENIC GLYCODSIDES.
(Cyanogenic glycosides are present in epidermal cells)

2. The accumulation of NITRATES in plants predisposes to cyanide formation.
(Cyanide is formed by the reaction of nitrates with amino acids).

3. Plant enzyme responsible for the release of hydrocyanic acid from cyanogenic glycosides is β-GLYCOSIDASE.
(β-glycosidase is present in mesochymal cells).

4. Why chopping, cutting or chewing of plants increases cyanide toxicity?
Ans: Cyanogenic glycosides are present in epidermal cells whereas the enzyme β-glycosidase is present in mesenchymal cells. During chopping, cutting or chewing the mesenchymal cells are ruptured releasing the enzyme, which acts on glycosides releasing HCN)

5. The species that are susceptible to cyanogenic glycoside poisoning are RUMINANTS.

6. Why ruminants are more susceptible to cyanogenic glycoside poisoning?
Ans: The pH, water content and micro flora of rumen facilitates the release of cyanide from cyanogenic glycosides. Hence, ruminants are more susceptible to cyanogenic glycoside poisoning.

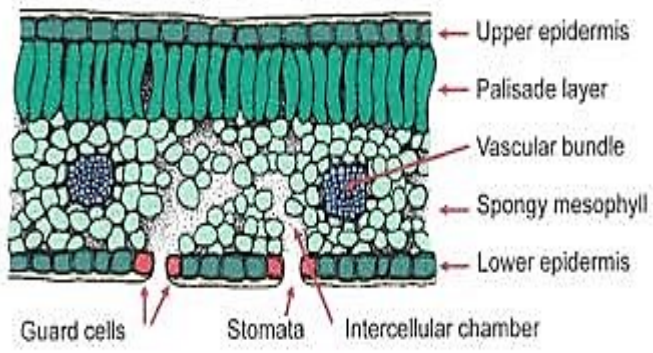


Fig: 24 Epidermal cells (contains cyanogenic glycosides) and Mesenchymal cells (contains β-glycosidase enzyme) in a leaf.[28]

7. Among ruminants, the more susceptible species for cyanogenic glycosides is CATTLE. *(Due to large rumen, which has more micro flora releasing more HCN)*

8. Why non-ruminants are not affected by cyanogenic glycosides?
   Ans: Acidic pH destroys β-glycosidase enzyme, which is responsible for the release of HCN. Hence, non-ruminants are not affected by cyanogenic glycosides.

9. The cyanogenic glycoside in bitter almond and wild cherry is AMYGDALINE.

10. The cyanogenic glycoside in sorghum and sudan grass is DHURRIN.

11. The cyanogenic glycoside in linseed and wild clover is LINAMARINE.

12. The part of the plant that is rich in cyanogenic glycosides is LEAF.

13. The level of HCN in plants, which can cause cyanide poisoning in animals is 200 PPM AND ABOVE.

14. The quantity of cyanogenic glycosides in young plants is MORE than mature plants.

15. After a period of drought or cloudy weather, the cyanide content in the plant
    a. Increases b. Decreases c. Does not change d. Becomes zero
    Ans: a. Increases.

16. The use of NITRATE fertilizers increases cyanide toxicity.
    (Nitrate fertilizers increases nitrate content in plants, which is subsequently converted to cyanide by combining with amino acids).

17. Spraying of the weedicide 2, 4-D can increase cyanide content in plants.
    (2,4-D increases nitrate content and consequently the cyanide content).

18. Soils that favour cyanide accumulation in plants are rich in NITROGEN and deficient in PHOSPHORUS content.

19. Watering animals after feeding on cyanogenic plants INCREASES toxicity.
(Water causes hydrolysis of cyanogenic glycosides releasing HCN).

20. The metabolite of cyanide, which is excreted through urine is THIOCYANATE.

21. Cyanide is converted to non-toxic thio-cyanate by the enzyme RHODANESE.

22. Good reserves of SULPHUR in the body reduces the toxic effects of cyanide.

23. Cyanide inhibits cellular respiration by binding with CYTOCHROME OXIDASE (cyt_{a3}).
(Cyanide has more affinity towards metallo-porphyrin (Fe) containing enzymes)

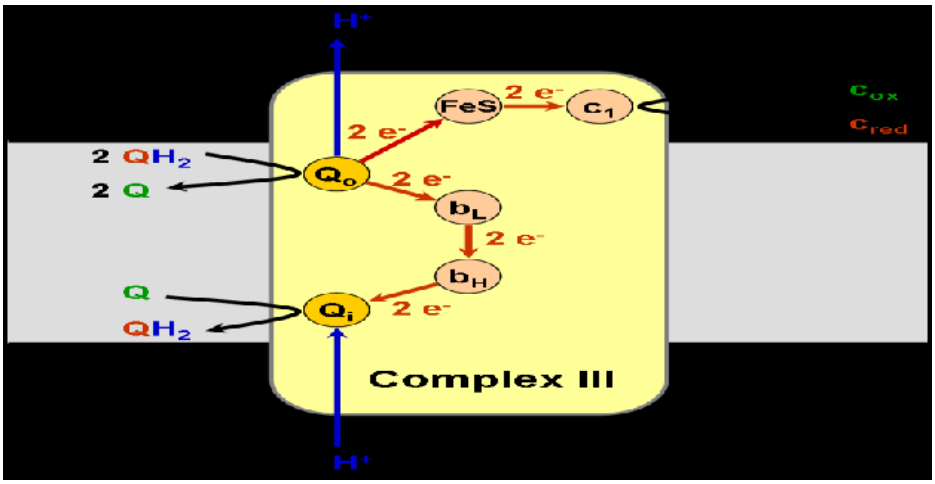


Fig: 25 Electron transport chain – Cytochrome Oxidase inhibition by cyanide [29]

24. Why cyanide has more affinity for cytochrome oxidase than hemoglobin?
Ans: Cyanide has more affinity for ferric (Fe^{3+}) form of iron. In hemoglobin, iron is present in ferrous (Fe^{2+}) form whereas cytochrome oxidase has ferric (Fe^{3+}) iron. Hence, cyanide prefers cytochrome oxidase.

25. Cyanide has more affinity for the following
a. Hemoglobin b. Cytochrome oxidase c. Met-hemoglobin d. Myoglobin
Ans: c. Met-hemoglobin (Fe^{3+}). *Sodium nitrate is used as a therapeutic strategy to convert hemoglobin to methemoglobin, which removes cyanide from cytochrome oxidase.*

26. The colour of blood in cyanide poisoning is BRIGHT RED.
(Due to non-utilization by tissues, oxygen stays in blood giving bright colour).

27. The characteristic smell of rumen contents that is suggestive of cyanide poisoning is BITTER ALMOND.
(The smell of cyanide is similar to bitter almonds. Consuming of about 18 bitter almonds can kill a human being. However, the variety used in household purpose is the non-toxic domesticated sweet version).

28. Chronic form of cyanide toxicity observed in humans due to consumption of cassava root is called as KONZO.

Fig:26 Cassava Root – causes chronic cyanide poisoning [30]

29. The level of cyanide in rumen contents that is indicative of cyanide poisoning is 10 PPM.

30. Specific treatment for cyanide toxicity is SODIUM NITRATE followed by SODIUM THIOSULPHATE.
($NaNO_3$ converts hemoglobin into met-hemoglobin, which dissociates cyanide from cytochrome oxidase and brings into blood; Na_2SO_3 helps in the conversion of cyanide to non-toxic thio-cyanate, which is easily excreted through urine).

3.5 Selenium

1. The Italian traveler who first reported the association between sloughing of hooves in horses with consumption of specific plants is MARCO POLO.

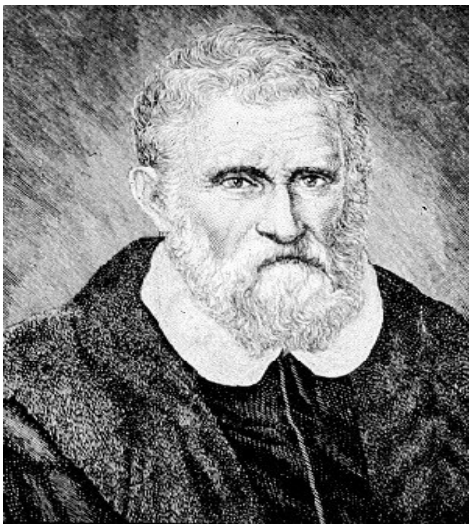


Fig:27 Morco Polo – Italian merchant traveler –Reported selenium accumulator plants [31]

2. The following form(s) of selenium is (are) soluble
   a. Elemental (0) b. Selenide (+2) c. Selenite (+2) d. Selenate (+6) e. Organo selenium
   Ans: c, d & e. Selenite (+2) and Selenate (+6) are soluble and hence cause toxicity in animals.

3. The most toxic from of selenium is ORGANO SELENIUM.
   (The magnitude of toxicity of selenium is Organo selenium > Selenite=Selenate > Selenide > Elemental Selenium).

4. Organo selenium is formed due to the replacement of SULPHUR by selenium in amino acids.
   (Sulphur containing amino acids being cysteine and methionine)

5. The level of selenium in plants that is toxic to animals is 5 PPM AND ABOVE.

6. The type of plants that have a physiological requirement for selenium and accumulate high levels of selenium are called OBLIGATE (PRIMARY) ACCUMULATORS or INDICATOR PLANTS.
   (Eg: Astragalus sps, Stanleya sps, Oonopsis sps etc. Animals avoid these plants as they are not palatable, but in scarcity they are consumed leading to toxicity)

7. The level of selenium found in obligate accumulators is 100-1500 PPM.

Astragalus sps (Milk vetches)

Stanleya sps (Princes plumes)

Oonopsis sps (Golden weeds)

Xylorrhiza sps (Woody aster)

Fig:28 Obligate selenium accumulators [32-35]

8. The type of plants that dont require selenium but accumulate high levels of selenium, if present in soil, are called FACULTATIVE ACCUMULATORS.
   (Eg: Acacia sps, Aster sps, Astriplex sps, Artemisia sps etc. As these plants are more palatable, toxicity is often seen in animals.).

9. The level of selenium found in facultative accumulators is 25-100 PPM.

Artimesia sps (Sages)

Aster sps (Asters)

Astriplex sps (Salt brush) *Acacia sps (Acacia)*

Fig:29 Facultative selenium accumulators [36-39]

10. The type of plants which do not require selenium but accumulate low levels of selenium, if it is present in the soil, are called NON- ACCUMULATORS.
(Eg: Maize, Wheat, Barley etc. These plants are more palatable than other accumulators and hence the chances of toxicity are more).

11. The level of selenium found in non-accumulators is 1-25 PPM.

12. Important natural source of selenium, apart from plants is VOLCANIC GASES.

13. The type of climates that favour selenium accumulation is ARID AND SEMI-ARID.

14. The pH of the soil that favours selenium accumulation is ALKALINE (>7.0).

15. Why selenium toxicity is more commonly seen in arid and semi-arid climatic zones?
Ans: In arid and semi-arid climatic zones, due to less rainfall, selenium accumulates in top layers of the soil – as it is not leached. Hence, selenium accumulation in plants is common

16. The metabolite of selenium that is excreted through urine is TRIMETHYL-SELENONIUM.

17. Cytotoxicity caused by selenium is a result of FREE RADICAL generation at cellular level.

18. The non-enzymatic antioxidant that is depleted by selenium is GLUTATHIONE (GSH).

19. In selenium poisoning, the characteristic odour is GARLIC-LIKE.
(Recollect, phosphorus also produces garlic-like odour)

20. The vitamin that aggrevates selenium poisoning is VITAMIN E.
(Se and Vit E have inter-relationship)

21. Sub-acute toxicity of selenium is also known as BLIND STAGGERS.

22. Chronic selenium toxicity is also called as ALKALI DISEASE.

23. In cattle, cracked and overgrown hooves are suggestive of SELENIUM poisoning.

24. In horses, loss of hair from the mane is the primary symptom of SELENIUM poisoning.

25. Why cracked and overgrown hooves are seen in selenium poisoning?
Ans: Selenium replaces sulphur in sulphur-containing amino acids such as cysteine and methionine leading to structural abnormalities in proteins. Hence, overgrown and cracked hooves are seen.

Fig:30 Cracked Hooves – Selenium Poisoning [40-41]

26. In selenium toxicity, depleted glutathione levels can be restored by administering ACETYL CYSTEINE during treatment.

27. In selenium poisoning, the level of selenium detected in blood is 1-4 PPM and in hooves is 5-20 PPM.

28. The use of DIMERCAPROL (BAL) metal chelator is contraindicated in selenium toxicity.

3.6 Oxalate

1. The common source of oxalate poisoning in animals is PLANTS.

2. Important oxalate containing plants responsible for oxalate poisoning in animals are HALOGETON GLOMERATUS and OXALIS PESCAPRAE.

3. The oxalate salt of the following element(s) is (are) soluble
a. Sodium b. Potassium c. Magnesium d. Calcium
Ans: a & b. Sodium and Potassium.

H.glomeratus

O.pescaprae

Fig:31 Oxalate accumulators [42-43]

4. Why the oxalates in *Halogeton glomeratus* and *Oxalis pescaprae* are the common cause of oxalate poisoning?
   Ans: The oxalates present in the above species are soluble. Hence, these plants cause oxalate poisoning. In H.glomeratus, both sodium and potassium oxalates are present whereas in O.pescapre, only potassium oxalates are present.

5. Halogeton species contains 34% of oxalates on dry matter basis.

6. The species of fungus that is rich in oxalates and can cause oxalate poisoning, is ASPERGILLUS.

7. Why ruminants are less susceptible for oxalate poisoning?
   Ans: Rumen has the ability to convert soluble oxalates into insoluble form. Hence, ruminants are less susceptible for oxalate poisoning. But if the rumen's ability for conversion is exceeded, oxalate poisoning is observed.

8. The species that is commonly affected by oxalate poisoning is SHEEP.

9. Pastures containing 2% of oxalates is toxic for sheep.

10. The important mineral with which oxalates interact in the body is CALCIUM.

11. The primary clinical sign in oxalate poisoning is HYPOCALCAEMIA.

12. The deposition of insoluble calcium oxalate crystals in renal tubules causes OXALATE NEPHROSIS.

13. The common site for urinary obstruction in bulls and rams due to oxalate stones is SIGMOID FLEXURE.
    (In rams, the oxalate crystals also deposit in urethral process).

Fig:32 Oxalate Nephrosis – Deposition of calcium oxalates in kidney [44]

14. Detection of oxalate crystals in KIDNEY and RUMEN EPITHELIUM is indicative of oxalate poisoning.

15. In oxalate poisoning, CALCIUM salts are used as a part of the treatment.
(Calcium salts are used to correct hypocalcaemia observed in oxalate poisoning)

CHAPTER IV

TOXICOLOGY OF PLANTS

4.1 Photosensitization

1. Abnormal sensitivity of un-pigmented or less pigmented areas of skin to sun light due to the presence of photodynamic substances in peripheral circulation is known as PHOTOSENSITIZATION.

2. Substances that absorb UV light and emit energy while coming to ground state are called PHOTODYNAMIC SUBSTANCES.

3. The type of photosensitivity resulting from direct ingestion of photodynamic substances or metabolically activated agents is called PRIMARY photosensitization.
   (Eg: Plants: Hypericin- Hypericium species; Fagopyrin – Fagopyrum species; Parthenium sps; Drugs -phenothiazines, tetracyclines, sulphonamides, acridine dyes etc)

Hypericium sps (St. John Wort)

Fagopyrum sps

Fig:33 Direct photosensitization causing plants [45-46]

4. The type of photosensitivity resulting from hepatic damage consequent to ingestion of hepatotoxic plants or substances is called SECONDARY / HEPATOGENOUS photosensitization.
   (Eg. Pyrrolizidine alkaloid containing plants- Senicio sps, Heliotropium sps,; Lantana camara; Mycotoxins- Sporodesmins; Blue green algae – Microcystis sps)

5. The photodynamic substance formed due to bacterial break down of chlorophyll that is responsible for secondary photosensitization is PHYLLOERYTHRIN.

6. The species that is more susceptible to secondary photosensitization from pyrrolizidine alkaloids is PIG.

7. *Lantana camara* causes SECONDARY type of photosensitization.

Senicio sps *Lantana camara*

Fig:34 Secondary / Hepatogenous photosensitization causing plants [47-48]

8. Secondary photosensitization in *Lantana camara* is a result of BILE DUCT OCCLUSION.

9. The phyto-constituents of *L.camara* that are responsible for bile duct occlusion and liver damage are LANTADENE A & B.

10. Why the lesionsin photosensitization are localized?
Ans: Melanin pigment protects skin from UV light. In less pigmented areas or in areas devoid of fur/wool, more UV light is absorbed leading to sun burns. Hence, less pigmented areas and areas devoid of hair/wool like face, eyelids, muzzle, coronary band, udder etc are more prone for photosensitization.

11. Hepatic lesions such as FIBROSIS and BILIARY HYPERPLASIA are useful for differential diagnosisis of primary and secondary photosensitization.

12. Visible lesions in photosensitization are SUN BURNS.

Fig:35 Photosensitization – Sun burns [49-50]

13. The prognosis is poor in SECONDARY type of photosensitization.
(The hepatic damage is generally irreversible leading to death).

4.2 Thiamine deficiency causing plants

1. Bracken fern poisoning is a result of consuming the plant PTERIDIUM AQUILINUM.

2. The enzyme present in bracken fern that breaks down vitamin B_1 is THIAMINASE.
   (Other thiaminase containing plants include Horse tail, Australian nardoo fern, rock fern)

Pteridium aquilinum (Bracken fern)

Equisetum arvense (Horse tail)

Marsilea drummondii (Austr. Nardoo)

Cheilanthes sieberi (Rock fern)

Fig:36 Thiaminase containing plants [51-54]

3. Aplasia of bone marrow due to bracken fern poisoning is observed in RUMINANTS species.

4. The following part of bracken fern is more toxic
   a. Rhizome b. Stem c. Leaves d. Tips
   Ans: a. Rhizome. All parts of bracken fern are toxic, however, rhizome is more toxic.

5. Carcinogenic and aplastic anemia inducing factor present in bracken fern is PTAQUILOSIDE.

6. The active carcinogenic formed from ptaquiloside in alkaline pH is DIENON.

7. The co-carcinogen in bracken fern, which causes malignant tumours in mouth, esophagus and rumen along with papilloma virus is QUERCETIN.

8. The most susceptible species for bracken fern poisoning are HORSE and CATTLE.

9. The species in which anemia due to bracken fern poisoning is absent is HORSE.
   (The main symptoms in horses are neurological)

10. The main symptom of bracken fern poisoning in cattle is APLASTIC ANEMIA.

11. Long term consumption of bracken fern in cattle causes tumours of URINARY BLADDER.

12. Why bracken fern causes urinary bladder tumours in cattle?
    Ans: Ptaquiloside is converted into an active carcinogen dienon in alkaline medium. As the urine in cattle is alkaline, the conversion of ptaquiloside to dienon is increased, leading to urinary bladder tumours.

13. In bracken fern poisoning, enzootic hematuria is seen in CATTLE species.

14. Bracken fern produces permanent blindness in SHEEP species.

15. Diagnosis of bracken fern poisoning involves the estimating THIAMINE vitamin in blood.
    (Normal thiamine (B_1) content in blood of cattle is 8.5 µg/dL which is reduced to 2.5 µg/dL).

16. Specific treatment for bracken fern induced thiamine deficiency is THIAMINE (vit B_1).
    (However, there is no specific treatment for bone marrow aplasia and tumours caused by bracken fern).

4.3 *Abrus precatorius* (Abrus) and *Ricinus communis* (Castor)

1. *Abrus precatorius* is commonly known as ROSARY PEA or RATHI.
2. The toxicity due to seeds of *Abrus precatorius* is commonly referred to as SUI/ NEEDLE poisoning.
   (Needles prepared from abrus seeds are inserted subcutaneously causing malicious poisoning. Hence, the name sui or needle poisoning).

3. Chemically, abrin is a
   a. Toxalbumin b. Toxglobulin c. Polypeptide d. Carbohydrate
   Ans: a.Toxalbumin.

4. The toxic principle in the seeds of *Abrus precatorius* is ABRIN.

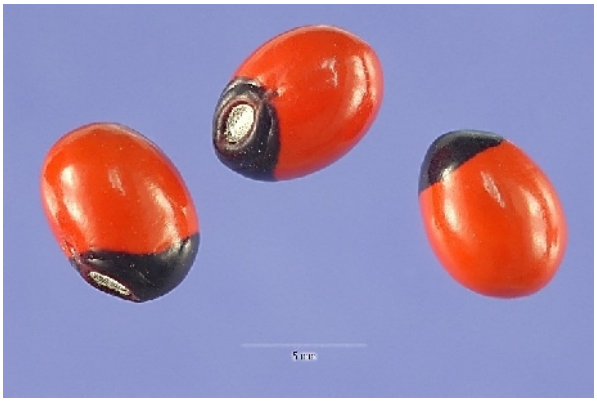



Fig:37 Abrus precatorius – seeds contain abrin [55]

4. *Ricinus communis* is commonly referred to as CASTOR BEAN.


Fig:38 Ricinus communis – seeds contain ricin [56-57]

5. The toxic principle present in the seeds of *Ricinus communis* is RICIN.

6. Abrin and ricin belongs to the class of glycoproteins known as LECTINS.

7. Why swallowing of seeds of abrus and castor is not toxic?
   Ans: Abrus and castor seeds have tough outer coating which resists digestion and hence are passed through GIT without causing toxicity. However, crushing or chewing prior to swallowing will produce toxicity.

8. During extraction of oil from castor seeds, the toxic principle ricin is detected only in
   a. Oil b. Seed Cake c. Both d. None
   Ans: Seed cake. Ricin is not expressed in oil but is retained in seed cake.

9. The toxic principle present in castor bean that causes hemagglutination and hemolysis is RCA (Ricinus communis agglutinin).
   (However, RCA is not absorbed through GIT; it needs parenteral administration)

10. The species that is more susceptible to abrin and ricin poisoning is HORSE.

11. At the cellular level, lectins (abrin & ricin) decreases protein synthesis by inhibiting RIBOSOMES.
(one molecule of abrin inhibits up to 1500 ribosomes per second)

12. Why barley containing lectins is non-toxic where as abrin and castor are?
Ans: Lectins contains A and B chains linked by disulphide bond. The presence of both chains is necessary for producing toxicity. As barley is non-toxic due to the presence single A chain.

4.4 Datura, Nerium and Ipomea sps

1. The poisonous plant whose flowers are known as 'angel's trumpets' or 'moonflowers' is DATURA STRAMONIUM (Thorn apple).

2. Major tropane alkaloids present in *Datura stramonium* are HYOSCINE (Scopolamine) and ATROPINE (dl-hyoscyamine).

3. The toxic principles in datura are localized in SEEDS and FLOWERS.

Fig:39 Datura stramonium (Thron apple) [58-59]

4. The plant that is known as 'deadly night shade' is ATROPA BELLADONNA.
(Italian: Bella = Beautiful; Donna = Lady; Atropine causes dilatation of pupil (mydriasis) when instilled into eyes. In olden days, women used to beautify their eyes through atropine induced mydriasis. Hence the name bella-donna. Similar reasons are behind 'candle light dinners' of modern times, where dull candle light induces mydriasis, beautifying ones appearance.

5. The species that is more sensitive to datura poisoning is PIG.


Fig:40 Atropa belladonna (Deadly night shade)contains atropine [60-61]

6. The species that is resistant to *Atropa belladonna* is RABBIT.
   (Rabbits contains the enzyme atropinase enzyme, which destroys atropine)

6. Tropane alkaloids (atropine) act by inhibiting MUSCARINIC receptors in the body.

7. Symptoms of datura toxicity are termed as ANTICHOLINERGIC DELIRIUM.

8. Why fever is observed in atropine poisoning?
   Ans: 'Atropine fever' is a result of decreased secretions like salivation, sweating etc.

9. Urine from datura intoxicated animals produces MYDRIASIS in cat, which is used as experimental evidence in diagnosis.

10. The competitive inhibitor of acetyl choline esterase, which is used for treating datura (atropine) intoxication is PHYSOSTIGMINE.

11. Why phenothiazine tranquilizers are contraindicated in datura intoxication?
    Ans: In datura intoxication, anticholinergic symptoms are seen. As phenothiazines also possess anticholinergic activity, they are contraindicated for controlling CNS excitation in in datura intoxication.

12. The toxic principle in *Ipomoea turpethum* (Indian jalapa or morning glory) is TURPETHIN.

13. The toxic principle in *Ipomoea orizabensis* is SCAMMONIN (Jalapin)

14. Major symptom in ipomoea plant poisoning is DIARRHOEA.
    (The toxic resins present in ipomoea species produce drastic purgation).

15. Toxic principle in *Nerium oleandrum* (White oleander) is OLEANDRIN.

Fig:41 Ipomea trupethum (Operculina trupethum)- contains drastic purgation causing resin trupethin[62]

16. Toxic principle in *Nerium odorum* is NERIN.

17. The toxic glycosides in *Cerebra thevetia* are THEVETIN and CEREBRIN.

18. Steroidal glycoside in *Nerium indicum* is ODOROSIDE.

19. Most toxic part of the plants belonging to Nerium species are LEAVES.
(A leaf can kill a human and 2-3 leaves can kill a sheep. Leaf is effective even when dry).

Nerium oleandrum (Oleandrin)

Cerebra thevetia (Thevetin)

Fig:42 Cardiac glycoside containing plants [63-64]

20. The species that is most susceptible for oleander poisoning is HORSE.

21. Oleander glycosides act by inhibiting Na^+ - K^+ ATPase enzyme in cardiac cells.

22. Imbalance of the following electrolyte is observed in oleander poisoning
a. Sodium b. Potassium c. Magnesium d. Calcium
Ans: b.Potassium. Hyperkalemia produced in cardiac glycoside toxicity is fatal.

4.5 *Strychnous nuxvomica and Gossypium sps*

1. The toxic principle in *Strychnous nuxvomica* is STRYCHNINE.

2. Strychnine is localized in SEEDS part of *S.nuxvomica*.

3. Inhibition of GLYCINE neurotransmitter causes spinal stimulation in strychnine poisoning

4. The site of action of strychnine in spinal cord is RENSHAW cells.
   (*Recurrent inhibitory interneuron cells of reflex arc of spinal cord*).

5. The characteristic appearance of animal in strychnine poisoning is SAW HORSE.
   (Saw horse appearance is due to continuous tetanic seizures, which causes rigidity. Tetanus similarly produces saw horse appearance)

6. Preferred sedative for treating strychnine poisoning is PENTOBARBITONE (Barbiturate).

7. The following is (are) contraindicated in strychnine poisoning
   a. Ketamine b. Morphine c. Emesis d. All
   Ans: d. All. Ketamine causes motor stimulation; morphine causes respiratory depression; emesis causes seizures.

Fig:43 Strychnous nuxvomica – seeds contain strychnine [65]

8. Why strychnine is least toxic to chicken and pigeons?
   Ans:Strychnine is least toxic to chicken and pigeons as it is absorbed very slowly. However, other avians are easily affected (eg. crow).

9. The toxic principle in cotton seeds or seed cake is GOSSYPOL.

10. Gossypol binds with IRON mineral inhibiting heame synthesis and causing anemia.

11. Inhibition of the testicular enzyme LACTATE DEHYDROGENASE (LDH) is responsible for development of reproductive toxicity in males.

12. The maximum permissible amount of cotton seed cake in cattle ration ONE KG. (total dose)
(Further, feeding of cotton seed cake should be interrupted after 2-3 months for a period of 3-4 weeks).

Fig:44 Gossypium hirsutum – seeds contains the toxic principle gossypol [67-68]

13. Gossypol induced infertility is a result of LUTEOLYTIC effect on ovary.

14. Intake of high amounts of PROTEIN is protective against gossypol toxicity.

CHAPTER V

TOXICOLOGY OF AGROCHEMICALS

5.1 Organochlorine (OC) compounds

1. The group of heterogeneous substances that are used to control pests are known as PESTICIDES.
   (Pesticides include insecticides – insects; Herbicides – weeds; Fungicides – Fungi; Rodenticides – rats & mice)

2. Chlorinated hydrocarbons are known as ORGANOCHLORINE (OC) insecticides.
   (Eg. DDT, BHC, Endrin etc)

3. Why OC insecticides are being discouraged/banned?
   Ans: OC insecticides are not degradable and are persistent in the environment. And due to high lipid solubility, they accumulate in the food chain and enter human and animal bodies. Hence, OC compounds are being discouraged.

4. The following OC insecticides is not persistent in environment
   a. DDT b. Aldrin c. Methoxychlor d. Endosulfan
   Ans: c & d. Endosulfan and Methoxychlor

5. The most susceptible species for OC insecticide poisoning is CAT.

6. OC insecticides accumulate mainly in ADIPOSE tissue of the body.

7. The only OC insecticide that does not accumulate in adipose tissue is ENDOSULFAN.
   (Endosulfan is also biodegradable and not persistent in environment).

8. The OC compound that is bio-degradable by microbes is ENDOSULFAN.
   (Endosulfan has a sulphur ring in its structure, which is susceptible to microbial degradation. However, despite its ban in 2011 worldwide, it is still used in India).

9. The only isomer of benzene hexachloride (BHC) that is biodegradable is LINDANE (Gamma isomer).

10. OC compounds produce CNS excitation by prolonging DEPOLARIZATION phase of action potential.
    (Depolarization is prolonged by extending the opening of Na^+ channels and delaying the closure of K^+ channels. Aliphatic OC compounds like DDT, Methoxychlor etc follow this *mechanism).*

11. OC compounds act by inhibiting GABA receptors.
(Since, GABA is inhibitory, inhibition of GABA receptors leads to CNS excitation. Cyclodiene OC compounds like aldrin, endrin, endosulfan etc follow this mechanism).

12. Hyperthermia caused by OC insecticide poisoning is a result of altered metabolism of SEROTONIN and NORADRENALINE neurotransmitters.

13. The metabolite of DDT (Dichloro-diphenyl-trichloro-ethane) is DDE (Dichloro-Diphenyl-Dichloro- ethylene).

14. DDT acts as an agonist for ESTROGEN receptors, whereas, DDE acts as an antagonist for ANDROGEN receptors.

15. DDE, the metabolite of DDT causes thinning of egg shells due to inhibition of CALCIUM ATPase enzyme.
(DDE is known to cause thinning of egg shells causing decline of birds of prey – [Bald eagle, Pelican, Falcon] chicken, song birds).

16. The predominate symptoms in OC insecticide poisoning are BEHAVIORAL accompanied by HYPERTHERMIA.
(Organo phosphorus(OP) compounds do not produce behavioral symptoms and hyperthermia)

17. The sedatives of choice used in OC insecticide induced CNS excitation is BENZODIAZEPINES (Eg. Diazepam).
(Benzodiazepines also increases the affinity of GABA to its receptors, which was previously inhibited by OC insecticides).

18. Death in OC compound poisoning is due to RESPIRATORY failure.
(OC cause initial CNS stimulation followed by depression and coma. Hence, ultimately death is due to respiratory failure.

5.2 Organophosphate (OP) compounds

1. Organic insecticides which are esters of phosphorus are known as ORGANOPHOSPHATE (OP) compounds.

2. OP insecticides act as irreversible inhibitors of ACETYL CHOLINE ESTERASE (AChE) enzyme.
(AChE inhibition causes excess Ach accumulation producing cholinergic symptoms)

3. Why OP compounds are preferred insecticides for crops?
   Ans: OP compounds are biodegradable and are easily destroyed by sunlight, water, microbes, alkalis, metals etc. Hence, within 2-4 weeks of application, OP compounds are destroyed. However, they have considerable toxicity for mammals if consumed directly.

4. OP compounds, which are capable of inhibiting AChE without requiring metabolic activation are called DIRECT ACTING OP COMPOUNDS.
   (Direct acting compounds have P=O (Oxon) group, hence act without metabolic activation. Eg. TEPP, Dichlorovas, Sarin, Tabun).

5. Indirect acting OP compounds require metabolic activation to OXON form before producing toxicity.
   (Indirect acting agents have P=S (Thionate bond) which should be converted to P=O (Oxon) form before inhibiting AChE. Eg. Malathion, Parathion, Chlorpyrifos).

6. What are the metabolic active forms of Malathion and Parathion?
   Ans: Maloxon and Paroxon.

7. The toxicity of OP compounds INCREASES during storage.
   (Due to development of toxic isomers during storage)

8. The species of animal that is highly sensitive to OP poisoning is CAT.

9. The species of animal that is highly sensitive for delayed neuropathy caused by OP compounds (OPIDN) is CHICKEN.
   (Chicken brain has higher specific activity of NTE, hence is more susceptible)

10. OP compounds bind with AChE at ESTERATIC site causing irreversible inhibition.

11. Phosphorylation of AChE by OP compounds resulting in irreversible bonding due to the loss of alkyl group is called AGEING.

12. The agents, which are used to reactivate AChE inhibited by OP compounds are called OXIME REACTIVATORS (Eg. 2-PAM – Pralidoxamine).

13. Oxime reactivators are ineffective in reactivating AChE inhibited by OP compounds after AGEING stage.

14. OP compounds can inhibit the following enzyme(s)
    a. AChE b. BuChE c. NTE d. All
    Ans: d. All. AChE - true choline esterase; BuChE – Butryl choline esterase/ Pseudocholine esterase and NTE- Neurotoxic esterase.

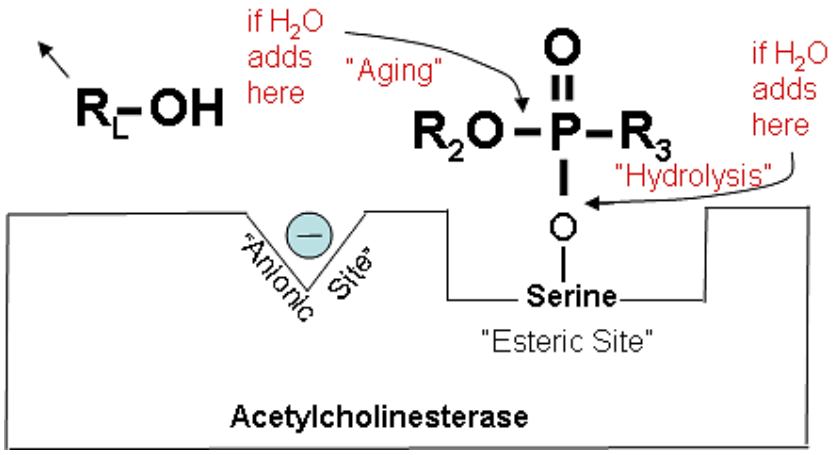


Fig:45 Ageing of acetylcholine esterase – Phosphorylation and loss of 'alkyl' group [69]

15. Pseudocholine esterase (BuChE) is present in PLASMA.
(Inhibition of BuChE is more important in birds than AChE inhibition).

16. OP induced delayed neuropathy (OPIDN) is caused due to the inhibition of NEURO TOXIC ESTERASE (NTE) enzyme. (also called Neuropathy Target Esterase)

17. The main symptoms in OP poisoning are CHOLINERGIC.
(Salivation, Lacrimation, Urination and Defecation).

18. In OPIDN, the type of paralysis observed is of ASCENDING FLACCID type.
(Hence, OPIDN is also known as dying back axonopathy. Mostly, large myelinated peripheral nerves are more affected).

19. The following type of toxicity produced by OP compounds is irreversible even with treatment
a. Acute b. Sub-acute c. Chronic d. OPIDN
Ans: d. OPIDN. There is permanent damage to the peripheral nerves.

20. Diagnosis of OP toxicity is carried out by measuring AChE activity in blood.
(Less than 25% activity of AChE is confirmative of OP poisoning. In birds, estimation of BuChE is important).

21. The specific antidote for OP poisoning is ATROPINE.
(Atropine inhibits cholinergic symptoms by blocking muscarinic receptors).

22. Oxime reactivators (2-PAM, DAM) should be used within 24-36 h time before ageing of AChE.

23. Use of atropine is contraindicated in CATS species, as it causes cyanosis.

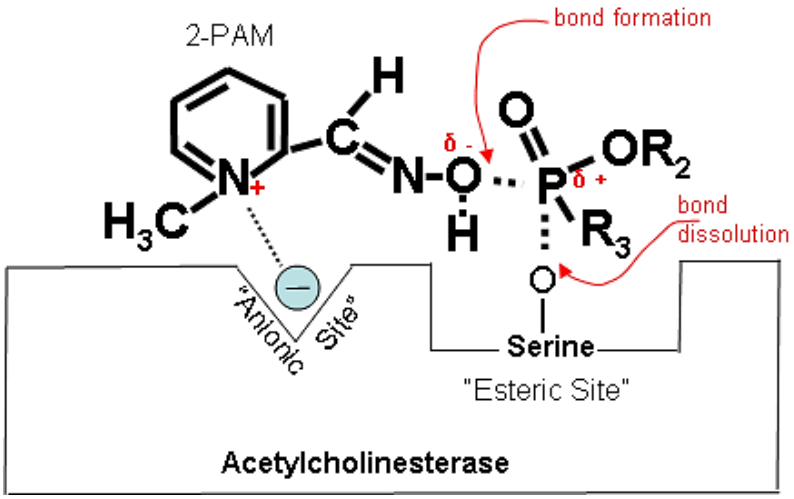


Fig:46 Mechanism of Pralidoxamine reactivation [69]

5.3 Carbamates

1. Why Carbamates are the preferred insecticides in veterinary use?
   Ans: Carbamates have broad spectrum of activity, low in mammalian toxicity and undergo rapid degradation in environment. Hence, carbamates are preferred for veterinary use. (Eg. Carbaryl, Propoxur, etc)

2. Insecticides that cause reversible inhibition of AChE are CARBAMATES.

3. Carbamates bind with AChE at the following site(s)
   a. Anionic b. Esteratic c. Cationic d. Both a & b
   Ans: d. Both a & b.

4. Oxime reactivators are contraindicated in CARBAMATE insecticide poisoning.
   (Carbamates bind with both sites of AChE leaving no room for oxime reactivators).

5. How carbamates and OP compounds differ interms of their binding to AChE?
   Ans: i. Carbamates bind with both anionic and esteratic sites where as OP compounds binds with esteratic site only
   ii. Carbamates cause carbomylation whereas OP compounds cause phosphorylation
   iii. Binding of carbamates is reversible whereas that of OP compounds is irreversible.

6. Why binding of carbamates with AChE is reversible?
   Ans: Carbamates causes carbomylation of AChE, which is weaker than phosphorylation caused by OP compounds. Hence, the binding is reversible.

7. Why carbamates are unable to produce delayed neuropathy?
   Ans; Carbamates cannot bind with neuro-toxic esterase. Hence, delayed neuropathy is not produced.

8. The least toxic carbamate that is used in veterinary practice against ectoparasites is CARBARYL.

9. Specific antidote for carbamate poisoning is ATROPINE.

5.4 Pyrethroids

1. Pyrethroids are natural insecticides obtained from CHRYSANTHEMUM flowers.
   (The use of marigold flowers is a common folk practice to prevent insects in food grains. The active ingredient in marigold is pyrethrin. Hence, synthetic compounds resembling it are called 'pyrethroids')

Fig:47 Chrysanthemum flowers – Source of natural pyrethrin [70]

2. The most widely used house-hold insecticides are SYNTHETIC PYRETHROIDS.
   (Least toxic and rapidly biodegradable. These agents are used in mosquito repellants - like good night, all out; Ant and cockroach repellants – Baygon, Cross line etc)

3. Type I pyrethroids resemble NATURAL PYRETHRINS in their structure and activity.
   (Eg. Allethrin, Pyrethrin, Permethrin etc)

4. Type II pyrethroids contain α-CYANO group in their structure and are more active and toxic.
   (Eg. Deltamethrin, Cypermenthrin, Fenvalarate etc)

5. The first commercial mosquito repellant pyrethroid still in use is ALLETHRIN.

6. Which of the following insecticides are more specific to arthropods?
   a. OC compounds b. OP compounds c. Carbamates d. Pyrethroids
   Ans: d. Pyrethroids. The LD_{50} of pyrethroids for insects and mammals is 1:4500, which means specific action towards insects.

7. The species that is highly susceptibility to pyrethroids toxicity is FISH.

8. The relationship between toxicity of pyrethroids and temperature is INVERSE.
   (As ambient temperature decreases the toxicity increases and vice versa).

9. Pyrethroids cause CNS stimulation by inhibiting the closure of VOLTAGE GATED SODIUM channels.
   (This leads to increased influx of Na^+ causes prolonged depolarization).

10. Voltage gated sodium channels are more sensitive to II (TWO) type of pyrethroids.

11. In type II pyrethroids produce characteristic 'TAIL' appearance in depolarization phase of action potential.

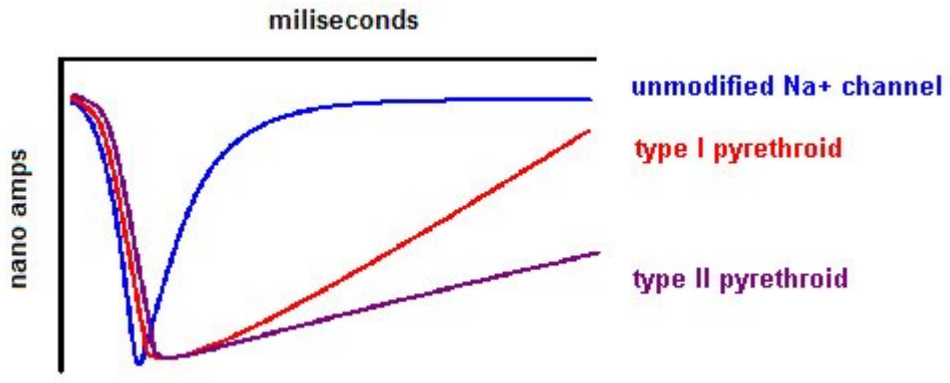


Fig:48 Characteristic 'tail' appearance in type II pyrethroid depolarization [71]

12. Type II pyrethroids acts as inhibitors for the receptors of GABA neurotransmitter.

13. The symptoms in type I pyrethroid toxicity are referred to as T-SYNDROME (Tremors).

14. Type II pyrethroids produces characteristic syndrome known as CS SYNDROME (Choreoathetosis and Salivation).

15. Tranquilizers that are contraindicated in pyrethroid poisoning are PHENOTHIAZINE.

16. The roots of plants belonging to Derris genus are the source of natural insecticide ROTENONE.

Fig:49 Derris plant – Root is the source of rotenone [72]

17. Rotenone is highly toxic for FISH species.
(Hence, derris is used to control not only insects but also fish (Piscicide)

5.5 Rodenticides

1. The agents which are used to control rodents are known as RODENTICIDES.

2. What is bait shyness?
Ans: A rodent surviving the exposure of a particular rodenticide will avoid the same rodenticide in the future. This phenomenon is called bait shyness.

3. What is secondary poisoning?
Ans: The poisoning that is seen in dogs and cats as a result of consuming rodenticide poisoned rodents.

4. Toxicity of Zinc phosphide (Zn_3P_2) is due to the release of PHOSPHINE (PH_3) gas.

5. The characteristic smell of phosphine gas, which can be used for diagnosis of zinc phosphide toxicity, is FISH LIKE or ACETYLENE.

6. Zinc phosphide releases phosphine gas in the following pH
a. Acidic b. Basic c. Neutral d. All
Ans: a. Acidic pH.
7. Why zinc phosphide preparations contain antimony potassium tartrate?
Ans: Antimony potassium tartrate is an emetic, which prevents accidentally ingested zinc phosphide through emesis in non-target species.

8. Why zinc phosphide is more toxic on full stomach than empty ?
   Ans: On full stomach, the acid production is increased. As zinc phosphide releases phosphine gas in acidic medium, full stomach increases the toxicity.

9. The metabolite of zinc phosphide is HYPOPHOSPHITE.
   (Hypophosphite is excreted in urine).

10. Phosphine gas acts as PROTOPLASMIC poison. *(It is also highly irritant to GIT).*

11. Bait shyness is not observed with ANTICOAGULANT rodenticides.
    (Anticoagulants are odourless and tasteless, and act after a lag period. Hence, the rodent forgets the exposure).

12. Warfarin inhibits clotting factors that are dependent on VITAMIN K for synthesis.
    (Vitamin K dependent clotting factors are II (Prothrombin), VII, IX and X).

13. Anticoagulant rodenticides decrease vitamin K synthesis through the inhibition of VITAMIN K EPOXIDE REDUCTASE enzyme.

14. Capillary damage seen in warfarin rodenticides is due to the presence of BENZALACTONE chemical moiety.
    (Mere reduction in clotting factors by anticoagulants is not fatal. Hence, benzolactone induced capillary damage is necessary to cause fatal hemorrhage)

15. The hematological tests used to confirm poisoning from anticoagulants are CLOTTING TIME and PROTHROMBIN TIME.
    (An increase of 2 to 6 times in clotting and prothrombin times are noted).

16. The specific treatment for anticoagulant poisoning is PHYTOMENADIONE (Vitamin K_1).
    (Menadione is a precursor of vitamin K. Menadione from synthetic sources is K_3; Menaquinone – K2 I from bacteria; Phytomenadione – K_1 is from plants).

17. Anticoagulant rodenticides effective against warfarin-resistant rats are SECOND GENERATION anticoagulants.
    (Hence, they are also called super-warfarins. Eg. Bromadiolone, Brodifacoum etc)

18. Risk of secondary poisoning is very high with 2^{ND} GENERATION ANTICOAGULANT rodenticides.

19. Fluoroacetate lowers energy production in the body by inhibiting TCA or KREBS CYCLE.

20. Why fluoroacetate rodenticide is devoid of bait shyness?
    Ans: Fluoroacetate develops toxicity after a lag period. Hence, the rodent forgets the exposure.

21. The prominent symptoms in fluoroacetate poisoning are NEUROLOGICAL.
(Brain is severely affected in the event of depletion of energy in body).

22. The competitive antagonist that is used in the treatment of fluoroacetate poisoning is GLYCERYL MONOACETATE.
(However, glyceryl monoacetate is not a specific antidote of fluoroacetate)

23. Why the use of fluoroacetate is restricted?
Ans: Fluoroacetate is highly toxic and non-specific affecting other species.

24. The rodenticide effective against warfarin-resistant rats is VITAMIN D (cholecalciferol).

25. The mechanism of toxicity of vitamin D compounds (cholecalciferol) rodenticides through the induction of HYPERCALCEMIA.
(Hypercalcemia causes calcification of visceral organs like kidney, blood vessels, heart and lungs).

26. Treatment for vitamin D rodenticide poisoning is CORTICOSTEROIDS and CALCITONIN.
(Both corticosteroids and calcitonin reduces hypercalcemia)

27. The phrase 'drowning in one's own fluids' is associated with ANTU (alpha-napthyl-thio-urea) rodenticide poisoning.
(ANTU causes leakage of fluid into lungs leading to froth formation and death)

28. Despite being very specific to rats, why ANTU is banned?
Ans: Due to carcinogenic potential of alpha napthylamine impurities found in ANTU.

29. The breed of rat, which is very sensitive for ANTU is BROWN or NORWAY rat.

29. Bait shyness and tolerance develops very rapidly for ANTU rodenticide.

30. The main symptom in ANTU poisoning is PULMONARY EDEMA.

31. What is the difference between pulmonary toxicity caused by ANTU (rodenticide) and Paraquat (herbicide)?
Ans: ANTU causes pulmonary edema which is fatal, whereas, paraquat causes pulmonary fibrosis which is not fatal.

32. Red squill, which is used as a rodenticide is obtained from the plant *URGENIA MARITIMA.*
(In south India, toxicity due to Indian squill (Urgenia indica) is more common)

33. Toxic glycosidic principle present in red squill is SCILLIROSIDE.

34. The concentration of scillirosides is highest in BULB part of the plant.

35. GIT micro flora act on scilliroside glycosides to release the aglycon portion SCILLIROSIDIN.

Urgenia maritime (Red squill)

Urgenia indica (Indian squill)

Fig:50 Red squill and Indian squill – Source of cardiac glycoside scilloriside [73-74]

36. The action of scilliroside glycosides is similar to DIGITALIS.
(The symptoms are cardiac arrhythmia and cardiac arrest)

37. The following rodenticides require multiple administration to cause death
a. Zinc phosphide b. Anticoagulants c. Vitamin D compounds d. Strychnine
Ans: b & c. Anticoagulants and vitamin D compounds require multiple administration to cause death.

5.6 Herbicides, Fungicides & Molluscicides

1. Phytotoxic chemicals used for controlling weeds are known as HERBICIDES.
2. The most widely used herbicide is 2, 4-D (2, 4-dichloro-phenoxy-acetic acid).
3. Herbicides enhance the palatability of certain poisonous plants by increasing the content of NITRATES.
4. The species of animal which is more sensitive for 2, 4-D is DOG.
5. Manufacturing impurities that make 2, 4-D extremely toxic are DIOXINS.
6. The herbicides which are potent uncouplers of oxidative phosphorylation are DINITROPHENOLS.
7. Urine is chrome-yellow coloured and turns black upon exposure to air in DINITROPHENOL herbicide poisoning.
8. The most toxic among the herbicides are BIPYRIDYL GROUP. (Eg. Paraquat, Diquat)

9. Paraquat selectively accumulates in LUNGS organ of the body.

10. Despite being highly toxic, bipyridyl herbicides are harmless after being sprayed on plants?
Ans: i. Bipyridyl herbicides are used in very low doses, which are not toxic
ii. They are inactivated immediately upon contact with soil.

11. The mechanism of toxicity of paraquat is through generation of SUPEROXIDE free radical, which affects unsaturated membrane lipids in lungs.

12. Why lung tissue is primarily affected by paraquat?
Ans: i. Paraquat accumulates up to 10 times in lungs.
ii. Lung tissue is deficient in superoxide dismutase (SOD) enzyme, which neutralizes superoxide radical. Hence, lungs are primarily affected.

13. The bipyridyl herbicide that can cause pulmonary fibrosis is PARAQUAT.

14. The agents which are used to prevent fungal infestation of plants or seeds are known as FUNGICIDES.

15. Fungicides that uncouple oxidative phosphorylation are PENTA-CHLORO-PHENOLS (PCP).

16. The mechanism of uncoupling of oxidative phosphorylation of PCP is due to PROTON INOPHORE activity.
(PCP being a lipid soluble weak acid carries protons (H^+) across mitochondrial membrane leading to depletion of proton gradient required for ATP synthesis. Hence, ATP synthesis is inhibited).

17. Dithio-carbamates such as ziram, thiram are derivatives of CARBAMATE insecticides.

18. Agents which are used to control snails and slugs are known as MOLLUSCICIDES.

19. The most commonly used molluscicide is METALDEHYDE.

20. Metaldehyde is extremely toxic though INHALATION route of exposure.

21. The active metabolite of metaldehyde is ACETALDEHYDE.

22. Metaldehyde inhibits GABA neurotransmitter in CNS, which results in excitation.

CHAPTER VI

RESIDUE TOXICOLOGY

1. What are Drug Residues?
   Ans: The presence of veterinary drugs and their metabolites along with associated impurities in any edible portion of the animal products is known as Drug Residue. (Codex Alimentarius Commission, 2004)

2. What is Withdrawal time?
   Ans: 'Withdrawal time' is the time between treatment and slaughter or milking in which, the drug/ pesticide residues deplete to a safe level (Tolerance/ Maximum residue level).

3. Who sets withdrawal times and levels for drug residues in animal products?
   Ans: Centre for Veterinary Medicine (CVM) of Food and Drug Administration (FDA) sets the withdrawal times and limits for approved food animal drugs.

4. Who sets the withdrawal times and limits for pesticide residues in animal products?
   Ans: Environment Protection Agency (EPA) sets the withdrawal times and limits for pesticide residues in plant and animal products.

5. How are with drawl times set for veterinary drugs?
   Ans: The following steps are involved in setting with drawl times

NOEL → ADI → Safety concentration → Tolerance/ MRL → Withdrawal time

- *No observable effect level (NOEL) is established in animals for the drug in question*
- *Average daily intake (ADI) values are determined*
  - *ADI = (NOAEL / 1000) x Ave. Body weight*

(ADI = Total dose of a drug that an average human can ingest on daily basis for entire life with no adverse effect)

- *Safe concentration is arrived by diving ADI with the amount of edible organ which is generally consumed in a day (Eg. Muscle 300 g; Liver 100g). Safe concentration includes all metabolites of a drug.*
  - *Safe concentration = ADI/300 (for muscle) ADI/100 (for liver)*

- *A marker residue which is sensitive and easy for detection is chosen (Parent compound or any of its metabolite).*

- *Tolerance level/ maximum residue level is ascertained through estimation of marker*

residue. Tolerance (MRL) = the % of safe concentration represented by the marker residue.

- *Studies are performed to know the <u>time</u> at which the target tissue is below tolerance, which is called withdrawal time.*

- *Withdrawal times are aplicable only when the drug is used according to label instructions. The tolerance for extra label use of a drug is Zero (i.e., detection of extalabel drug at any level leads to rejection of the product).*

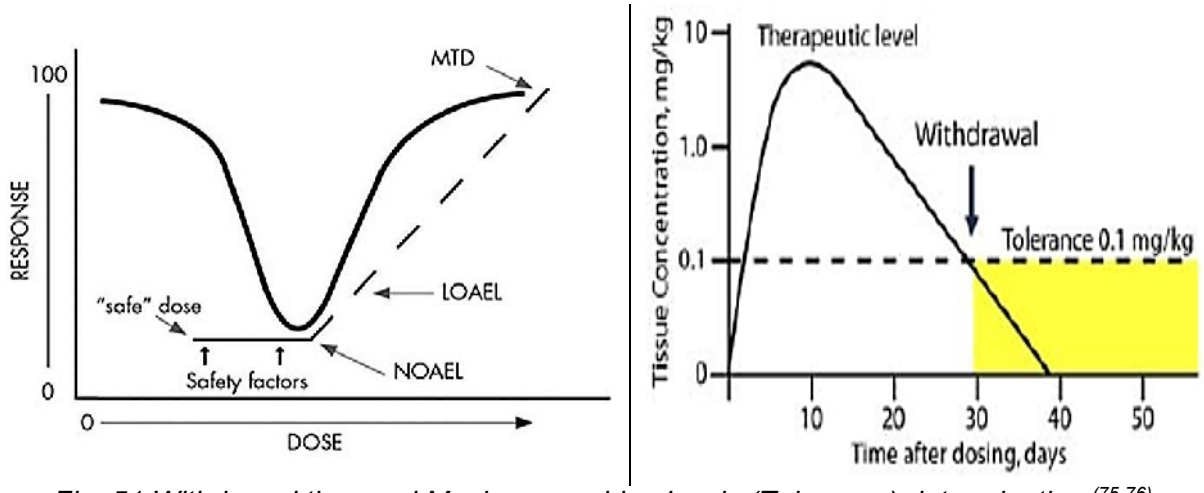


Fig: 51 Withdrawal time and Maximum residue levels (Tolerance) determination [75-76]

6. How withdrawal time can be extrapolated form pharmacokinetic parameters?
   Ans: Withdrawal time = 10 x $T_{1/2}$ ($T_{1/2}$ = half-life of the drug)

7. What is Tolerance / Maximum residue level?
   Ans: Maximum residue level is the maximum concentration of the administered veterinary drug, which is legally permitted or accepted in the food product.

8. What is the difference between 'Maximum residue level' and 'Maximum residue limit'?
   Ans: 'Maximum residue level' is used for drugs whereas 'maximum residue limit is used for pesticides.

9. What are the techniques used for detection of residues in foods?
   Ans: Techniques such as ELISA, HPLC, LC, GC, Paper chromatography.

10. What are the general categories of veterinary drugs found as residues in animal products?
    Ans: The general classes of veterinary drugs found in animal products are Antimicrobials, Anti-inflammatory, Growth promoters, Anti-parasitic and insecticides and Tranquilizers.

11. Which is the most common type of drug residue in animal products?
Ans: Antibiotics are the most frequently found drug residues (44%) followed by anti-inflammatory drugs.

12. In which type of animals drug residues are common?
Ans: Drug residues are most commonly found in Dairy cattle (43%) followed by beef cattle (17%).

13. What are the hazards of drug residues in animal products?
Ans: The hazards of drug residues in animal products are as follows
    - *Aesthetic issues: The presence of drugs in animal is not appealing for consumer.*
    - *Allergic reactions: Certain drug residues like Penicillins can cause allergic reaction in sensitive individuals at a level of 10 IU (0.6 µg)*
    - *Development of antibiotic resistance in microorganisms*
    - *Direct toxic effects – cancer, reproductive and developmental effects.*
    Eg: i. Clenbutarol residues were reported to have caused tachycardia, muscle tremor, headache, nausea and fever and chills in humans
    ii. Furazolidone and its metabolites are banned by FDA as they were reported to cause cancer in humans.
    - *Deleterious effects of hormone residues in humans*
    Eg: Diethyl stilbesterol (DES) – Vaginal clear cell adenocarcinoma in female off springs exposed in utero; Structural abnormalities of uterus
    - *International export barriers: Export of animal products need to comply with international standards for drug residues.*

14. What are the measures to be taken to prevent residues in animal products?
Ans: Measures to prevent drug residues include

    - *Education of veterinary personnel, organizations and government agencies involved in animal production.*
    - *Development of rapid screening methods for field use.*
    - *Avoiding irrational use of drugs in animals*
    - *Processing of animal products*
    Eg: Refrigeration completely destroys penicillins and pasteurization causes loss of activity of most of the antibiotics.
    - *Nationwide monitoring and surveillance for drug residues.*

CHAPTER VII

VENOUS ANIMALS

1. What is the difference between venom and poison (toxin)?
   Ans: Venom is produced by a specialized gland and is delivered either by biting or stinging. Poison (toxin) is contained in the tissues and is delivered through food. Eg: Snake – Venom; Puffer fish – Tetradotoxin (poison)

2. The order under the class insect that includes greatest number of poisonous insect species, is HYMENOPTERA.
   (The important families are Apidae [Honey bees]; Vespidae [Wasps] and Formicidae [Ants].Stinging from wasps is more common as they dwell in human and animal settlements)

Polistes annularis (Red paper wasp)

Apis mellifera (Honey bee)

Fig:52 Important stinging insects in the order Hymenoptera [77-78]

3. Why wasps can deliver multiple stings whereas honey bees only once?
   Ans: The stinger in honey bees is barbed, which is caught in the skin of the victim durig stinging. This results in the loss of stinging apparatus along with venom sac and death of the insect. In wasps, the stinger is plain and can be withdraw from victims skin. Hence, wasps sting multiple times.

Stinger of Wasp (Unbarbed)

Stinger of Honey bee (Barbed)

Fig: 53 Stinging apparatus of Wasp (Unbarbed) and Honey bee (Barbed) [79-80]

4. The antigenic component in honey bee venom that causes allergies or anaphylaxis is MELLITIN.

5. The drug of choice for treating systemic reactions from bee or wasp stings is EPINEPHRINE (ADRENALINE).

6. The potent cytotoxin present in ant venom is FORMIC ACID.

7. The piperidine alkaloid component of fire ant venom is SOLENOPSIN.
   (Piperidine alkaloids are also known as hemolytic factors and induce histamine release)

8. Focal necrotic ulcers of cornea and conjunctiva in calves are caused by the bite of FIRE ANTS.(*Soloenopsis invicta*) insect.

Fire ant (Solenopsis invicta) *Sterile pustule – fire ant bite*

Fig:54 Fire ant and bite of fire ant – sterile pustule [81-82]

9. The most potent neurotoxin in the venom of black widow spider (*Lactrodectus mactans*) is α-LACROTOXIN.

10. The most dangerous species of scorpion is *LEIURUS QUINQUESTRIATUS.*
    (The venom contains potent neurotoxins)

Fig: 55 Leirus quinquestriatus – Dangerous scorpion [83]

11. The most common tick species responsible for tick paralysis are RIPHICEPHALUS and DERMACENTOR.
    (Other species of ticks which can cause tick paralysis include Ixodes, Amblyomma, and Ornithodorus).

12. Tick paralysis is caused by the injection of neurotoxic SALIVA.

13. The most susceptible species for tick paralysis is DOG.

14. The type of paralysis seen induced by ticks is ASCENDING FLACCID.

Riphicephalus sps

Dermacenter sps

Fig:56 Important ticks causing tick paralysis [84-85]

15. The toxins from dermacentor species acts by blocking SODIUM channels, whereas, that of Ixodidae species by blocking the release of ACETYLCHOLINE in motor nerves.

16. Diagnosis of tick toxicity is made by observing FEMALE sex ticks on animal body along with paralysis.

17. Bufotixins (venom) in toads is produced primarily by PAROTOID glands.
(Skin also contains small glands, which produces bufotoxins but parotid glands are larger and are located dorsal and posterior to eyes [fig 57])

18. The most toxic toad is BUFO MARINUS.

19. The principal component of toad venom is BUFODIENOLIDES (Cardiac glycosides).
(The symptoms involve cardiac arrhythmia, ventricular fibrillation and heart failure)

20. The species in which frequent toad poisoning is observed is DOG.

21. Treatment of toad poisoning involves the use of PROPRANOLOL drug to control symptoms of cardiac arrhythmia and fibrillation.

22. Snake bite is commonly observed in DOGS and HORSES species of animals.

23. The type of toxins present in the venom of elapidae snakes (Cobra, Krait, Mamba, Coral snakes) are NEUROTOXINS.

24. The type of toxins present in the venom of viperidae snakes (Viper, Rattle snake, Adder) are HAEMOTOXINS.

Fig:57 Bufo marinus – Skin contains bufodienolide cardiac glycosides [86]

25. The neurotoxin present in the venom of kraits is α-BUNGAROTOXIN.

26. In snakes, the venomous glands are homologues to PAROTID glands in other animals.

27. Why snake bite in dogs is fatal compared to other animals?
Ans: Dogs are relatively smaller in size compared to other large animals like horses and cattle. Hence, the bite is fatal in dogs as the proportion of venom injected to body weight is very high.

28. The following snake bite results in prominent local reaction
a. Cobra b. Krait c. Sea snake d. Rattle snake
Ans: d. Rattle snake. The venom in viperidae (Vipers, rattle snake etc) snakes is hemotoxic and contains enzymes such as hyaluronidase. Hence, local reactions predominate.

29. The symptoms in elapine snake bite are predominantly NEUROLOGICAL.

30. Diagnosis of snake bite is possible by observing FANG marks on the body.

31. The main treatment for snake bite is MONOVALENT /POLYVALENT ANTIVENIN.
(If snake is identified, monovalent antivenin is sufficient otherwise polyvalent antivenin is necessary. Further, during treatment with antivenin, the development of allergic reactions should be controlled using epinephrine)

32. Why the use of alcohol is contraindicated for cleaning snake bite injuries?
Ans: Alcohol causes vasodilation promoting the spread of the venom in the body.

33. The only poisonous species of lizard is HELODERMA SUSPECTUM (Gila monster).

34. The inclusion of FISH MEAL component in feeds exposes animals to marine toxins.

Fig:58 Gila monster – Poisonous lizard [87]

35. The most potent among all marine toxins is TETRADOTOXIN (TTX).
(TTX is 10 times more toxic than snake venom; 100 times more toxic than black widow spider; 10000 times more toxic than cyanide)

36. Consuming TETRAODON (Puffer) /FUGU fish is commonly associated with tetradotoxin poisoning.

Fig:59 Puffer fish – Source of tetradotoxins [88-89]

37. In puffer fish, tetradotoxin (TTX) primarily accumulates in LIVER and OVARY.

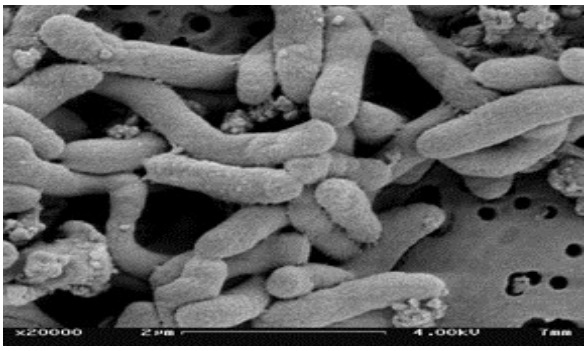


Fig:60 Pseudoaltermonas tetradonis – produces tetradotoxin [90]

38. Tetradotoxin is produced by the symbiotic bacteria PSEUDOALTEROMONAS TETRADONIS in puffer fish.

39. Why puffer fish are resistant to tetradotoxin?
Ans: Puffer fish have mutation altered sodium channels that are resistant to tetradotoxin.

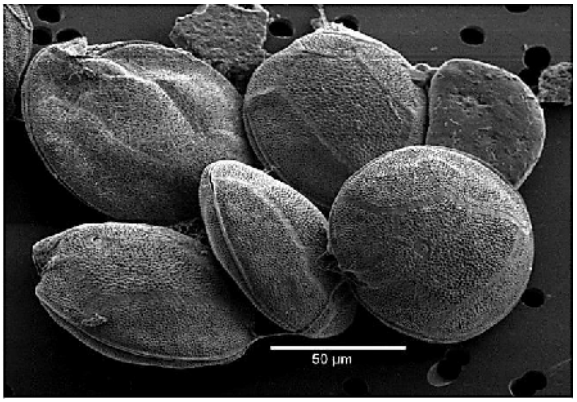
39. Tetradotoxin acts as a potent neurotoxin by blocking of SODIUM channel in central and peripheral nervous system.

40. Ciguatera is a fish-borne poisoning resulting from the consumption of REEF fish.
(Reef fish such as barracuda, eel etc).

Fig:61 Barracuda fish – Source of ciguatoxins [91]

41. The marine toxin responsible for ciguatera poisoning is CIGUATOXIN (CTX).

42. Ciguatoxin is produced by the dinoflagellate GAMBIERDISCUS TOXICUS present in reef fish.
(Similar to TTX, reef fish do not produce CTX, they only possess the toxin)



Gamberdiscus toxicus *Gamberdiscus toxicus (Ultrastructure)*

Fig:62 Microalgae (dinoflagellate) Gamberdiscus toxicans – produces ciguatoxin[92-93]

43. The most heat resistant marine toxin is CIGUATOXIN.

44. Why avoiding reef fish consumption is the only way to avoid ciguatera?
Ans: The detection of ciguatoxin is difficult as there are no perceptible organoleptic changes in fish. Further, CTX is not destroyed by temperature or gastric acid. Hence, avoiding reef fish consumption is the only way to prevent ciguatera.

45. Consumption of tuna and mackerel fish is associated with SCROMBROID poisoning.

46. Scrombroid poisoning is caused by HISTAMINE, which is produced from histidine through bacterial action.
(Other compounds such as puterscien and cadaverine are also involved in triggering histamine toxicity)

Tuna Fish *Mackerel Fish*

Fig:63 Tuna and Mackerel fish contains scrombroid toxins [94-95]

CHAPTER VIII

RADIATION TOXICOLOGY

1. The process of transmission of electromagnetic waves through a medium or vacuum is known as RADIATION.

2. Ionizing radiations produce ions by knocking out ELECTRONS from atoms.

3. Ionizing radiations – Particles type (α-particles, β-particles, neutrons) and Electromagnetic waves (X-rays, γ-rays, UV-rays).

4. The ionizing radiation that has least penetrating capacity is ALPHA (α)-PARTICLES.
(α-particles are large in size (2 protons+2 neutrons = α-particle). Hence, they have least penetration ability. However, due to their high mass, they have high linear energy transfer (LET) ability.

5. The ionizing radiation that has highest penetrating capacity is GAMMA (γ) RAYS.
(Since γ-rays are non-particulate electromagnetic radiation. Hence, they have the highest penetration ability. However, as the mass is least, LET is very low).

6. The type of UV rays, which reach earth are UV-A TYPE.
(UV-B&C are more powerful than UV-A rays, however, they are filtered by ozone layer (up to 99%).A sunscreen with both UV-A&B sun protection factor (SPF) of at least 25 is recommended in Indian conditions.

7. Non-ionizing radiations include– Visible light, Infrared rays, microwaves, radio waves.

8. The SI unit of radiation is GRAY (Gy) and the CGS unit is RAD (rad).

9. One Gray is equal to 100 rad. (or 1 rad = 0.01 Gy).

10. The instrument used to measure radiation is GEIGER-MULLER COUNTER.

11. What is the difference between radiation exposure and irradiation?
Ans: Both radiation exposure and irradiation, exposure to radiation occurs. However, in irradiation, post exposure contamination with radioactive material is absent (Eg. γ-irradiation of syringes; irradiation used for treating cancers etc).

12. Which two elements are responsible for natural background radiation in earth?
Ans: Radon (from uranium) and Thoron (from thorium) are responsible for 54% of natural background radiation in earth.

13. During nuclear explosion, the ascent and subsequent descent of radioactive material either in the vicinity or away from site of explosion is known as FALL OUT.
(Fall out can occur within few minutes to months and either in the vicinity or far away).

14. Among the radioactive material produced in nuclear explosions, the elements with biological significance are STRONTIUM (ST^{90}) and CESIUM (CS^{137}).
(Due to long half-lives)

15. The environmental hazard from nuclear power plants is due to the release of COOLANT, which is radioactive.
(Light water, Heavy water, liquid sodium etc are used as coolants).

16. Cells are more susceptible for radiation in the following stage(s) of cell cycle
a. M-Phase (mitosis) b. Early G-Phase c. Late G-Phase d. S-phase
Ans: a & b. M-phase and early G-phase. S-phase is resistant.

17. The group of symptoms that appear within 48h of exposure to radiation are known as PRODROMAL SYNDROME.
(The symptoms include nausea, vomition, anorexia, fatigue, diarrhea, sweating etc).

18. The sensitivity of rapidly proliferating cells to radiation is HIGHER than non-proliferative cells.

19. The most sensitive system for radiation exposure is HAEMOPOETIC SYSTEM.

20. The lethal dose of radiation in animals is expressed as $LD_{50/30}$
(Lethal dose required to causes 50% mortality in 30 days; In humans it is $LD_{50/60}$).

21. The cellular component, which is affected during radiation damage is DNA.

22. Molecules with unpaired electrons in the outer shells are known as FREE RADICALS.

23. Most common free radicals generated in the body are REACTIVE OXYGEN SPECIES (ROS).
(ROS include – Hydroxyl (OH^-), Superoxide (O^-_2), Singlet oxygen (O^-), Hydrogen peroxide (H_2O_2) etc; Reactive nitrogen species (RNS) – Nitric oxide (NO), peroxynitrate)

24. The most potent among the free radical is HYDROXYL (OH^-).
(OH^- is produced through Fenton's reaction where hydrogen peroxide acts on iron).

25. Why the presence of heavy metals generates more free radicals?
Ans; Heavy metals (like Fe, Pb, Cd etc) have more number of electrons in their outer shell. Hence, when a free radical attacks a heavy metal, there will be shower of electrons, which in turn produces more free radicals.

26. Damaging effect of free radicals on protein is PROTEIN OXIDATION and on DNA is DNA FRAGMENTATION.

27. The most sensitive hematological test for detecting radiation damage is ABSOLUTE LYMPHOCYTE COUNT.
(Lymphocyte count (LC) <1000 cells/cc within 24h or <500 cells/cc within 48h indicates severe exposure. If LC is >1500/cc after 48h post exposure, rules out radiation).

28. The most common type of cancer induced by radiation is LEUKAEMIA.

29. Grazing animals on fall-out pastures primarily affects GASTROINTESTINAL system.

30. The following organ is resistant to radiation
a. Endocrine glands b. Kidney c. Bone marrow d. Germinal cells.
Ans: a. Endocrine glands; as they have very low proliferation rate. The other organs are very sensitive due to high proliferative rate.

31. The amount of radio frequency radiation absorbed by human body while using mobile phone is measured by SPECIFIC ABSORPTION RATE (SAR).
(In India and US, permissible SAR is set at 1.6 Watt/Kg user mass; In Europe, SAR is set at 2.0 W/Kg).

32. The part of the head, which is sensitive to the heating (diathermic) effects of mobile phone is CORNEA.
(Cornea of the eye lacks temperature regulating mechanism and hence could be affected by dielectric heating from mobile phones during usage)

CHAPTER IX

TOXICOLOGY OF FOOD AND FEED ADDITIVES

9.1 Food & Feed additives

1. Non-nutritive substances added to food to improve the physical, organoleptic, nutritive properties or shelf life are called as FOOD ADDITIVES.

2. Standards relating to food production and safety are covered by CODEX ALIMENTARIUS COMMITTEE,

3. Agents which prevent oxidative damage of food are called as ANTIOXIDANTS.

4. The most toxic and suspected carcinogens among the food additives are COLOURING AGENTS.

5. The rare adverse effect of the colouring agent Tartrazine in humans is ALLERGY OR ANAPHYLAXIS.

6. The flavouring agent that produces characteristic 'candy-shop' aroma banned by FDA due to its carcinogenic potential is SAFROLE.

7. The flavor enhancing agent that produces 'umami' type taste in foods is MONOSODIUM GLUTAMATE (MSG).

8. 'Chinese restaurant syndrome' is caused by the food additive MONOSODIUM GLUMATE (MSG).
   (Symptoms include headache, flushing, tingling, numbness around mouth, palpitations etc. However, as exact association with adverse effects have not be found, MSG is still used. The use of MSG needs to be listed in food label).

9. The oldest artificial sweetener used in foods is SACCHARIN.
   (Saccharin was reported to produce epithelia hyperplasia of urinary bladder in male rats. However, due to lack of association between saccharin and cancer in humans at normal doses, WHO approved saccharine for human use).

10. The most common artificial sweetener approved by FDA for use in pharmaceutical products and foods is ASPARTAME.
    (Breakdown of aspartame produces phenylalanine and hence should be avoided by persons with 'Phenyl Ketonuria. Diet coke and other sugar-free products contain aspartame)

11. Vitamin C can reacts with benzoic acid – used as preservative in foods - to form BENZENE.

12. The substances which are added to animal feeds form improving quality of feeds or improving animal performance are called as FEED ADDITIVES.

13. Feed additives which increases growth rate and feed conversion in animals are called GROWTH PROMOTERS.

14. Antibiotics approved for use as growth promoters in food producing animals are IONOPHORE ANTIBIOTICS.
(Monensin, lasalocid and salinomycin are the commonly used ionophore antibiotics).

15. The most sensitive species for ionophores toxicity is HORSE.
(Horses are 10 times more sensitive than cattle).

16. The most common symptom in chronic ionophore toxicity is CARDIOMYOPATHY.

17. Antibiotic fed additive that should not be used simultaneously with other feed additives or growth promoters is ZINC BACITRACIN.

18. The use of Zinc bacitracin is contraindicated in LACTATING type of animals.

9.2 Urea and Salt Poisoning

1. Apart from dietary sources, animals get exposure to urea from FERTILIZERS
.
2. Excessive use of ammonium nitrate and urea fertilizers for crops can result in NITRATE poisoning in animals.

3. Urea is added to the diets of ruminants as a source of NITROGEN.
(Urea contains 46.7% nitrogen and 1 g of urea is equivalent to 2.92 g of protein)

4. The lethal dose of urea in cattle is 1 to 1.5 g /day/animal.
(4g/day/animal in horses)

5. Urea is permitted in the diets of ruminants and horses due to the presence of UREASE microbial enzyme in rumen and caecum respectively.
(Urease converts urea in to ammonia which is used for microbial amino acid synthesis).

6. The following reason(s) increases the susceptibility of ruminants to urea poisoning
   a. Urease present in plants b. Alkaline pH of rumen
   c. Acidic pH of stomach d. Urease activity of rumen
   Ans: a & b. Urease converts urea to ammonia. Both plants and rumen microbes contain urease enzyme.Further, alkaline pH of rumen facilitates this conversion. Henc, ruminants are more susceptible. In non-ruminants, the pH of stomach is acidic and the diet is not primarily from plants, hence they are least susceptible.

7. Which of the following age group is more resistant to urea toxicity?
   a. Calf b. Heifer c. Bull d. Cow
   Ans: a. Calf. In young ruminants the rumen microbial is not well developed hence less urease activity is seen.

8. The feed component that is required for efficient utilization of urea is CARBOHYDRATES or TOTAL DIGESTIBLE NUTRIENTS (TDN).
   (Carbohydrates (CHO) provide carbon skeleton for the production of amino acids. In cases of CHO deficiency, urea toxicity occurs due to unutilized ammonia).

9. The recommended ratio of urea to molasses for straws and other high fiber diets is 1:5.

10. In horses, the site of conversion of urea to ammonia is CAECUM.
    (The urease activity in horses is about 25% that of cattle)

11. Excessive ammonia inhibits TCA/ KREBS cycle in body decreasing energy production.

12. Ruminal pH above 7.5 is diagnostic of urea / non-protein nitrogen (NPN) poisoning.
    (Normal ruminal pH is 6.0 to 6.2, which increases due to release of ammonia)

13. Why the pH of the blood is acidic in urea poisoning?
    Ans: In liver detoxification of ammonia to urea requires bi-carbonate (HCO_3^-), which depletes blood HCO_3^- buffer leading to acidosis. Blood pH changes from 7.4 to 7.0 contrary to the assumption that released ammonia alkalizesblood.

13. The treatment in urea poisoning includes infusion of 5% ACETIC ACID (VINEGAR) and COLD WATER into rumen.
    (Acetic acid reduces ruminal pH and decreases conversion of urea to ammonia. Cold water reduces rumen temperature there by decreasing urease activity).

14. Intravenous infusions containing GLUCOSE/ DEXTROSE are contraindicated in urea poisoning as they cause hyperglycemia.

15. The toxic principle produced from the interaction of ammonia with reducing sugars in feeds is 4-METHYL IMMIDAZOLE (4-MI).
    (Similar to urea, ammonia treatment is used to improve the N_2 content of roughages)

16. 4-Methyl imidazole (4-MI) in ammoniated feeds produces CNS symptoms in cattle known as BOVINE BONKER'S SYNDROME.

17. Salt poisoning is commonly associated with deprivation of WATER.
(Hence, salt poisoning is termed aptly as water-deprivation-induced salt poisoning).

18. The most susceptible species for salt poisoning are POULTRY and PIGS.
(Poultry have poor sense of taste and indiscriminate feeding behavior).

19. Salt toxicity is consequent to the development of HYPEROSMOLALITY or HYPERTONICITY in blood and CSF.

20. Major symptoms evident in salt poisoning are NERVOUS SYMPTOMS.

21. Nervous symptoms in salt poisoning are a result of edem of BRAIN.

22. Dragging of hind limbs and knuckling of fetlock joints is characteristic feature of SALT poisoning in cattle.

23. In salt poisoning, pathognomonic histological appearance in brain is EOSINOPHILIC MENINGIOENCEPHALITIS.
(Perivascular cuffing of eosinophils is observed in cerebral cortex and meninges).

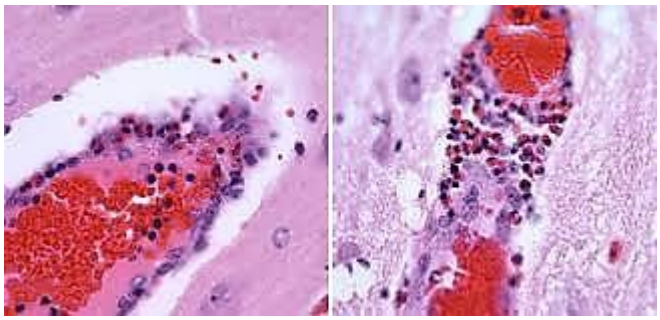


Fig:64 Eosinophilic Perivascular Cuffing – Salt poisoning [96]

24. The level of sodium in plasma and CSF that is diagnostic of salt poisoning is > 1mEq/L.
(Further, the level of sodium in CSF should be more than plasma).

25. Prognosis in case of salt poisoning is GRAVE.
(Less than 50% of affected animals survive with treatment; recumbent and convulsing animals definitely die irrespective of treatment).

CHAPTER X

MYCOTOXINS & BACTERIAL TOXINS

1. Secondary metabolites of fungus, which cause deleterious effects to animal and human life are called as MYCOTOXINS.

2. Molds generally grow in stored fed stuffs containing a moisture content more than 15%.
   (The other factors being a relative humidity of >85% and optimum temperature of 25-30^{o} C i.e., without refrigeration).

3. Mycotoxins are classified based on ORGAN SYSTEM affected.
   (Hepatotoxic – Aflatoxins, Rubra toxins; Nephrotoxic – Ochratoxin, Citrinin; Estrogenic – Zearalenone (F-2); Cytotoxic – Trichothecenes (T-2); Neurotoxic – Tremorgens).

4. Turkey X disease is caused by AFLATOXIN mycotoxins.

5. Aflatoxins are produced by *ASPERGILLUS FLAVUS* and *A.PARASITICUS.*

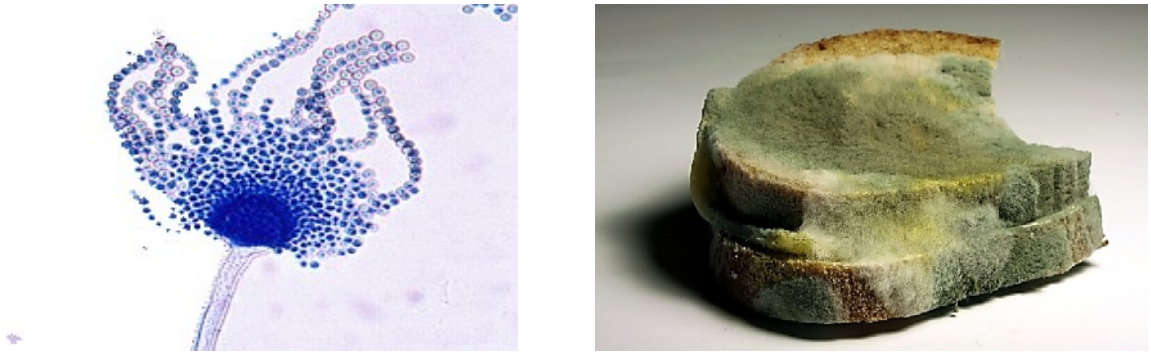


Fig:65 Aspergillus flavus – Source of aflatoxins (97-98)

6. The most potent among the aflatoxins is AFB_1.
   (The other being AFB_2, AFG_1 and AFG_2. The letters B (blue) and G (green) represent the type of light under which the respective toxin exhibits fluorescence.

7. Aflatoxins are heat resistant but are unstable in UV LIGHT.

8. Aspergillus species grows in stored feed stuffs containing moisture content more than 15%.

9. The following feed stuff(s) supports growth of aflatoxins
   a. Groundnut cake b. Soybean cake c. Cotton seed meal d. All
   Ans: All

10. The domestic species that are highly susceptible to aflatoxicosis are DOG and DUCKLINGS.

11. Aflatoxin has the following characteristic (s)
    a. Carcinogenic b. Mutagenic c. Teratogenic d. Immunosuppressive
    Ans: All of the above. Hence aflatoxins are considered more dangerous than other mycotoxins.

12. The metabolite of aflatoxins excreted through milk and urine is AFM_1.

13. Aflatoxin content in cattle feeds should not exceed 20 PPB.

14. The carcinogenic metabolite of aflatoxins is AFLATOXIN 8, 9-EPOXIDE.
    (Aflatoxins need biological activation to produce their toxic effects i.e., lethal synthesis)

15. Aflatoxins causes defective protein synthesis by binding with N-7 GUANINE residue of DNA causing mis-pairing of nucleotides.

16. Aflatoxin epoxide causes carcinogenic and mutagenic effect by causing ALKYLATION of the strands of DNA.
    (Alkylation forms cross bridges between DNA strands)

17. Hemorrhage in aflatoxicosis is due to the decrease in PROTHROMBIN and VITAMIN K

18. Which of the following forms of aflatoxicosis is most common
    a. Per acute b. Acute c. Sub acute d. Chronic
    Ans: d. Chronic. Chronic form occurs due to continuous intake of low level of aflatoxins.

19. The type of carcinoma caused by aflatoxins is HEPATOCELLULAR CARCINOMA.

20. Alfatoxins can be detected by THIN LAYER CHROMATOGRAPHY method.

21. Hemorrhagic syndrome in poultry is caused by RUBRATOXINS mycotoxins.

22. Rubratoxins are produced by *PENICILLIUM RUBRUM* and *P.PURPUROGENUM.*

23. The most toxic metabolite of rubratoxins is RUBRATOXIN B.
    (Rubratoxin B is hepatotoxic, mutagenic and teratogenic)

24. Rubratoxins are destroyed at the following temperature
    a. Freezing b. Room temperature c. 50 to 60^{o}C d. 85 to 100^{o}C
    Ans: d. 85 to 100^{o}C for two hours can destroy rubratoxins

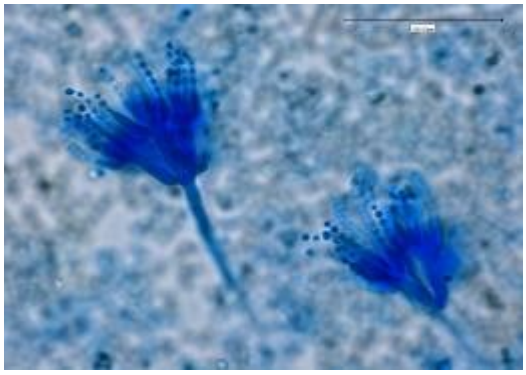



Fig:66 Penicillium rubrum (painting brush appearance) – source of rubratoxins [99-100]

25. Ruminant's facial eczema is caused by SPORIDESMIN mycotoxicosis.

26. Nephrotoxic mycotoxins responsible for mold nephrosis or mycotoxic nephropathy are OCHRATOXINS.

27. Ochratoxins are produced by *ASPERGILLUS OCHRACEUS* and *PENICILLIUM VIRIDICATUM.*

28. The most toxic among the ochratoxins is OCHRATOXIN A.

29. The most susceptible species for ochratoxicosis are BIRDS and PIG.

30. The site of action of ochratoxins in the nephron is
    a. Proximal Convoluted Tubule b. Loop of henle
    c. Distal Convoluted Tubule d. Collecting duct
    Ans: a.Proximal convoluted tubule. Inhibits anion transport and damages renal brush border.

31. The level of ochratoxin in feed should not exceed 10 PPB.

32. Estrogenic mycotoxin responsible for reproductive disorders is ZEARALENONE (F-2).

33. Zearalenone (F-2) mycotoxins are produced by *FUSARIUM ROSEUM* mold.

34. The most susceptible species for zearalenone toxicosis is PIG.

35. Vulvo-vaginitis or hyper-estrogenic syndrome in pigs is caused by ZEARALENONE mycotoxin.

36. The maximum permitted level of zearalenone in feed is 10 PPB.

Fig:67 Fusarium roseum – Source of Zearalenone toxins [101-102]

37. Why pigs and cattle differ in their susceptibility to zearalenone?
Ans: In pigs, the major metabolite of Zearalenone is α-zearalenol, which has greater affinity for estrogen receptors than β-zearalenol, which is formed in cattle. Hence, pigs are more susceptible than cattle.

38. Name the hormone involved in ovarian follicle maturation that is inhibited by Zearalenone.
Ans: Follicle Stimulating hormone (FSH).Henc, the characteristic symptom of follicular atresia is observed in ovaries.

39. Histological picture of uterus and vaginal in zearalenone toxicosis is METAPLASIA.

40. Alimentary toxic aleukia (ATA) in human being is caused by TRICHOTHECENE mycotoxins.
(Trichothecenes commonly includes T-2 toxin, DON and DAS. DON -Deoxy-nevalenol [vomition] and DAS -Diacetoxy-scirpenol).

41. The mycotoxins that were used as biological warfare agents are TRICHOTHECENES.
(Alleged 'yellow rain' attacks of Vietnam on local tribes, who supported US during Vietnam war)

42. The species that is more susceptible to trichothecenes is CAT.

43. An example for neurotoxic mycotoxin is TREMORGENS.
(The name 'tremorgens' indicates muscle tremors produced by the toxins due to CNS effects).

44. The most potent among the tremorgens is PENITREM A.

45. The most susceptible species for tremorgen mycotoxins are CATTLE and DOG.

46. Staggers syndrome in cattle is produced by TREMORGEN mycotoxins.

47. Ergotoxins are produced by the mold *CLAVICEPS PURPUREA*.
(Important ergot alkaloids are ergotamine and ergometrine)

Fig: Rye affected with C.purpurea *Sclerotia of C.purpurea around grains*

Fig:68 Claviceps purpurea – source of ergot alkaloids – Ergotism [103-104]

48. Most common cause of ergotism in cattle is feeding of RYE grass..
(Claviceps purpurea grows mainly on rye grass. The fungi forms a dense sclerotia around the seeds)

49. Ergot alkaloids are partial agonist for ALPHA receptors.
(Hence, vasoconstriction is seen in ergotism leading to gangrene in extremities)

50. The ergot alkaloid which has oxytocic effect on uterus is ERGOMETRINE or ERGONOVINE.

51. Acute egotism is manifested as NERVOUS form while chronic ergotism is manifested as GANGRENOUS form.

52. The type of ergotism commonly found in cattle is of GANGRENOUS form.

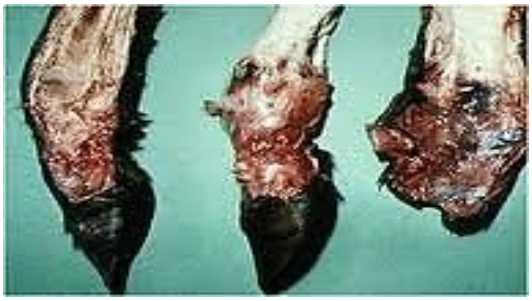


Fig:69 Gangrenous ergotism – due to vasoconstrction in extremities [105-106]

53. In buffaloes, fusarium mycotoxins cause DEGNALA disease, which is similar to chronic ergot posioing.
(In 2012, an epidemic of degnala disease was reported in south east Asia due to feeding of rice straw contaminated with fusarium species)

54. Botulin exo-toxins are produced by *CLOSTRIDIUM BOTULINUM* microorganism.

55. Botulism is most commonly seen in CHICKEN species.

56. Which of the following serotype(s) of botulinum is most commonly implicated in animals and poultry?
a. A-type b. B-type c. C-type d. D-type
Ans: c & d. Type C and D botulinum toxins are mostly implicated in animals and birds.

57. The food borne toxicity resulting from pre-formed toxins is BOTULISM.
(Anaerobic environment favours the production of botulinum toxin. Hence, tinned foods are more likely to contain botulinum toxins).

58. In birds, limber neck - due to paralysis of neck muscles - is caused by BOTULINUM toxins.

59. The practice of using chicken manure as cattle feed or fertilizer can cause BOTULISM food borne toxicosis.

60. Botulinum acts as a neurotoxin by inhibiting the release of ACETYL CHOLINE neurotransmitter.
(Due to inhibition of ACh release, flaccid paralysis is seen).

61. Specific treatment for botulism is POLYVALENT BOTULINUM ANTITOXIN.

62. Tetanus toxin (also called tetanospasmin) is produced by *CLOSTRIDIUM TETANI.*

63. Which of the following toxicity is infectious?
a. Botulism b. Tetanus
Ans: b. Tetanus. Tetanus can spread from one animal to another whereas botulism is a food-borne toxicity.

64. The most susceptible species for tetanus is HORSE.

65. 'Saw-horse' or 'Wooden-horse' condition in horses is caused by TETANUS.
(Due to spasms and stiffness in neck, back and leg muscles, the animal assumes wooden horse appearance. Prolapse of third eyelid is also observed)

66. Spinal stimulation in botulism is due to the inhibition of GLYCINE neurotransmitter.

67. Skeletal muscle contractions seen in tetanus are of the following type
a. Clonic b. Tonic c. Both d. Twitching
Ans: b. Tonic. Tonic muscle contraction without twitching seen in tetanus is called Tetany.

68. The specific treatment for tetanus is TETANUS ANTITOXIN.

69. The vaccine used against tetanus consists of TETANUS TOXOID.
(All toxins inactivated with formaldehyde are termed as toxoids)

CHAPTER XI

ENVIRONMENTAL POLLUTANTS

1. The process of contamination of environment (air, water, soil) through the discharge of harmful substances is known as POLLUTION.

2. The size of the particles that can reach alveoli of lungs is <1 µm.
(Particles of 5 µm or more are deposited in upper respiratory tract and between 1-5 µm in the terminal airways)

3. Pollutants released directly into atmosphere either through natural or anthropogenic sources are called PRIMARY pollutants.
(Eg. Carbon monoxide, carbon dioxide etc)

4. Pollutants synthesized as a result of interaction of primary pollutants in the atmosphere are known as SECONDARY pollutants.
(Eg. Ozone, Peroxyacetyl nitrates [PAN])

5. Man-made sources or anthropogenic sources constitute 98% percent of pollution.

6. The major air pollutant is CARBON MONOXIDE (CO).
(CO – 52%, Sulphur oxide – 18%, hydrocarbons – 12% are the top three air pollutants).

7. The pollutant that is produced due to incomplete combustion is CO.

8. The lethal concentration of *CO* in air is 400 PPM.

9. The major source of CO in urban setting is AUTOMOBILES.

10. CO binds with hemoglobin forming CARBOXY HAEMOGLOBIN.
(Since CO has 200 times more affinity for HB than oxygen, the oxygen carrying capacity of blood is severely hampered).

11. The colour of blood in CO poisoning is CHERRY RED.
(Carboxy hemoglobin gives characteristic cherry red colour to blood. Hence, despite of asphyxia, cyanosis is absent. The skin and mucosa are red .)

12. The main symptom in CO poisoning is HYPOXIA.
(Brain and heart are very sensitive for hypoxia as they have high oxygen requirement)

13. Specific therapy for CO poisoning is OXYGEN.

14. The normal concentration of carbon dioxide in atmosphere is 0.5% (5000 PPM)

15. Symptoms of CO_2 poisoning are evident at a concentration of 5% in air.

16. The process of elevation of earth's temperature due to increase in green-house gases in the atmosphere is called GLOBAL WARMING.
(Important green-house gases are CO_2, Methane, water vapour etc)

17. Name the green-house gas of animal origin, which is implicated in global warming?
Ans: Methane; It is produced in ruminants due to bacterial fermentation in rumen.

18. Primary pollutants responsible for acid-rains are SULPHUR & NITROGEN OXIDES.
(Sulphur oxides combine with water vapour either in air or body to form sulphurous or sulphuric acid. The other component is nitrogen oxides, which similarly form nitric acid upon contact with moisture)

19. The level of SO_2 in air which is considered dangerous is 100 PPM.

20. Why SO_2 is used in canned meat products?
Ans: SO_2 acts as a preservative; further, it masks the odour of the meat and improves colour.

21. 'Silo-fillers disease' or 'Silage gas' poisoning is caused by NITROGEN DIOXIDE (NO_2).

22. The level of nitrogen oxides (NO_2 and N_2O_4) in air harmful to life is 100 PPM.

23. The major system affected by NO_2 and SO_2 poisoning is RESPIRATORY system.

24. Painter's syndrome is caused due to continuous exposure to SOLVENT vapours.

25. Encephalopathy is a result of chronic exposure to SOLVENTS.
(Endocrine disruption is also reported for organic solvents. Organic solvents include benzene, toluene, gasoline, petrol, kerosene etc. Bad news for those who like the smell of petrol, kerosene etc)

26. The pollutant which can form ozone in the atmosphere by absorbing UV light is NO_2.

27. The pollutant that can cause permanent damage to lungs even with short-term exposure at low concentrations is OZONE (O_3).

28. The combination of smoke and fog results in the formation of SMOG which considerably reduces visibility.

Fig:70 Smog – Smoke + Fog – Reduces visibility [107]

29. Important contributor for development of photochemical smog is PEROXY ACETYL NITRATE (PAN).
(PAN is mutagenic and can cause skin cancer).

30. Safe level of total dissolved solids (TDS) in drinking water for animals is <1000 PPM.

31. The species that is highly sensitive for polychlorinated biphenyls (PCB) toxicity is MINK.

Fig:71 Mink – Most susceptible to PCB posioning [108]

32. PCB inhibits the synthesis of the central neurotransmitter DOPAMINE in brain.

33. Feminization of male foetus is caused by PCB pollutants.
(PCB are endocrine disruptors possessing estrogenic activity that causes feminization of male foetus).

34. 'Cola-coloured babies' are born when mothers are exposed to PCB pollutants during pregnancy.

35. 'Chick oedema' is a characteristic clinical condition produced by PCB pollutants.

36. Water-treatment processes such as chlorination leads to the production of TRIHALOMETHANES (THM) due to reaction of chlorine with organic matter.

-END-

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