

Animal Science

Animal Health - II

Grade 10



Government of Nepal
Ministry of Education, Science and Technology
Curriculum Development Centre
Sanothimi, Bhaktapur

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Technical and Vocational Stream
Learning Resource Material

Animal Health - II
(Grade 10)
Animal Science



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Preface

The curriculum and curricular materials have been developed and revised on a regular basis with the aim of making education objective-oriented, practical, relevant and job oriented. It is necessary to instill the feelings of nationalism, national integrity and democratic spirit in students and equip them with morality, discipline, self-reliance, creativity and thoughtfulness. It is essential to develop linguistic and mathematical skills, knowledge of science, information and communication technology, environment, health and population and life skills in students. It is also necessary to bring the feeling of preserving and promoting arts and aesthetics, humanistic norms, values and ideals. It has become the need of the present time to make them aware of respect for ethnicity, gender, disabilities, languages, religions, cultures, regional diversity, human rights and social values to make them capable of playing the role of responsible citizens with applied technical and vocational knowledge and skills. This learning resource material for Animal Science has been developed in line with the Secondary Level Animal Science Curriculum with an aim to facilitate the students in their study and learning on the subject by incorporating the recommendations and feedback obtained from various schools, workshops, seminars and interaction programs attended by teachers, students and parents.

In bringing out the learning resource material in this form, the contribution of the Director General of CDC Mr. Yubaraj Paudel and members of the subject committee Dr. Manraj Kolakshpati, Madhukumari Tiwari, Lavdev Bhatta is highly acknowledged. The learning resource material is written by Dr. Ganesh Gautam Dr. Shibalal Bhandari and Dr. Asis Mahat the subject matter of the materials, was edited by Mr. Badrinath Timsina and Mr. Khilanath Dhamala and language was edited by Mr. Raju Shrestha. CDC extends sincere thanks to all those who have contributed to developing this material in this form.

This learning resource material contains a wide coverage of subject matters and sample exercises which will help the learners to achieve the competencies and learning outcomes set in the curriculum. Each chapter in the material clearly and concisely deals with the subject matters required for the accomplishment of the learning outcomes. The Curriculum Development Centre always welcomes constructive feedback for the betterment of the material.

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Guidelines to Teachers

A. Facilitation Methods

The goal of this course is to combine the theoretical and practical aspects of the contents needed for the subject. The nature of contents included in this course demands the use of practical or learner focused facilitation processes. Therefore, the practical side of the facilitation process has been focused much. The instructor is expected to design and conduct a variety of practical methods, strategies or techniques which encourage students engage in the process of reflection, sharing, collaboration, exploration and innovation new ideas or learning. For this, the following teaching methods, strategies or techniques are suggested to adopt as per the course content nature and context.

Brainstorming

Brainstorming is a technique of teaching which is creative thinking process. In this technique, students freely speak or share their ideas on a given topic. The instructor does not judge students' ideas as being right or wrong, but rather encourages them to think and speak creatively and innovatively. In brainstorming time, the instructor expects students to generate their tentative and rough ideas on a given topic which are not judgmental. It is, therefore, brainstorming is free-wheeling, non-judgmental and unstructured in nature. Students or participants are encouraged to freely express their ideas throughout the brainstorming time. Whiteboard and other visual aids can be used to help organize the ideas as they are developed. Following the brainstorming session, concepts are examined and ranked in order of importance, opening the door for more development and execution. Brainstorming is an effective technique for problem-solving, invention, and decision-making because it taps into the group's combined knowledge and creative ideas.

Demonstration

Demonstration is a practical method of teaching in which the instructor shows

or demonstrates the actions, materials, or processes. While demonstrating something the students in the class see, observe, discuss and share ideas on a given topic. Most importantly, abstract and complicated concepts can be presented into visible form through demonstration. Visualization bridges the gap between abstract ideas and concrete manifestations by utilizing the innate human ability to think visually. This enables students to make better decisions, develop their creative potential, and obtain deeper insights across a variety of subject areas.

Peer Discussion

Peer conversation is a cooperative process where students converse with their peers to exchange viewpoints, share ideas, and jointly investigate subjects that are relevant or of mutual interest. Peer discussion is an effective teaching strategy used in the classroom to encourage critical thinking, active learning, and knowledge development. Peer discussions encourage students to express their ideas clearly, listen to opposing points of view, and participate in debate or dialogue, all of which contribute to a deeper comprehension and memory of the course material. Peer discussions also help participants develop critical communication and teamwork skills by teaching them how to effectively articulate their views, persuasively defend their positions, and constructively respond to criticism.

Peer conversation is essential for professional growth and community building outside of the classroom because it allows practitioners to share best practices, work together, and solve problems as a group. In addition to expanding their knowledge horizon and deepening their understanding, peer discussions help students build lasting relationships and a feeling of community within their peer networks.

Group Work

Group work is a technique of teaching where more than two students or participants work together to complete a task, solve a problem or discuss on a

given topic collaboratively. Group work is also a cooperative working process where students join and share their perspectives, abilities, and knowledge to take on challenging job or project. Group work in academic contexts promotes active learning, peer teaching, and the development of collaboration and communication skills. Group work helps individuals to do more together than they might individually do or achieve.

Gallery Walk

Gallery walk is a critical thinking strategy. It creates interactive learning environment in the classroom. It offers participants or students a structured way to observe exhibition or presentation and also provides opportunity to share ideas. It promotes peer-to-peer or group-to-group engagement by encouraging participants to observe, evaluate and comment on each other's work or ideas. Students who engage in this process improve their communication and critical thinking abilities in addition to their comprehension of the subject matter, which leads to a deeper and more sophisticated investigation of the subjects at hand.

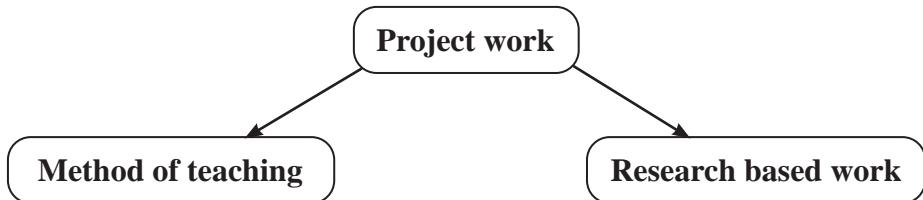
Interaction

The dynamic sharing of ideas, knowledge, and experiences between people or things is referred to as interaction, and it frequently takes place in social, academic, or professional settings. It includes a broad range of activities such as dialogue, collaboration or team work, negotiation, problem solving, etc. Mutual understanding, knowledge sharing, and interpersonal relationships are all facilitated by effective interaction. Interaction is essential for building relationships, encouraging learning, and stimulating creativity in both in-person and virtual contexts. Students can broaden their viewpoints, hone their abilities, and jointly achieve solutions to difficult problems by actively interacting with others.

Project Work

Project work is a special kind of work that consists of a problematic situation which requires systematic investigation to explore innovative ideas and solutions.

Project work can be used in two senses. First, it is a method of teaching in regular class. The next is: it is a research work that requires planned investigation to explore something new. This concept can be presented in the following figure.



Project work entails individuals or teams working together to achieve particular educational objectives. It consists of a number of organized tasks, activities, and deliverables. The end product is important for project work. Generally, project work will be carried out in three stages. They are:

- Planning
- Investigation
- Reporting

B. Instructional Materials

Instructional materials are the tools and resources that teachers use to help students. These resources/materials engage students, strengthen learning, and improve conceptual comprehension while supporting the educational goals of a course or program. Different learning styles and preferences can be accommodated by the variety of instructional resources available. Here are a few examples of typical educational resource types:

- Daily used materials
- Related Pictures
- Reference books
- **Slides and Presentation:** PowerPoint slides, keynote presentations, or other visual aids that help convey information in a visually appealing and organized manner.
- **Audiovisual Materials:** Videos, animations, podcasts, and other

multimedia resources that bring concepts to life and cater to auditory and visual learners.

- **Online Resources:** Websites, online articles, e-books, and other web-based materials that can be accessed for further reading and research.

Maps, Charts, and Graphs: Visual representations that help learners understand relationships, patterns, and trends in different subjects.

Real-life Examples and Case Studies: Stories, examples, or case studies that illustrate the practical application of theoretical concepts and principles.

C. Assessment

Formative Test

Classroom discussions: Engage students in discussions to assess their understanding of concepts.

Quizzes and polls: Use short quizzes or polls to check comprehension during or after a lesson.

Homework exercises: Assign tasks that provide ongoing feedback on individual progress.

Peer review: Have students review and provide feedback on each other's work.

Summative Test

Exams: Conduct comprehensive exams at the end of a unit or semester.

Final projects: Assign projects that demonstrate overall understanding of the subject.

Peer Assessment

Group projects: Evaluate individual contributions within a group project.

Peer feedback forms: Provide structured forms for students to assess their peers.

Classroom presentations: Have students assess each other's presentations.

Objective Test

Multiple-choice tests: Use multiple-choice questions to assess knowledge.

True/False questions: Assess factual understanding with true/false questions.

Matching exercises: Evaluate associations between concepts or terms.

Portfolio Assessment

Compilation of work: Collect and assess a variety of student work samples.

Reflection statements: Ask students to write reflective statements about their work.

Showcase events: Organize events where students present their portfolios to peers or instructors.

Observational Assessment

Classroom observations: Observe students' behavior and engagement during class.

Performance observations: Assess practical skills through direct observation.

Field trips: Evaluate students' ability to apply knowledge in real-world settings.

Introduction to Parasite and Parasitology

Unit
1

1.1 Parasite and Parasitology

Parasite is an organism that lives inside or outside the body of organism and drives substances from it without rendering any benefit to it. A parasite is a small organism that lives on or in, at the expense of a larger organism called host. Host generally provides food and shelter for parasite. The study of the phenomenon of the parasitism is called parasitology.

Parasitology is multidisciplinary subject which is related to the field of biochemistry, Physiology, Cell biology, Immunology, pathology, and Pharmacology.

Animal association may be divided into :

1. **Phoresis**- to carry.
2. **Commensalism**- host is neither benefited nor harm.
3. **Symbiosis**- Interdependent upon each other that one can't live without the help of the other.
4. **Mutualism**- It is similar to symbiosis but association between host and parasite is not essential.
5. **Parasitism**- Parasite is always benefited and host is almost always harmed.

1.2 Types of Parasites: External and Internal Parasites

1. **Internal parasite (Endo-parasite)** : It lives inside the body of the host. Internal parasites live in the the blood, tissue, body cavities, digestive tract and other organs e.g. trematodes, cestodes, and nematodes.

2. **External parasite (Ecto-parasites)** : It lives outside on the surface of the body of the host e.g. lice, ticks, mites, fleas, and leech.
3. **Temporary parasite**: It visits the host for a short period e.g. mosquito, sand fly, fleas, and bedbug.
4. **Permanent parasite**: lives a parasitic life in or on the host body throughout the whole period of its life.
5. **Facultative parasite**: lives a parasite life when opportunity arises or a facultative parasite is able to live either free or as parasite e.g. *Strongyloids* spp., *Noegleria fowleri* etc
6. **Obligatory parasite**: Can't exist without a parasite life e.g. *Trichinella spiralis*, malarial parasite
7. **Incidental parasite (accidental parasite)**: Attacks an unusual host in which they do not occur normally e.g. *Fasciola hepatica* sometimes occur in men.
8. **Wandering parasite** : Happens to reach a place or organ where it can't live e.g. *Stephanurus dentatus* (sometimes found in the liver of pig but the usual site is the pelvis of the kidney).
9. **Periodic parasite**: Visits their host at the time to obtain some metabolic requirement or some other benefits e.g. mosquitoes, flies, and tabanus.

1.3 Types of Host: Definitive Host and Intermediate Host

A **host** is an organism that provides a habitat, nourishment, or shelter for a parasite, either temporarily or permanently. The types of host are described below with examples:

1. Definitive Host (Primary Host)

The host in which the parasite reaches maturity and undergoes sexual reproduction. for example: Humans as the definitive host for *Plasmodium* species (malaria parasite). Cattle acts as definitive host for Liver fluke.

2. Intermediate Host

The host which harbors the parasite during its immature or asexual stages is called **intermediate host**. Example: Mosquitoes as intermediate hosts for *Plasmodium* species. Goat is an intermediate host of *Taenia multiceps*.

3. Carrier Host

It is the host in which the parasite remains viable without further development, later on the parasite host is ingested by the definitive host and parasite infection occurs. e.g. Earthworms, slugs, flies, snail, dragon flies crab etc.

4. Principal Host

It is that animal in which a particular parasite is most commonly found e.g. *Haemonchus contortus* in sheep, *Heterakis gallinarum* in fowl.

5. Reservoir Host

An animal that harbors a parasite and acts as a source of infection for other hosts, including humans. for example: Dogs as reservoir hosts for *Leishmania* parasites.

Exercise

Choose the correct answer from the given alternatives.

1. Which of the following is an example of an ecto-parasite?
 - a. *Fasciola hepatica*
 - b. *Taenia solium*
 - c. Flea
 - d. *Toxoplasma gondii*
2. A facultative parasite is one that.....
 - a. Always lives on or in a host.
 - b. Can survive independently or as a parasite.
 - c. Depends entirely on the host for survival.
 - d. Is only parasitic in its larval stage.
3. Which of the following parasites is classified as an endo-parasite?
 - a. Tick
 - b. Flea
 - c. *Liver fluke*
 - d. *Mites*
4. A parasite that requires two or more hosts to complete its life cycle is known as.....
 - a. Monoxenous
 - b. Heteroxenous
 - c. Facultative
 - d. Obligate
5. The term "definitive host" refers to.....
 - a. The host where the parasite lives as an immature stage.
 - b. The host where the parasite reproduces sexually.
 - c. A host that transports the parasite without development.
 - d. An accidental host.
6. What is the role of an intermediate host in the life cycle of a parasite?
 - a. It provides shelter to the adult stage of the parasite.
 - b. It harbors the parasite during its larval or asexual stage.

- c. It serves as a dead-end host for the parasite.
- d. It harbors the parasite without affecting its development.

7. Which type of host provides a habitat for the adult parasite and supports its sexual reproduction?

- a. Reservoir host
- b. Paratenic host
- c. Definitive host
- d. Intermediate host

8. **Which type of parasite can live independently or as a parasite?**

- a. Obligate parasite
- b. Facultative parasite
- c. Endoparasite
- d. Ectoparasite

9. **What is an endo-parasite?**

- a. A parasite that lives on the surface of the host
- b. A parasite that lives inside the body of the host
- c. A parasite that does not need a host
- d. A parasite that causes no harm to the host

10. What type of parasite is ticks ?

- a. External parasite
- b. Internal parasite
- c. Both
- d. None of above

Write short answer to the following questions.

1. Define parasite and parasitology
2. Define external parasite with examples.
3. Define internal parasite with examples.
4. Define host and explain its type.
5. Enlist the name of any four external parasite.
6. Enlist the name of any four internal parasite.

Project work

1. Make a list of common external parasites affecting farm animals (e.g., cattle, sheep, goats, and poultry).
2. Choose at least three common external parasites of farm animals (e.g., ticks, fleas, lice). Study and describe their morphological structures.

2.1 Introduction and Types of External Parasites, General Symptoms and Treatment of Lice, Ticks, Mite, Leech and Fleas

External parasites are organisms that live on the surface of a host animal, feeding on its blood, tissue, or skin secretions. These parasites can cause irritation, reduced productivity, disease transmission, and secondary infections in livestock, pets, and wildlife. Control and treatment of these parasites are essential for animal health, welfare, and economic productivity.

Types of External Parasites

1. **Lice:** Small, wingless insects that live on the skin and hair is called lice . It isDivided into two types:
 - **Biting lice** (Mallophaga): Feed on skin debris and secretions.
 - **Sucking lice** (Anoplura): Feed on blood.
2. **Ticks:** Blood-feeding arachnids that attach to the host's skin are called ticks. Types include:
 - **Hard ticks** (Ixodidae): Have a tough outer shell.
 - **Soft ticks** (Argasidae): Lack a hard shell.
3. **Mites:** Microscopic arachnids that burrow into the skin or live on the surface. Examples of mites are;
 - **Sarcoptes scabiei:** Causes mange.
 - **Demodex spp.:** Lives in hair follicles.
4. **Leeches:**leeches are segmented worms with suction cups, often found in aquatic environments. They attach to the skin and feed on blood.

5. **Fleas:** Small, wingless insects with powerful legs for jumping are called fleas. They feed on blood and can transmit diseases.

General Symptoms

1. Lice

- Itching and scratching.
- Hair loss or matting.
- Pale gums (in severe infestations due to blood loss).
- Restlessness and reduced productivity.

2. Ticks

- Visible ticks on the skin.
- Irritation and swelling at attachment sites.
- Anemia and weakness in severe cases.
- Disease symptoms (e.g., fever in tick-borne illnesses like Babesiosis).

3. Mites

- Severe itching and scratching.
- Crusting and scaling of the skin.
- Hair loss and thickened skin.
- Skin redness and secondary infections.

4. Leeches

- Visible leeches attached to the skin.
- Blood loss at feeding sites.
- Persistent bleeding due to anticoagulants in saliva.

5. Fleas

- Intense itching and scratching.
- Red or irritated skin.

- Hair loss and skin infections.
- Presence of flea dirt (black specks) in the fur.

General Treatment

1. Lice

- Insecticidal sprays, dips, or powders containing pyrethroids or organophosphates.
- Ivermectin injections for sucking lice.
- Proper grooming and isolation of affected animals.

2. Ticks

- Manual removal using tweezers (avoid crushing the tick).
- Application of acaricides (e.g., permethrin, amitraz).
- Tick collars for pets and dipping for livestock.

3. Mites

- Topical acaricides (e.g., lime sulfur dips, amitraz).
- Ivermectin or moxidectin injections for burrowing mites.
- Regular cleaning of bedding and environment.

4. Leeches

- Manual removal using salt or alcohol (do not pull directly to avoid leaving mouthparts).
- Disinfection of attachment sites.
- Prevention through avoiding infested water bodies.

5. Fleas

- Flea shampoos, sprays, or spot-on treatments (e.g., fipronil, imidacloprid).
- Oral medications (e.g., nitenpyram, afoxolaner).
- Vacuuming and cleaning the environment to remove eggs and larvae.

Prevention and Control

- Regular grooming and inspection of animals.
- Maintain hygiene in housing and bedding.
- Use preventive treatments such as medicated collars or sprays.
- Rotate pastures and avoid overstocking to reduce parasite load.
- Control wildlife access to prevent parasite spread.

2.2 Important Diseases Caused by External Parasites

Diseases Caused by Lice

1. Pediculosis

- Caused by infestation with lice.
- Symptoms: Intense itching, anemia (from sucking lice), poor coat condition, and secondary bacterial infections.
- Affects livestock, pets, and humans.

Diseases caused by Ticks

1. Babesiosis

- Caused by protozoa (*Babesia* spp.) transmitted by ticks (e.g., *Rhipicephalus* and *Ixodes*).
- Symptoms: Fever, anemia, jaundice, and death in severe cases.

2. Theileriosis:

- Caused by protozoa (*Theileria* spp.) transmitted by ticks.
- Symptoms: Lymph node swelling, fever, anemia, and reduced productivity.

3. Ehrlichiosis

- Caused by *Ehrlichia* bacteria transmitted by ticks (e.g., *Rhipicephalus sanguineus*).
- Symptoms: Fever, lethargy, anorexia, and bleeding disorders.

4. Anaplasmosis

- Caused by *Anaplasma* bacteria.
- Symptoms: Fever, severe anemia, and weight loss.

5. Tick Paralysis

- Caused by neurotoxic saliva of certain tick species.
- Symptoms: Weakness, paralysis, and potentially death if respiratory muscles are affected.

6. Lyme Disease

- Caused by *Borrelia burgdorferi* bacteria transmitted by *Ixodes* ticks.
- Symptoms: Lameness, fever, joint swelling, and kidney problems (in severe cases).

Diseases Caused by Mites

1. Scabies (Sarcoptic Mange)

- Caused by *Sarcoptes scabiei* mites that burrow into the skin.
- Symptoms: Severe itching, thickened skin, hair loss, and crusting.

2. Demodicosis (Demodectic Mange)

- caused by *Demodex* mites in hair follicles.
- symptoms: Hair loss, skin redness, and secondary infections.

3. Psoroptic Mange (Sheep Scab)

- Caused by *Psoroptes* mites.
- Symptoms: Intense itching, wool loss, and open wounds.

4. Otodectic Mange

- caused by *Otodectes cynotis* (ear mites).
- symptoms: Ear itching, dark discharge, and head shaking.

Diseases caused by leeches

1. Anemia and Secondary Infections

- Result from blood loss due to heavy leech infestations.
- Symptoms: Weakness, pale mucous membranes, and susceptibility to infections.

2. Transmission of Pathogens

- Leeches can transmit bacteria like *Aeromonas* species, leading to localized infections.

Diseases Caused by Fleas

1. Flea Allergy Dermatitis (FAD)

- caused by an allergic reaction to flea saliva.
- symptoms: Severe itching, redness, hair loss, and skin infections.

2. Plague

- caused by *Yersinia pestis* bacteria transmitted by fleas.
- symptoms (in animals and humans): Fever, swollen lymph nodes, and respiratory distress.

3. Myxomatosis

- viral disease in rabbits transmitted by fleas.
- symptoms: Swelling, skin lesions, and high mortality.

4. Tapeworm Infestation

- fleas act as intermediate hosts for *Dipylidium caninum* tapeworms.
- symptoms: Anal itching, segments of tapeworms in feces.

Exercise

Choose the correct answer from the given alternatives.

1. External parasites live.....
 - a. Inside the host's body
 - b. On the surface of the host's body
 - c. In water only
 - d. None of the above
2. Which of the following is a sucking parasite?
 - a. Demodex
 - b. Biting lice
 - c. Sucking lice
 - d. Sarcoptes
3. Hard ticks are classified under.....
 - a. Insects
 - b. Arachnids
 - c. Nematodes
 - d. Protozoa
4. Which parasite is commonly found in aquatic environments?
 - a. Mites
 - b. Leeches
 - c. Fleas
 - d. Ticks
5. Intense itching and presence of black specks in the fur indicate an infestation of.....
 - a. Ticks
 - b. Fleas
 - c. Lice
 - d. Mites
6. Hair loss, thickened skin, and crusting are typical symptoms of.....
 - a. Scabies
 - b. Anaplasmosis
 - c. Tick paralysis
 - d. Pediculosis
7. Persistent bleeding at the attachment site is most commonly caused by.....
 - a. Ticks
 - b. Leeches
 - c. Mites
 - d. Fleas

8. Which chemical is commonly used to treat mange caused by mites?
 - a. Acaricides
 - b. Antibiotics
 - c. Anthelmintics
 - d. Antifungals
9. Which tick-borne disease causes anemia and jaundice?
 - a. Babesiosis
 - b. Theileriosis
 - c. Scabies
 - d. Demodicosis
10. Fleas are intermediate hosts for.....
 - a. Babesia
 - b. Dipylidium caninum
 - c. Ehrlichia
 - d. Sarcoptes scabiei
11. The causative agent of scabies is.....
 - a. Demodex mites
 - b. Sarcoptes scabiei
 - c. Otodectes cynotis
 - d. Ixodes ticks

Write short answer to the following questions.

1. Enlist any four diseases caused by external parasite. Write about the treatment of process of the disease caused by lice.
2. Define external parasite. What are the symptoms of flea infestation?
3. Write general symptoms of ticks and mites.

Write long answer to the following questions.

1. Describe about prevention and control of external parasites in animal farm.

Project work

1. List and describe five common external parasites found in farm animals. Mention the species they affect and the diseases they cause.also report in the class.

3.1 Introduction to Common Helminth Parasites of Ruminants and Non-ruminants.

Helminth parasites are worms that inhabit and infect the gastrointestinal tract or other organs of animals. They are a major cause of economic losses in livestock farming, leading to reduced productivity, weight loss, and diseases. These parasites are classified into three major groups: nematodes, trematodes, and cestodes. Both ruminants (e.g., cattle, sheep, goats) and non-ruminants (e.g., pigs, horses, poultry) are affected by helminths.

1. Nematodes (*roundworms*)

Nematodes are cylindrical, unsegmented worms with a complete digestive system.

Common Nematodes in Ruminants

1. **Haemonchus Contortus (Barber pole worm)**

- affects sheep, goats, and cattle.
- lives in the abomasum and sucks blood, causing anemia and bottle jaw.

2. **Ostertagia spp. (Brown stomach worm)**

- causes ostertagiasis in cattle.
- disrupts digestion by damaging the stomach lining.

3. **Trichostrongylus spp.**

- affects sheep, goats, and cattle.
- found in the small intestine, leading to diarrhea and weight loss.

Common Nematodes in Non-ruminants

1. **Ascaris Suum (pig roundworm)**

- affects pigs.
- causes liver and lung damage during larval migration.

2. **Strongylus spp. (large strongyles in horses)**

- damages blood vessels and causes colic.

3. **Ascaridia Galli (roundworm of poultry)**

- found in the intestines of chickens, causing growth retardation.

2. **Trematodes (Flukes)**

Trematodes are flat, leaf-shaped worms that lack a complete digestive system and are usually hermaphroditic.

Common Trematodes in Ruminants

1. **Fasciola hepatica (liver fluke)**

- Affects cattle and sheep.
- Lives in the bile ducts and causes fascioliasis, leading to liver damage and reduced milk production.

2. **Paramphistomum spp. (rumen fluke)**

- found in the rumen and reticulum of cattle and sheep.
- causes anorexia and diarrhea during the immature stages.

Common Trematodes in Non-ruminants

1. **Schistosoma spp. (Blood fluke)**

- affects pigs and horses.
- causes schistosomiasis, which leads to liver and intestinal damage.

3. **Cestodes (tapeworms)**

Cestodes are segmented, ribbon-like worms that live in the intestines and absorb

nutrients directly through their body surface.

Common Cestodes in Ruminants

1. **Moniezia spp.**

- affects sheep, goats, and cattle.
- found in the small intestine, causing mild digestive disturbances.

2. **Taenia Saginata (beef tapeworm)**

- larval stage infects cattle (cysticercosis), while adult worms infect humans.

Common Cestodes in Non-ruminants

1. **Taenia Solium (pork tapeworm)**

- larval stage infects pigs (cysticercosis), while adult worms infect humans.

2. **Hymenolepis spp. (tapeworms in poultry)**

- found in the intestines of birds, causing reduced growth and productivity.

3.2 Effects of Helminths on Host

Helminth infections have significant negative impacts on the health and productivity of farm animals. These effects can range from subclinical losses to severe diseases and death. The severity of the impact depends on the type of helminth, the intensity of infection, the host species, and the animal's overall health and nutritional status.

1. Nutritional Impact

• Reduced Nutrient Absorption

- Helminths, particularly intestinal parasites like *Haemonchus* and *Moniezia spp.*, compete with the host for nutrients.
- Damage to the gastrointestinal lining by worms like *Trichostrongylus spp.* decreases the efficiency of nutrient absorption.

- **Protein Loss**

- Blood-feeding helminths such as *Haemonchus contortus* and liver flukes (*Fasciola hepatica*) cause anemia and hypoproteinemia due to blood and plasma loss.

- **Weight Loss and Poor Growth**

- Infected animals often show reduced feed efficiency, leading to poor weight gain and growth retardation.

2. Organ Damage

- **Liver Damage**

- *Fasciola hepatica* (liver fluke) causes liver fibrosis, bile duct inflammation, and hepatic insufficiency, leading to "liver rot."
- Migration of larvae through the liver (e.g., *Ascaris suum*) can cause hemorrhages and liver cirrhosis.

- **Lung Damage**

- Migratory nematodes like *Dictyocaulus viviparus* (lungworm) cause pneumonia, coughing, and difficulty breathing (verminous bronchitis).

- **Gastrointestinal Damage**

- Helminths like *Ostertagia spp.* destroy the stomach lining, leading to impaired digestion and diarrhea.

3. Blood Loss and Anemia

- Blood-sucking helminths such as *Haemonchus contortus* in sheep and goats, and *Fasciola hepatica* in cattle, cause severe anemia and hypoproteinemia.
- Signs include pale mucous membranes, lethargy, and edema ("bottle jaw").

4. Reduced Reproductive Performance

- Helminths affect fertility and reproductive efficiency by:
 - causing poor body condition, delaying puberty, and reducing conception rates.
 - lowering milk yield in lactating animals due to nutrient competition and systemic stress.

5. Immune Suppression

- Chronic helminth infections suppress the host's immune system, making animals more susceptible to secondary infections (e.g., bacterial or viral diseases).
- Co-infections with other pathogens are common in helminth-infected animals.

6. Economic Losses

- **Reduced productivity:** Lower meat, milk, and wool production due to poor health.
- **Mortality:** High worm burdens can cause death, particularly in young animals.
- **Increased costs:** Expenses related to anthelmintics, veterinary care, and labor for management.

7. Behavioral Changes

- Helminth-infected animals often exhibit lethargy, reduced grazing activity, and poor social interaction, which further impacts productivity.

3.3.1 Liver Fluke Disease

Liver fluke disease, or fascioliasis, is caused by parasitic trematodes, primarily *Fasciola hepatica* and *Fasciola gigantica*. These flukes infect the livers of various domestic and wild animals, causing economic losses due to liver damage, decreased productivity, and, in severe cases, death. The disease is also zoonotic,

posing risks to humans.

Morphology

- **Adult Fluke**
 - leaf-shaped, dorsoventrally flattened.
 - *F. hepatica*: 20–30 mm long and 5–12 mm wide.
 - *F. gigantica*: Larger, up to 75 mm long.
 - anterior end has a conical projection with a sucker.
- **Eggs**
 - Oval, yellow-brown, operculated (lid-like structure).
 - Size: 130–150 μm .

Hosts

Definitive Hosts: Cattle, sheep, goats, buffaloes, pigs, humans (accidental host) are definitive host. **Intermediate Host:** Freshwater snails .

Lifecycle

- a. **Eggs:** Passed in the feces of the definitive host.
- b. **Miracidium:** Hatches in water and infects snails.
- c. **Sporocyst and rediae:** Develop within the snail.
- d. **Cercariae:** Released from the snail into water.
- e. **Metacercariae:** Encyst on vegetation; infective stage.
- f. **Ingestion:** Definitive host consumes contaminated vegetation.
- g. **Immature Flukes:** Migrate through the intestinal wall, liver parenchyma, and bile ducts, maturing into adults.

Lifecycle duration: ~12–16 weeks.

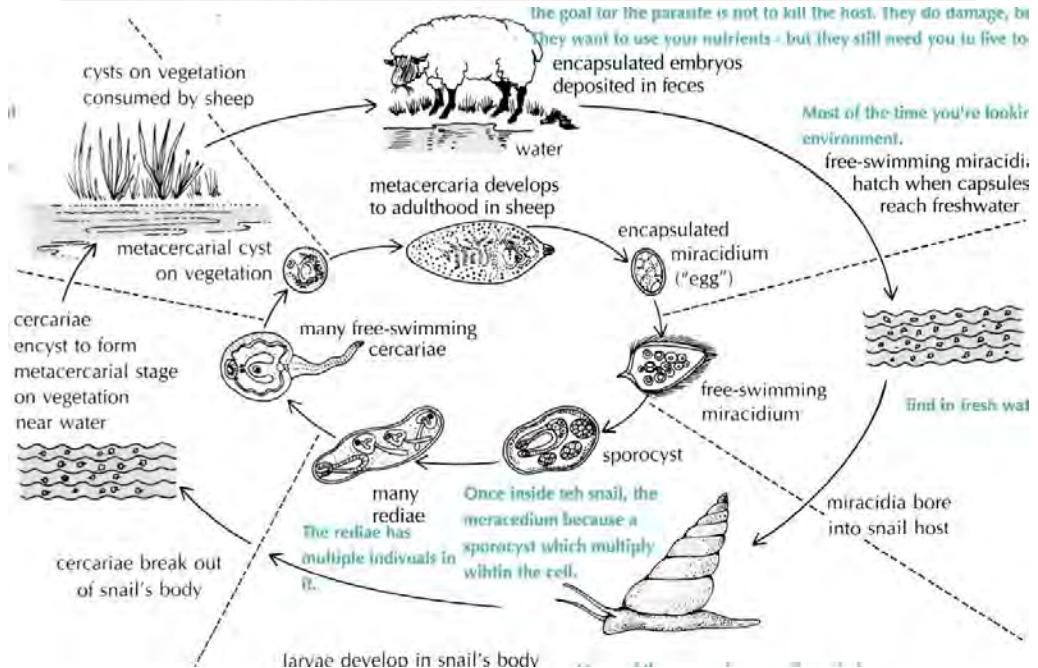
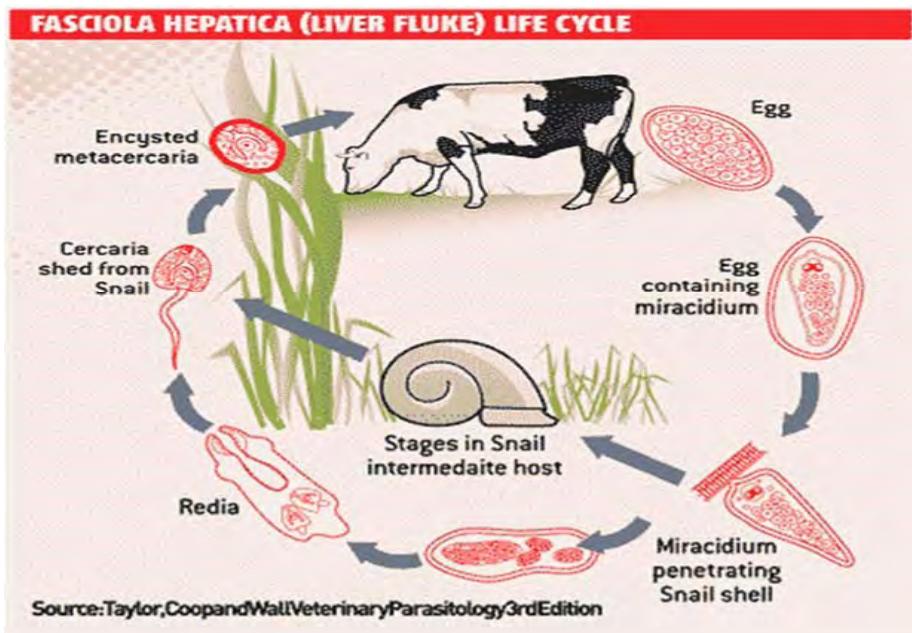


Fig1-Life Cycle of *Fasciola hepatica*

5. Clinical Signs

Acute Fascioliasis

- occurs due to migration of immature flukes.
- Signs:
 - sudden death (in severe cases);
 - abdominal pain, anemia, jaundice;
 - enlarged, painful liver;

Chronic Fascioliasis

- caused by adult flukes in bile ducts;
- Signs
 - weight loss, reduced appetite;
 - anemia, pale mucous membranes;
 - bottle jaw (submandibular edema);
 - reduced milk production;

6. Diagnosis

- **Clinical signs:** based on history of grazing in wet areas and symptoms like anemia and bottle jaw.
- **Fecal examination:** detection of eggs via sedimentation technique.
- **Serological tests:** ELISA to detect antibodies.
- **Ultrasound or liver biopsy:** assess liver damage.
- **Post-mortem examination:** presence of flukes in bile ducts or liver tissue.

7. Treatment

- **Anthelmintics**
 - *Triclabendazole*: effective against both immature and adult flukes;
 - *Albendazole* and *Clorsulon*: Effective against adult flukes.

- **Supportive Therapy**
 - Iron supplementation for anemia.
 - High-energy diets to aid recovery.

8. Prevention and Control

Preventing Infection

1. Snail Control

- Drain marshy areas or ponds where snails thrive.
- Use molluscicides (e.g., copper sulfate) where appropriate.

2. Grazing Management

- avoid grazing animals in wet or marshy pastures during high-risk periods;

3. Water Sources

- Provide clean water, avoiding natural water bodies that may harbor metacercariae.

Herd health Programs

- Regular deworming based on risk assessment and fecal egg count monitoring;
- Vaccination (under development for liver flukes).

3.3.2 Round Worm of Ruminants and Non-ruminants

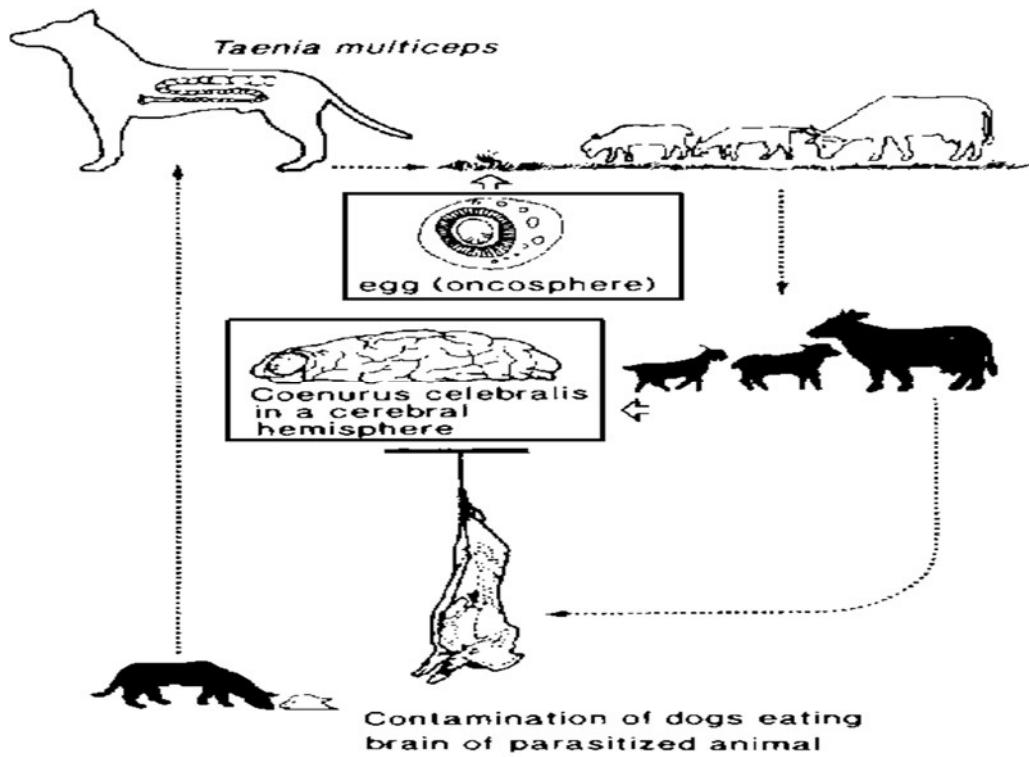
Host	Round worm	Scientific name	Location in host	Key features
Ruminants	Brown stomach worm	<i>Ostertagia ostertagi</i>	Abomasum	Causes severe diarrhea and weight loss
	Barber's pole worm	<i>Haemonchus contortus</i>	Abomasum	Blood-sucking; anemia, bottle jaw
	Threadworm	<i>Trichostrongylus</i> spp.	Small intestine	Diarrhea, weight loss
	Lungworm	<i>Dictyocaulus viviparus</i>	Lungs	Respiratory distress, coughing
	Nodular worm	<i>Oesophagostomum</i> spp.	Large intestine	Nodules in intestinal wall, diarrhea
	Whipworm	<i>Trichuris ovis</i>	Large intestine and cecum	Causes colitis and weight loss
	Large roundworm (swine)	<i>Ascaris suum</i>	Small intestine	Stunted growth, liver damage ("milk spots")
Non-Ruminants	Large roundworm (equines)	<i>Parascaris equorum</i>	Small intestine	Colic, poor growth, intestinal blockage
	Large strongyles (equines)	<i>Strongylus vulgaris</i>	Large intestine	Arteritis, colic, thromboembolism
	Whipworm (dogs)	<i>Trichuris vulpis</i>	Large intestine and cecum	Chronic diarrhea and weight loss
	Roundworm (dogs and cats)	<i>Toxocara canis, T. cati</i>	Small intestine	Potbelly, poor growth, zoonotic risk

3.3.3 Gid

Gid also known as Coenurosis is a disease of the central nervous system in goats and sheep. Coenurosis is infection by the metacestode larval stage (coenurus) of *Taenia multiceps* which infests the small intestine of carnivores. The definitive hosts for *Taenia multiceps* are members of the family Canidae. In 80%–90% of cases, the cyst is located in one cerebral hemisphere, whilst in 5%–10% of cases, it is localised in the cerebellum; rarely it involves two sites in the brain of the affected animal.

Coenurus cerebralis, the larval form of *Taenia multiceps* which is seen in the small intestines of carnivores. Infection occurs as a result of the oral intake of eggs spreading via faeces of those animals by intermediate hosts. The disease is known as gid or sturdy which primarily localises in the central nervous system of sheep and goats mostly, but can also be seen in camels, deer, pigs, horses, however, rarely in cattle and humans. Most of the cysts are located in the cerebral hemispheres and spinal cord, while rarely invading the subcutaneous and intramuscular tissues along with other organs symptoms vary depending on the cyst's location, size and compression. *Coenurus cerebralis* initially causes purulent meningoencephalitis, later as the cyst grows, it leads to central nervous system symptoms resulting in. Most of the characteristic,s clinical findings are observed 2-8 months after the intake of pathogen. Infected animals manifest circling, head tilt towards the side of the cyst location, in coordinated and uncontrolled movements, ataxia, failure to hold the head straight, blindness, teeth grinding, salivation, paresis, convulsions.

Life cycle: Eggs expelled with dog faeces are ingested by the intermediate host (sheep). The larvae hatch in the intestine and pass with the blood stream towards different organs. The larvae which reach the brain and spinal cord grow to the *coenurid* stage. *Coenurus cerebralis* will further mature in the brain and spinal cord.



*Fig2: Life cycle of *Taenia multiceps**

Clinical Findings

During migration of larval stage

- Blindness
- Muscular tremor and incoordination
- Excitability and collapse
- Infection with the fully developed larval stage
- Salivation
- Wild expressions
- Frenzied running and convulsion
- Deviation of eye and head
- Loss of function

- j. Dullness
- k. Incomplete mastication
- l. Head pressing
- m. Incomplete paralysis and, in spinal cord involvement, inability to rise

Postmortem Findings

- a. Thin walled cyst in the brain.
- b. Lesion in the lumbar region and rarely, in the cervical area of the spine

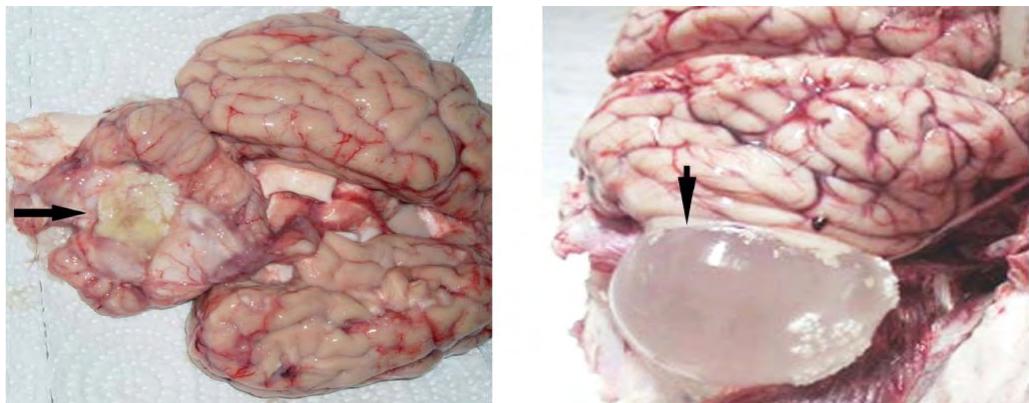


Fig 3: Coenurus cerebralis. Thin walled cyst in the brain

Diagnostic Information

Infected dogs pass *taenias*-type eggs in their faeces. Sheep at the chronic stage of the infection may show circular movements, jerky movements or staggering gait.

Treatment

For adult tapeworm a number of effective drugs are available including praziquantel, mebendazole, fenbendazole, nitroscanate, and dichlorophen.

Surgical removal is possible if the cyst is situated on the brain surface.

Prevention and Control

For Definitive Hosts

- regular deworming with **praziquantel** every 4–6 weeks to eliminate

- adult tapeworms;
- proper disposal of sheep/goat carcasses to prevent access by dogs;

For Intermediate Hosts

- prevent grazing on contaminated pastures.
- ensure clean feed and water sources.
- reduce dog access to grazing areas;
- **Public awareness:** Educate farmers about the lifecycle and transmission to encourage preventive practices.

3.3.4 Hydatidosis

Hydatidosis is a zoonotic parasitic disease caused by the **larval stage (hydatid cyst)** of the tapeworm *Echinococcus granulosus*. It is an economically and medically significant condition affecting livestock, wildlife, and humans. The disease results in cyst formation, primarily in the liver and lungs of intermediate hosts, leading to organ dysfunction.

Morphology

- **Adult Tapeworm**
 - found in the small intestine of canids.
 - tiny, measuring **3–7 mm** in length.
 - scolex has **four suckers** and a **double crown of hooks**.
 - consists of **three proglottids**: immature, mature, and gravid.
- **Hydatid Cyst**
 - spherical, fluid-filled cysts with an **outer laminated layer** and an **inner germinal layer**.
 - cysts contain **brood capsules** with protoscolices ("hydatid sand");
 - cysts may grow up to **20 cm or more** and can contain several liters of fluid.

Hosts

- **Definitive hosts:** Canids (e.g., dogs, foxes) harbor adult tapeworms.
- **Intermediate hosts:** Livestock (e.g., sheep, cattle, goats) and wild ungulates;
- **Accidental hosts:** Humans, through ingestion of eggs, develop hydatid cysts in organs;

4. Lifecycle

1. **Adult tapeworms** in the small intestine of canids release eggs into the environment via feces.
2. Intermediate hosts ingest eggs while grazing or consuming contaminated food/water.
3. **Eggs hatch** in the intestine, releasing oncospheres that penetrate the intestinal wall.
4. Oncospheres travel via blood to organs (primarily the liver and lungs), where they develop into hydatid cysts over months to years.
5. Canids ingest cysts when feeding on infected offal (organ meat). Protoscolices develop into adult tapeworms in the canid's intestine, completing the lifecycle.

Clinical Signs

- Usually asymptomatic unless cysts are large or numerous;
- Reduced productivity (e.g., weight gain, milk production);
- Organ dysfunction if cysts affect vital organs (e.g., liver, lungs);

Diagnosis

- Post-mortem inspection: Hydatid cysts in organs like the liver and lungs;
- Ultrasound or imaging techniques in live animals (rare);

Treatment

- No effective treatment; management focuses on prevention and control;

Prevention and Control

For Definitive Hosts (canids)

- Regular deworming with **praziquantel** every 4–6 weeks;
- Prevent access to raw offal from slaughtered animals;
- Proper disposal of infected organs (incineration or burial);

For intermediate Hosts (livestock)

- Minimize contact with infected canids.
- Improve sanitation and management of grazing areas.

3.3.5 Dog Tapeworm

Dipylidium caninum is a common tapeworm that infects dogs, cats, and occasionally humans (especially children). It is transmitted through ingestion of infected fleas or lice. The adult tapeworm lives in the small intestine of the definitive host and causes mild clinical signs.

Morphology

- Measures **15–70 cm** in length.
- The body consists of **proglottids** (segments) that resemble cucumber seeds when fresh.
- The scolex (head) has **four suckers** and a retractable rostellum armed with hooks.
- Each proglottid contains **two genital pores** (one on each side), a characteristic feature of *Dipylidium caninum*.

Hosts

- **Definitive hosts:** Dogs, cats, and occasionally humans;
- **Intermediate hosts:** Fleas (*Ctenocephalides felis*, *C. canis*) and biting lice (*Trichodectes canis*);

Lifecycle

- **Adult tapeworms** in the small intestine of definitive hosts produce gravid proglottids, which are passed in feces or crawl out of the anus.
- Gravid proglottids release **egg packets** into the environment.
- **Intermediate hosts** (flea larvae or lice) ingest egg packets while feeding on organic debris.
- Eggs develop into **cysticercoid larvae** inside the flea or louse.
- Definitive hosts become infected by ingesting infected fleas or lice during grooming or biting.
- Cysticercoid larvae develop into adult tapeworms in the intestine of the definitive host, completing the lifecycle.

Clinical Signs

- **Mild infections:** Often asymptomatic.
- **Moderate to Heavy Infections**
 - Scooting or rubbing the anal region (due to irritation from proglottids).
 - Mild gastrointestinal upset (vomiting, diarrhea).
 - Poor coat condition or weight loss (in severe cases).
 - Visible proglottids in feces or around the anus (look like rice grains);

Diagnosis

- **Fecal Examination**
 - Identification of egg packets or proglottids in feces.
 - Proglottids may be found crawling around the perianal area.
- **Visualization:** owners often notice rice-like proglottids on bedding, fur, or feces.

Treatment

- **Anthelmintics**

- Praziquantel: highly effective at killing adult tapeworms.
- Epsiprantel: another effective option.
- Combination dewormers (e.g., praziquantel with pyrantel and febantel) for broad-spectrum activity.
- **Follow-up:** Treat concurrent flea or lice infestations to prevent reinfection.

Prevention and Control

Flea and Lice Control

- Use of **flea control products** (e.g., fipronil, imidacloprid, or fluralaner) on pets and in the environment;
- Regular grooming and bathing to remove lice and fleas;
- Treat all animals in the household to prevent cross-infestation;
- Maintain clean bedding and surroundings to reduce flea populations;

Deworming

- Regular administration of dewormers, especially in areas with high flea burdens.
- Deworm puppies and kittens frequently as they are more prone to infection.

Hygiene

- Educate pet owners about the importance of flea control.
- Discourage close contact between children and untreated pets to minimize zoonotic transmission.

3.3.6 Pork Tapeworm

- *Taenia solium* is a cestode (tapeworm) that causes **taeniasis** in its definitive hosts (humans) and **cysticercosis** in its intermediate hosts (pigs and humans).
- It is primarily found in regions with poor sanitation and where pigs are raised in close proximity to humans.

- Human cysticercosis, caused by the larval stage, is a serious condition that can affect the central nervous system (neurocysticercosis).

Morphology

- **Adult Tapeworm**
 - measures **2–8 meters** in length;
 - composed of a scolex, neck, and multiple proglottids;
 - the scolex has **four suckers** and a **double crown of hooks** for attachment;
 - gravid proglottids (posterior segments) are longer than wide and contain **15–30 lateral branches** in the uterus;
- **Eggs**
 - spherical, brown, with a thick striated shell, and contain an oncosphere (hexacanth embryo).
- **Cysticercus Larva**
 - fluid-filled, bladder-like structure containing an invaginated scolex.

Hosts

- **Definitive host:** humans, where adult tapeworms reside in the small intestine.
- **Intermediate host:** pigs, and occasionally humans, harbor cysticerci in their tissues.

Lifecycle

- **humans ingest undercooked pork** containing viable cysticerci;
- cysticerci develop into adult tapeworms in the small intestine, where they attach using their scolex;
- gravid proglottids release **eggs** into the environment through human feces;
- **pigs ingest eggs** via contaminated feed or water;

- eggs hatch in the pig's intestine, releasing oncospheres that penetrate the intestinal wall and migrate via blood to muscles, brain, and other tissues, forming cysticerci;
- humans can also develop **cysticercosis** if they ingest eggs directly (e.g., via contaminated food, water, or poor hygiene);

5. Clinical Signs

Taeniasis (intestinal infection in humans)

- often asymptomatic.
- mild symptoms: Abdominal discomfort, diarrhea, nausea, and weight loss;
- passage of proglottids in feces or crawling out of the anus;

Cysticercosis (larval infection in humans)

- symptoms depend on the location of cysticerci:
 - **Neurocysticercosis**: headache, seizures, hydrocephalus, and neurological deficits;
 - **Ocular cysticercosis**: vision disturbances or blindness;
 - **Muscular cysticercosis**: asymptomatic or swelling and pain;

Diagnosis

For Taeniasis

- **Microscopy**: detection of eggs or gravid proglottids in feces.
- **Molecular tests**: PCR for species identification;
- **Stool examination**: concentration techniques improve sensitivity;

For Cysticercosis

- **Imaging**: CT or MRI to detect cysts in tissues (e.g., brain, muscles).
- **Serology**: ELISA or Western blot for specific antibodies or antigens.
- **Biopsy**: identification of cysticerci in subcutaneous nodules.

Treatment

Taeniasis

- **Praziquantel** (5–10 mg/kg): First-line treatment.
- **Niclosamide** (2 g single dose for adults): Alternative drug.

Cysticercosis

- **Albendazole** (15 mg/kg/day for 8–30 days) or **Praziquantel** (50–100 mg/kg/day for 15–30 days).
- **Anti-inflammatory drugs** (e.g., corticosteroids): To reduce inflammation from dying cysticerci.
- **Antiepileptics**: for managing seizures in neurocysticercosis.
- **Surgical removal**: for cysts causing severe complications (e.g., hydrocephalus)

Prevention and Control

Public Health Measures

- Educate communities about the risks of eating undercooked pork and poor hygiene.
- Promote thorough cooking of pork (internal temperature of at least 70°C).
- Improve sanitation to prevent fecal contamination of the environment.

Veterinary Measures

- Routine deworming of pigs with **oxfendazole**.
- Prevent pigs from accessing human feces.
- Meat inspection and proper disposal of infected carcasses.

Personal Hygiene

- Wash hands thoroughly with soap and water, especially after using the toilet and before handling food.
- Ensure safe water supply to prevent contamination.

Exercise

Choose the correct answer from the given alternatives.

1. Which of the following is a common helminth parasite of ruminants?
 - a. *Fasciola hepatica*
 - b. *Taenia solium*
 - c. *Dipylidium caninum*
 - d. *Echinococcus granulosus*
2. The primary host for *Echinococcus granulosus* is.....
 - a. Humans
 - b. Pigs
 - c. Dogs
 - d. Cattle
3. Helminths are typically transmitted through.....
 - a. Airborne droplets
 - b. Contaminated food, water, or grazing areas
 - c. Direct physical contact with infected animals
 - d. Biting insects
4. Chronic helminth infections in livestock commonly cause.....
 - a. Weight gain
 - b. Increased productivity
 - c. Anemia and poor growth
 - d. Neurological disorders
5. Which of the following is NOT a common effect of helminth infections?
 - a. Immune suppression
 - b. Organ damage
 - c. Improved digestion
 - d. Nutrient depletion
6. Liver fluke disease often results in.....
 - a. Gastrointestinal blockage
 - b. Liver fibrosis and reduced productivity
 - c. Respiratory distress
 - d. Brain lesions

7. The causative agent of liver fluke disease in cattle is.....

- Fasciola hepatica*
- Taenia saginata*
- Ancylostoma caninum*
- Ascaris suum*

8. The intermediate host of *Fasciola hepatica* is.....

- Earthworms
- Snails
- Fleas
- Mites

9. The preferred treatment for liver fluke infections is.....

- Praziquantel
- Triclabendazole
- Albendazole
- Niclosamide

10. Which roundworm is commonly found in the intestines of ruminants?

- Haemonchus contortus*
- Taenia solium*
- Fasciola gigantica*
- Echinococcus granulosus*

11. In roundworms, the infective stage is typically.....

- Egg
- Adult
- Larva
- Proglottid

12. Gid is caused by the larval stage of.....

- Taenia multiceps*
- Taenia saginata*
- Fasciola hepatica*
- Echinococcus granulosus*

13. The definitive host of *Taenia multiceps* is.....

- Cattle
- Sheep
- Dogs
- Humans

14. Hydatid cysts in intermediate hosts are caused by.....

- Dipylidium caninum*
- Echinococcus granulosus*
- Taenia solium*
- Fasciola hepatica*

15. Humans develop hydatidosis by.....
 - a. Ingesting undercooked meat
 - b. Ingesting eggs from contaminated food or water
 - c. Being bitten by infected fleas
 - d. Direct contact with infected animals
16. The intermediate host of *Dipylidium caninum* is.....
 - a. Snails
 - b. Fleas or lice
 - c. Pigs
 - d. Sheep
17. The most effective treatment for dog tapeworm is.....
 - a. Albendazole
 - b. Praziquantel
 - c. Niclosamide
 - d. Triclabendazole
18. The adult form of *Taenia solium* resides in the.....
 - a. Liver
 - b. Brain
 - c. Small intestine
 - d. Lungs
19. Neurocysticercosis in humans is caused by.....
 - a. Adult *Taenia solium*
 - b. *Taenia saginata* cysticerci
 - c. *Taenia solium* larvae (cysticerci)
 - d. *Dipylidium caninum* larvae
20. Preventing pork tapeworm infections involves.....
 - a. Regular deworming of pigs
 - b. Cooking pork thoroughly
 - c. Improving sanitation
 - d. All of the above

Write short answer to the following questions.

1. Describe the lifecycle of Liver fluke.
2. Write clinical signs and methods of diagnosis of GID.
3. Write etiology of hydatid cyst in goat. How can Hydatidosis be prevented?
4. What is the intermediate host of *Taenia solium*. Describe the life cycle of *Taenia solium*.
5. Write clinical signs and prevention and control measures of Liver fluke.
6. Write short notes on effect of helminth parasite on host.

Write long answer to the following questions.

1. Describe the life cycle, clinical signs and prevention and control of *Fasciola hepatica*.
2. Describe the life cycle of *Taenia multiceps*. Write its clinical signs.
3. Write the name of dog tapeworm. Explain its life cycle and preventive measures.
4. Write the name of pork tape worm. Explain its life cycle and preventive measures.

Project work

1. How is examination of helminths fecal sample performed in lab?

4.1 Babesiosis

Babesiosis, also known as **Red Water Disease**, is a tick-borne protozoan disease affecting a wide range of domestic and wild animals, as well as humans. It is caused by the genus *Babesia* and is characterized by fever, anemia, hemoglobinuria (red-colored urine), and significant morbidity and mortality, especially in cattle.

Synonym

1. Piroplasmosis

Texas cattle fever (specific to *Babesia bigemina* in cattle)

Red water fever (due to hemoglobinuria caused by red blood cell destruction)

Tick fever

2. Etiology

- Causative agent: Protozoa of the genus *Babesia*.
- Common species in cattle:
 - *Babesia bovis*
 - *Babesia bigemina*
- Other affected species include dogs (*Babesia canis*, *Babesia gibsoni*), horses (*Babesia caballi*, *Theileria equi*), and humans (*Babesia microti*).

3. Mode of Transmission

1. Tick-borne Transmission

- *Babesia* parasites are transmitted primarily by ixodid (hard) ticks, such as *Rhipicephalus* and *Boophilus* species.
- Ticks become infected when they feed on the blood of infected animals and transmit the parasite during their next feeding.

2. Transovarial Transmission

- Female ticks pass the infection to their offspring via their eggs.

3. Mechanical Transmission

- Rarely, through contaminated needles or surgical instruments.

4. Blood Transfusion

- Direct transmission via infected blood in humans and animals.

4. Symptoms

Acute babesiosis

- High fever (up to 41°C or 105.8°F).
- Anemia due to the destruction of red blood cells (hemolysis).
- Jaundice (yellowing of mucous membranes).
- Hemoglobinuria (red or coffee-colored urine).
- Weakness, depression, and reduced appetite.
- Increased heart and respiratory rates.

Chronic Babesiosis

- Milder clinical signs such as intermittent fever and weight loss.

In severe cases

- Collapse and death within days if untreated.

5. Diagnosis

1. Microscopic Examination

- Stained blood smears (e.g., Giemsa stain) to detect *Babesia* in red blood cells.

2. Serological Tests

- Indirect fluorescent antibody test (IFAT).
- Enzyme-linked immunosorbent assay (ELISA).

3. Molecular Diagnosis

- PCR for species-specific identification.

4. Clinical Signs

- Hemoglobinuria, fever, and anemia provide presumptive diagnosis.

5. Post-mortem Findings

- Enlarged spleen, jaundice, and dark-colored kidneys due to hemoglobin deposition.

6. Treatment

1. Antiprotozoal Drugs

- *Imidocarb dipropionate* (2.5–3 mg/kg body weight subcutaneously or intramuscularly).
- *Diminazene aceturate* (3.5–5 mg/kg body weight intramuscularly).
- Combination therapies may be required in resistant cases.

2. Supportive Therapy

- Blood transfusion for severe anemia.
- Fluid therapy to correct dehydration and electrolyte imbalances.
- Anti-inflammatory drugs to reduce fever and inflammation.

3. Antibiotics

- Often administered to prevent secondary bacterial infections.

7. Prevention and Control

1. Tick Control

- Regular use of acaricides on livestock (e.g., permethrin, cypermethrin, or amitraz).
- Maintaining tick-free environments by pasture rotation and habitat management.

2. Vaccination

- In endemic areas, vaccines containing attenuated *Babesia* strains are used to develop immunity.

3. Herd Management

- Monitor and treat new animals entering the herd to prevent the introduction of infected animals.
- Avoid overgrazing and maintain hygiene to reduce tick habitats.

4. Minimizing Stress

- Stress in animals can exacerbate susceptibility to infection.

4.2 Coccidiosis in Calf

Coccidiosis is a parasitic disease caused by protozoa of the genus *Eimeria*. It primarily affects young calves (1-6 months old) and is a significant cause of diarrhea, poor growth, and economic losses in livestock farming. Calves are particularly vulnerable during periods of stress, overcrowding, and poor hygiene.

1. Etiology

• Causative Agents

- *Eimeria zuernii*
- *Eimeria bovis*
- *Eimeria alabamensis* (less common)
- These protozoa invade and multiply in the intestinal lining, causing damage to epithelial cells.

2. Mode of Transmission

1. Fecal-oral Route

- Ingestion of sporulated oocysts from contaminated feed, water, or environment (e.g., bedding, soil).

2. Risk Factors

- Overcrowding.
- Poor sanitation.
- Stress (weaning, transportation, or weather changes).

3. Symptoms

Subclinical infection

- No visible signs but reduced growth and feed efficiency.

Clinical infection

• Mild Cases

- Reduced appetite.
- Soft stools.

• Severe Cases

- Profuse, watery diarrhea, often with mucus or blood.
- Dehydration.
- Weight loss and weakness.
- Straining during defecation (tenesmus).
- Fever (in some cases).
- Death in severe and untreated cases.

4. Diagnosis

1. Clinical Signs

- Diarrhea, dehydration, and poor growth in young calves.

2. Fecal Examination

- Identification of *Eimeria* oocysts using microscopic techniques.

3. Histopathology

- Examination of intestinal tissue for the presence of *Eimeria* life stages.

4. Molecular Diagnostics

- PCR for species-specific identification of *Eimeria*.

5. Differential Diagnosis

- Rule out other causes of diarrhea such as cryptosporidiosis, salmonellosis, and rotavirus infection.

6. Treatment

1. Anticoccidial Drugs

- **Sulfonamides** (e.g., sulfadimethoxine).
- **Amprolium** (10 mg/kg for 5 days).
- **Toltrazuril** (single dose, 15–20 mg/kg).

2. Supportive Therapy

- Oral or intravenous fluids to manage dehydration.
- Electrolyte supplementation.

3. Probiotics

- To restore gut microflora after treatment.

7. Prevention and Control

1. Management Practices

- Maintain proper sanitation in calf housing.
- Reduce overcrowding and improve ventilation.
- Separate younger calves from older ones to limit exposure.

2. Prophylactic Treatment

- Use anticoccidials (e.g., coccidiostats like amprolium or decoquinate) in feed or water for high-risk calves.

3. Environmental Control

- Regularly clean and disinfect feeding and watering equipment.

- Remove manure frequently to reduce oocyst contamination.

4. Nutrition and Stress Management

- Provide a balanced diet to strengthen the calf's immune system.
- Minimize stress during handling and weaning.

Exercise

Choose the correct answer from the given alternatives.

1. What is the causative agent of babesiosis?
 - a. *Eimeria bovis*
 - b. *Babesia bigemina*
 - c. *Trypanosoma evansi*
 - d. *Theileria parva*
2. What is the main vector responsible for the transmission of Babesiosis?
 - a. Fleas
 - b. Snails
 - c. Ticks (*Rhipicephalus* species)
 - d. Flies
3. Which clinical sign is commonly associated with Babesiosis?
 - a. Red-colored urine (hemoglobinuria)
 - b. Greenish diarrhea
 - c. Respiratory distress
 - d. Skin lesions
4. How is Babesiosis diagnosed?
 - a. Fecal flotation
 - b. Giemsa-stained blood smears
 - c. Skin biopsy
 - d. ELISA for bacterial antibodies
5. Which drug is commonly used to treat Babesiosis?
 - a. Praziquantel
 - b. Imidocarb dipropionate
 - c. Albendazole
 - d. Metronidazole
6. What is a key preventive measure for Babesiosis?
 - a. Use of anticoccidials in feed
 - b. Tick control with acaricides
 - c. Vaccination with *Babesia* toxoids
 - d. Improving ventilation in barns

7. What is the main causative agent of Coccidiosis in calves?
 - a. *Babesia bovis*
 - b. *Eimeria zuernii*
 - c. *Cryptosporidium parvum*
 - d. *Theileria annulata*
8. Which drug is commonly used to treat Coccidiosis?
 - a. Amprolium
 - b. Imidocarb
 - c. Praziquantel
 - d. Triclabendazole
9. What is a key measure to prevent coccidiosis in calves?
 - a. Regular deworming with albendazole
 - b. Tick control in grazing areas
 - c. Hygiene management and oocyst reduction in the environment
 - d. Vaccination with live attenuated *Eimeria* vaccine
10. Coccidiosis is most commonly observed in calves aged.....
 - a. Less than 1 month
 - b. 1-6 months
 - c. 6-12 months
 - d. Above 1 year

Write short answer to the following questions.

1. Write etiology and clinical signs of babesiosis.
2. Write etiology and clinical signs of babesiosis.
3. How is Babesiosis diagnosed?
4. How is coccidiosis diagnosed?

Write long answer to the following questions.

1. Write etiology, clinical signs, method of diagnosis, treatment and prevention of Babesiosis.
2. Write etiology, clinical signs, method of diagnosis, treatment and prevention of Coccidiosis in calf.

Project Work

1. Write the procedure of blood sample examination for protozoal disease.

5.1 Hemorrhagic Septicemia

Synonyms

- Pasteurellosis
- Shipping fever

Definition

- It is an acute septicemic disease occurring very often in cattle, buffalo, camel, sheep, goat and pig usually following some form of stress, such as transportation, exertion etc.

Distribution

- Tropical and subtropical countries.
- most commonly during or following monsoon.
- The outbreaks are seen during the period of high humidity.

Etiology

- The disease is caused by *Pasteurella multocida* type -1 organism.
- The organism may be present in the respiratory tract as commensal and may not be able to produce the disease alone.

Susceptible Hosts

- Cattle and buffaloes are the most susceptible species.
- Young growing cattle within the age group of 6 months to 2 years are most often affected.
- Over fed cattle may suffer when they are put under stress.
- Sheep, goats and pigs

Clinical Findings

- The incubation period is 2-5 days.
- There is high rise of temperature (104-1070F) with concurrent shivering.
- Profuse salivation, lacrimation and nasal discharge.
- There is sign of conjunctivitis and the visible mucous membrane assume deep red color.
- There is sharp drop in milk yield in lactating cows.
- Abdominal pain, severe diarrhea or dysentery.
- Respiration rate is rapid in nature.
- Auscultation of lung reveals increased vesicular rales and moist rales. Due to the progression of the disease lung sound may become inaudible as a result of consolidation.
- In less acute cases localization of edema in the subcutaneous pocket of the head, neck, dewlap and brisket region.
- The edematous swelling are hot and painful.
- The edema produces severe dyspnea due to obstruction of respiratory passage.
- Death usually occurs within 20-24 hours.

Diagnosis

- History of predisposing factors and clinical findings.
- Nasal swab or washing and the exudates may be used for culture from living animals.
- Smears from heart blood, liver, lungs, spleen and intestinal content from dead animals should be stained with leishman stain. Bipolar short ovoid rods may be located.

Treatment

- In the initial phase of the disease intravenous administration of

sulphonamides i.e. sulphamethazine @ 150 mg / kg body weight for 3 days or sodium sulphadimidine in the similar dose may give effective response.

- Oxytetracycline @ 5-10 mg/kg body weight for 3-5 days.
- Besides, treatment may be extended with chloramphenicol @ 10 mg/kg body weight or ampicillin @ 10 mg/kg body weight.
- Long acting oxytetracycline may be given to reduce the treatment cost.
- Symptomatic treatment with anti-inflammatory drugs eg. Betamethazone or Dexamethazone @ 1 mg/5kg body weight may be required.

5.2 Anthrax

Synonyms

- Splenic fever
- Milzbrand (German term)
- Charbon (Malignant charcoal)
- Wool sorter's disease
- Patkhe (Nepal)

Distribution

- It is one of the ancient diseases that is worldwide in distribution.

Susceptible Hosts

- Most of the animals food
- The most susceptible animals are cattle and sheep.
- Horses and pigs are also affected.
- Not uncommon in camels.
- Anthrax is a zoonotic disease.

Etiology

- *Bacillus anthracis*.

Mode of Transmission

Soil Borne

- Animals while grazing in the infected pasture pick up the infection (spore) through ingestion or through breach in the oral mucosa or skin. The new area may be infected due to contaminated animal products such as bone meal, fertilizers, hide, hair, wool, grain or forage.

Clinical Findings

Per-acute Form

- Sudden death of animal

Acute Form

- Elevation of body temperature (104 to 1080F).
- Animal refuses to eat and there is development of bloat.
- Ruminal stasis is evident.
- Animal is extremely depressed.
- Pulse and respiratory rates are accelerated to a great extent.
- With the advancement of the disease process there is development of muscular tremor.
- Some animals may show extreme aggressive behaviour
- At the end, animal show distressed breathing and mouth breathing.
- Death usually takes place within 48 hours.
- Death is followed with bleeding from all the natural orifices.

Sub-acute Form

- It is characterized by oedema.
- Edema is predominantly noticed under the neck, brisket region, thorax, abdomen and flank.
- Pregnant cattle may abort.
- Some may survive for 2 to 3 months.

Diagnosis

- Diagnosis is based on clinical history of abrasion of skin, history of occurrence near about area.
- Post mortem examination
- Exudation of tarry blood from body orifices.
- Failure of the blood clotting
- Absence of rigormortis.
- Splenomegaly.
- If anthrax is suspected post mortem examination should be avoided to reduce environmental contamination and health hazard.

Treatment

- Penicillin 10,000 units per kg body weight twice daily through parenteral route.
- Oxytetracycline, erythromycin, or sulphonamide has been used.
- Anti-anthrax serum @ 100 – 200 cc through intravenous route along with a course of penicillin may be given.

Prevention & Control

- Control of meat and milk from animals in infected herds–
- Avoid any risk to the human population
- Avoid unnecessary waste & imposition of unnecessarily harsh prohibitions on farmer
- When an outbreak occurs, followings are part of animal disease control program and indirectly reduce human exposure
- Placing of farm on quarantine
- Destruction of discharges and cadavers
- Vaccination of survivors
- Prohibition of movement of meat and milk from farm during quarantine

period should prevent entry of infection into the human food chain

- Hygiene is the biggest single factor in the prevention of spread of disease–
- Careful disposal of infected material
- Infected carcasses should not be opened but immediately burned / buried, together with bedding and soil contaminated by discharges
- Burial should be at least 2 m deep with an ample supply of quicklime added
- All suspected case and in-contact animals must be segregated until cases cease and for 2 weeks thereafter the affected farm placed in quarantine to prevent movement of animals

5.3 Black Quarter

Synonyms

- Black leg
- Quarter ill
- Quarter evil
- Emphysematous gangrene
- Charchare (Nepal)

Definition

This is an acute infectious disease of cattle, goat and sheep. The disease is characterized by development of focal gangrenous and emphysematous myositis.

Etiology

Black quarter is caused by *Clostridium chauvoei*, a gram positive rod shaped, spore forming toxin producing anaerobe.

Transmission

- Disease spreads from contaminated soils.

- Contamination of the soil is due to infected feces and carcasses which cause pollution of the land.
- Organisms gain entry through ingestion of infected feeds or possibly through contamination of wounds.

Clinical Findings

- The first sign is rise in body temperature which may be as high as 1060 F 1080 F.
- The appetite is lost and rumination is suspended and there is stiffness or lameness in one of the limb.
- Very soon characteristic swelling develops in one of the thick layers of muscles. Most commonly the lesions are located on the thigh, buttock, shoulder, neck and lumbar region. Swelling are hot and painful in the early stage and become cold and painless later.
- On pressure swellings emit crackling or crepitating sound due to emphysema.
- Skin over the swelling is discolored and dry.
- There is labored breathing and accelerated pulse rate (100-120/minute).
- Occasionally there is distinct abdominal pain.
- Finally, the temperature drops and the patient dies within 12 to 48 hours.

Diagnosis

- History of outbreak.
- Clinical observation and postmortem findings.
- Microscopic examination of smear

Treatment

- Satisfactory response has been reported from the use of penicillin, aureomycin and oxytetracycline.

- The antibiotic may be injected into the affected muscles.
- Penicillin is extensively used and considered as drug of choice.
- Penicillin @ 20,000 to 40,000 units per kg body weight per day may be used.
- Crystalline penicillin may be given through intravenous route followed by procaine penicillin through i/m route.

Control

- Since the disease is associated with infection from the soil, the cultivation in that soil may be avoided.
- The young animals should be kept out of such area.
- The dead body should be burnt or buried.
- The dead body should not be allowed to be skinned.
- The calf should not be allowed to graze in endemic pasture.
- All the animals of the endemic zones should be vaccinated with suitable vaccine.

5.4 Mastitis

Synonyms

- Mammitis
- Mammite
- Inflammation de la ubres.

Definition

Mastitis is the term which denotes inflammatory condition of the udder irrespective of causes. It is characterized by physical, chemical and microbiological changes in the milk and pathological changes in the glandular tissues of the udder. The changes in the milk include change of color, change of consistency (clot) and presence of abnormally large number of leukocytes.

Etiology

Cattle

Bacteria: *Staphylococcus aureus*, *Str. agalactiae*, *Str. zooepidemicus*, *Str. faecalis*, *Str. pyogenes*, *Corynebacterium pyogenes*, *Klebsiella Spp*, *Mycobacterium bovis*, *Escherichia coli*, *Brucella abortus*; *Pseudomonas pyocyaneus*, *Leptospira pomona*, *Pasteurella multocida*.

Mycoplasma: *Mycoplasma bovis*; *Mycoplasma bovigenetelium*.

Fungus: *Trichosporon spp*, *Aspergillus fumigatus*, *Aspergillus midulus*, *Candida Spp*, *Cryptococcus neoformans*.

Virus: Some viruses (Vesicular stomatitis, Infectious rhinotracheitis) have been implicated as cause of mastitis in cattle.

Buffalo

Bacteria: *Staphylococcus aureus*, *Streptococcus agalactiae*, *Streptococcus dysagalactiae*, *Streptococcus uberis*, *Streptococcus bovis*, *Escherichia coli*; *Pseudomonas spp.*

Mycoplasma: *Mycoplasma bovis*, *Mycoplasma bovigenetelium*.

Fungus: *Trichosporon spp.*; *Aspergillus fumigatus*; *Aspergillus midulus*, *Candida Spp*, *Cryptococcus neoformans*.

Horse

Bacteria: *Corynebacterium pseudotuberculosis*, *Streptococcus zooepidemicus*

Mode of Transmission

- The venue of infection is the teat canal. Through teat canal the infection reaches the mammary gland.
- There are two sources of infective agents. They are the *udder*-where many bacteria like *Streptococcus agalactiae* and *Staphylococcus aureus* may continue to persist as normal inhabitant and the *environment*-where bacteria like *Escherichia coli* and *Pseudomonas pyocyaneus*

continue to persist.

- These organisms when multiply and invade the tissues produce much damaging effect and they are not amenable to common hygienic practices.
- Cutaneous surface of the cow may have many organisms as resident population and from where the organisms may have the chance to invade through contamination by handlers.
- Contamination of milkers hands, clothes and machine cup by milk from the affected quarter may lead to the spread of the disease to other non-infected teats of cows.
- Fly and other insects may also spread the infection from one place to the other.
- Spread of infection is also possible through bedding ground by discharges of affected gland.

Clinical Findings

Mastitis may clinically be classified as (i) Per-acute mastitis (ii) Acute mastitis (iii) Sub acute mastitis and (iv) chronic mastitis.

Per-acute Mastitis

- Affected animal shows a very high temperature (106-1070 F).
- Offfed and show respiratory distress.
- Udder is swollen and extremely painful.
- Cessation of milk secretion and exudates are often blood stained.

Acute Mastitis

- There is no systemic reaction.
- Udder becomes swollen and there is changes in milk.
- Milk may be replaced by custard material yellow or brown fluid with flakes or clots.

- Infection may be localized in one quarter or the entire udder may be involved. ***Subacute mastitis***
- Variable changes in the milk but practically no
- Changes in the udder tissues.
- Culture of milk will show presence of pathogenic bacteria.

Chronic Mastitis

- Chronic mastitis is the terminal stage of the disease.
- Udder becomes hard due to fibrosis.
- Supra-mammary lymphnodes become palpable.
- Quarters may become thickened, firm, nodular and atrophic.
- Parenchymatous cells are replaced by connective tissues.
- Milk changes vary considerably. The milk may appear as yellowish fluid or white with clots and flakes. Sometime it may look as green or yellow-green and foul smelling.
- Teats may reveal injuries or sores near the orifices with involvement of supramammary lymph nodes.

Diagnosis

- History
- Clinical findings
- *Physical examination of the udder:* Palpation and inspection of the udder are directed for the detection of fibrosis, inflammation, swelling and atrophy
- Pathological tests to detect bacteria, cells, clots and chemical changes
- Direct microscopic examination for detection of bacteria and WBC
- Bromothymol blue test
- Bromocresol purple test
- Chloride test

- California mastitis test (CMT)
- Strip cup test

Treatment

Treatment strategies vary with the clinical severity of the disease.

- The sub-acute case requires only intramammary therapy for 3 days after each milking.
- The acutely affected cow should be treated with systemic and intramammary antibiotics for a minimum of 3 days.
- The per-acute cow needs to be treated with systemic and intra-mammary antibiotics, oral or intravenous fluids and anti-inflammatory drugs.

Per-acute Treatment

- Although the rate of diffusion of antibiotic is greater but still the selection of parenteral antibacterial drugs should be made on the basis of diffusion from the blood stream into the milk.
- Erythromycin, tylosin, penethemate, chloramphenicol and trimethoprim are diffused well.
- Penicillin and tetracyclines are medium performers and neomycin and streptomycin are poor diffusers.
- Treatment of mastitis should be made on the basis of resistant of the antibacterial drugs.

Udder Infusions

- Disposable tube containing suitable drugs in a water soluble ointment base are best suited for dispensing and the treatment of individual cow.
- Complete emptying of the quarter before infusion by the parenteral injection of oxytocin is advisable in cases of acute mastitis. Strict hygienic measures is necessary during treatment to avoid the introduction of bacteria, yeasts and fungi into the treated quarters.
- The drugs which have the best record of diffusion through the udder

after intra-mammary infusion are penethemate, ampicillin, amoxycillin, chloramphenicol, novobiocin, erythromycin and tylosin.

- Those of medium performance are penicillin G, cloxacillin and tetracyclines.

5.5 Brucellosis

Synonyms

- Bang's disease
- Contagious abortion
- Infectious abortion
- Enzootic abortion
- Undulant fever
- Malta fever
- Mediterranean fever

Definition

Brucellosis is an acute or chronic contagious disease of domestic animals that mainly causes placentitis and abortion in females, and orchitis and epididymitis in males.

Etiology

Brucella spp

Brucella abortus (cattle), *Brucella melitensis* (goat), *Brucella suis* (swine), *Brucella ovis* (sheep)etc.

Clinical Findings

- Abortion takes place from 6 months onwards.
- Greyish white mucoid or muco-purulent discharges from vagina.
- Prior to parturition cow may show the clinical pattern of normal parturition like swelling of vulva, relaxation of pelvic ligament,

enlargement of udder and discharges from vulva.

- Retention of foetal membranes.
- There may be chronic endometritis.
- In bull epididymitis, orchitis and inflammation of accessory genital organ.

Diagnosis

- By clinical findings.
- Laboratory diagnosis
- Isolation and identification of organisms.
- Serological test: RBPAT, Standard tube agglutination test, CFT, MRT, ELISA.

Treatment

- The organisms live inside the cells of the infected animal and that is why the success rate of the treatment is not impressive.
- Long acting Oxytetracycline 20 mg/kg b wt i/m 5 injections at 3-4 days intervals and streptomycin 25 mg/kg b wt i/m or i/v daily for 7 days.
- In case of infected horse, Chloramphenicol 1 gm/ 100 kg b wt i/m or i/v daily for 12-20 days.

5.6 Enterotoxaemia

Enterotoxaemia is an infectious disease of ruminants that result due to absorption of certain bacterial toxin which is being formed in the intestine.

Etiology

Enterotoxaemia is caused by *Clostridium perfringen's* that produce toxins starting from A to F of which type A, B, C, D, E are important.

Clinical Signs

- Dysentery

- Abdominal pain
- Spasms and aimless wandering
- Weakness
- Depression
- Haemorrhagic diarrhea

Treatment

Oral antibiotics (tetracycline, chlortetracycline, penicillin) may be used.

5.7 Pneumonia

Pneumonia is the inflammation of the pulmonary parenchyma usually accompanied by inflammation of the bronchioles and often by pleurisy. It is manifested clinically by an increase in respiratory rate, cough, abnormal breath sound on auscultation and in most bacterial pneumonia, by evidence of toxemia.

Causes

Cattle

1. Pneumonic pasteurellosis (Shipping fever). *Pasteurella hemolytica*, *P.multocida* with or without parainfluenza –3 virus (PI3).
2. Enzootic pneumonia of calves: PI3, adenovirus 1,2, and 3 Rhinovirus, Bovine Syncytial Virus (BSV), reoviruses, Bovine herpes virus 1 (BSV virus) plus chlamydia spp. *Mycoplasma* spp. *Pasteurella* spp. *Corynebacterium pyogenes*, *Strept* spp. and *bedsonia* spp. (*Actinobacillus actinoides*).
3. Viral Pneumonia of yearling and adult cattle is caused by either PI3 or adenoviruses.
4. Contagious bovine pleuropneumonia caused by *Mycoplasma mycoides*.
5. Atypical interstitial pneumonia.
6. Massive infestation with ascarid larvae.
7. Lungworm pneumonia (*dictyocaulus viviparous*)

8. Klebsiella pneumonia infection in calves and nursing cows suffering from mastitis caused by this organism.
9. Sporadically in T.B. (mycobacterium bovis).
10. Sporadically in calf Diphtheria (spherous necrophenous).
11. Hemophilus somnus, possibly in young cattle affected with the more common septicemic form of the disease. Its role as primary cause is uncertain.

Horses

1. **Newborn foals:** Any of the septicemia which occur at those time, Strep. Sp., E. Coli, Actinobacillus equi.
2. In immunodeficient foals, pneumonia is caused by adenoviruses or pneumocystis carini.
3. **Older foals:** Corynebacterium, Rhodococcus equi and equine herpesvirus.
4. Dictycaulus arnifeldi and Parascarsis equorum rarely cause significant pneumonia.
5. As a sequel to Strangles.
6. Rarely as sequelae to equine viral arteritis or equine viral rhinopneumonitis in adult animals.
7. Glanders and epizootic lymphangitis (histomonus farcinicus) usually include pneumonic lesions.

Sheep

1. Pneumonic pasteurellosis (pasteurella sp.) as acute primary pneumonia in feedlot lambs, or secondary to PI3 or Chlamydia spp.
2. Newborn lambs: Uncommonly Strept. Zooepidemicus, Salmonella abortus ovis.
3. Mycoplasma spp. (severe pneumonia).
4. Symptomless pneumonia without secondary infection adenovirus, RSV, reovirus mycoplasma sp.

5. *Corynebacterium pseudotuberculosis* (sporadic).
6. *Melioidosis (pseudomonas pseudomallei)*.
7. Lungworm pneumonia (*dictycolus filarial*).
8. Progressive interstitial pneumonia

Goats

1. Pleuropneumonia caused by mycoplasma strain F38 or *M.Capri* is a devastating disease.
2. Chronic interstitial pneumonia with pulmonale as common sequel by a number of mycoplasma spp. *M. mycoides var. mycoides* appears to be the most commonly recorded.
3. Rotavirus infection.

All species

1. Toxoplasmosis (sporadic cases).
2. Systemic mycosis lesion are focal only.
3. Aspiration pneumonia.
4. Secondary pneumonia caused by Strept. spp. Corynbact. spp. *dermatophilus* sp.
5. Interstitial pneumonia, pulmonary consolidation and fibrosis by toxins in plants (*Eupatorium glandulosum* in horses), *Zieria arborescens* (stinkwood) in cattle, *Astragalus* sp. in all species.

Clinical findings

1. Rapid, shallow respiration, is the cardinal signs of early pneumonia, dyspnea occurring in the later stages.
2. Polypnoea may be quite maker with only minor pneumonic lesions and the rapidity of the respiration is an inaccurate guide to the degree of pulmonary involvement.
3. Cough is other important sign (the type of the cough varies with the type of

the lesion). Bronchopneumonia is usually accompanied by a moist painful cough, interstitial pneumonia by frequent, dry, hacking cough, often in paroxysms.

4. Cyanosis is not a common sign and occurs only when large areas of the lung are affected.
5. A nasal discharge may or may not be present in the bronchioles whether or not there is accompanying inflammation of the upper respiratory tract.
6. The odour of the breath may be informative. It may have an odour of decay when there is a large accumulation of inspissated pus present in the air passages, or putrid, especially in horses, when pulmonary gangrene is present.
7. Auscultation of the thorax before and after coughing may detect exudate in the air passages.
8. By auscultation in the early congestive stage of bronchopneumonia and interstitial pneumonia the vascular murmur is increased. Moist rales develop in broncho-pneumonia as bronchiolar exudation increases but in uncomplicated interstitial pneumonia, clear, harsh bronchial tones are audible. When complete consolidation occurs in either form, bronchial tones are the only sounds audible over the affected lung but moist or crepitant rales can be heard at the periphery of the affected area in bronchopneumonia.
9. Consolidation also causes increased audibility of the heart sounds. When pleurisy is also present it causes a pleuritic friction rub in the early stages and muffling of the pulmonary sounds in the late exudative stages. Consolidation can be detected also by percussion of the thorax or by tracheal percussion.
10. There may be an observable difference in the amount of movement in the two sides of the chest if the degree of consolidation is much greater in one lung.

Additional signs evident in pneumonia include fever of variable severity, anorexia, depression and an increase in pulse rate.

- a. Swab or tracheal sputum and determination of the sensitivity of the isolated bacteria to antibacterial agents.
- b. Transtracheal aspiration has been described earlier and is valuable tool for an intensive investigation of a respiratory tract infection.
- c. Radiographic examinations are undertaken only in animals of suitable size.
- d. Hematological examination usually reveals a leukocytosis with shift to the left in bacterial pneumonia.
- e. A leukopenia and lymphopenia occurs in some cases of acute viral pneumonia. In viral pneumonia, the serological testing of acute and convalescent sera, in addition to the isolation of the virus, are useful tools supports in evidence of the presence of an active infection.

Diagnosis

There are two kinds of errors in the clinical diagnosis of pneumonia.

- One of that is the pneumonia is not detected clinically, because the abnormal lung sound are apparently not obvious.
- The other is to make a diagnosis of pneumonia, because of the presence of dyspnoea which was due to disease in some other body systems.
- The major clinical findings of pneumonia are polypnoea in the early stage and dyspnoea later, abnormal lung sounds, and fever and toxemia in bacterial pneumonia. Polypnoea and dyspnoea may result from involvement of other body systems as congestive heart failure, terminal stage of anemia, poisoning by histotoxic agents such as hydrocyanic acid, hyperthermia and acidosis are accompanied by respiratory embarrassment, but not by the abnormal sounds typical of pulmonary involvement. Pulmonary edema and congestion, embolism of the pulmonary artery and emphysema are often mistaken for

pneumonia, but can be usually differentiated by the absence of fever and toxemia, on the basis of the history and on auscultation findings.

- All of the practical laboratory aids described should be used when necessary. They are of particular importance when outbreaks of pneumonia are encountered. In a single routine case of pneumonia, the cause is usually not determined.
- However the age and class of the animal, the history and epidemiological findings and the clinical findings can usually be correlated and a presumptive etiological diagnosis done.

Treatment

1. In specific infection isolation of affected animals and careful surveillance of the remainder of the group to detect cases on the early stages should accompany the administration of the specific antibacterial drugs or biological preparations to affected animals. The choice of antibacterial agents will depend on the tentative diagnosis, the experience with drugs in previous cases and the results of the drug sensitivity tests.
2. The common bacterial pneumonia of all species will usually recover quickly (24 hr.) if treated with an adequate dose of the drug of choice early in the course of the disease.
3. Animals with severe pneumonia will require daily treatment for the several days until recovery occurs.
4. Those with bacterial pneumonia and toxemia must be treated early on an individual basis. Each case should be identified and carefully monitored for failure to recover.
5. Antimicrobial agents in a long acting base may be used to provide therapy over a 4-6 day period instead of the daily administration of the shorter-acting preparations. However, the blood level from the long-acting preparations are not as high as the shorter-acting preparations and may not be as effective in severely affected animals.

6. The common causes for failure to respond favourably to treatment for bacterial pneumonia include: (A) advanced disease when treatment was undertaken, (B) the development of pleurisy and pulmonary abscesses, (C) drug resistant bacteria.
7. There is no specific treatment for the viral pneumonia because viral and mycoplasmal pneumonia are commonly complicated by secondary bacterial infections. It is common practice to treat acute viral and mycoplasmal pneumonia with antibacterial until recovery is apparent.
8. In outbreaks of pneumonia where many animals are affected and new cases occur each day for several days, the use of mass medication of the feed and / or water supplies should be considered.
9. Mass medication may assist in the early treatment of subclinical pneumonia and is a labor-saving method of providing convalescent therapy to animals which have been treated individual.
10. When outbreaks of pneumonia occur and new cases are being recognized at the rate of 5% -10 % per day of the total in the group, all the remaining in contact animals may help to treat subclinical cases before they become clinical and thus “abort” the outbreak.
11. Corticosteroids have been used for their anti-inflammatory effect in the treatment of acute pneumonia. However, there is no clinical evidence that they are beneficial. Affected animals should be housed in warm, well ventilated, draft-free accommodation, provided with ample, fresh water, and light nourishing food.
12. During convalescence condition, the return to work or exposure to bad or cold weather should be avoided. If the animal does not eat, oral or parenteral forcefeeding should be initiated. If fluids are given intravenously care should be exercised in the speed with which they are administered. Injection at too rapid rate may cause overload on the heart ventricle and death may occur due to acute heart failure.

13. Supportive treatment may include the provision of O₂ supply to be available especially in the critical stages when hypoxia is evident. In foals, the oxygen can be administered through an intranasal tube passed back to the nasopharynx and delivered at the rate of about 3 liters / min. for several hours.

Expectorants may be of value in chronic cases and during convalescence.

5.8 Tuberculosis

Tuberculosis (TB) is a chronic, infectious disease caused by bacteria in the genus *Mycobacterium*, primarily *Mycobacterium bovis* in animals. It affects multiple species, including cattle, goats, deer, and occasionally companion animals, leading to significant economic and public health concerns due to its zoonotic potential.

Etiology

- **Causative Agent**
 - *Mycobacterium bovis* (primary cause in animals).
 - *Mycobacterium avium* (in birds).
 - *Mycobacterium tuberculosis* (occasionally transmitted to animals from humans).
- These are acid-fast, slow-growing, aerobic bacilli.

Mode of Transmission

1. Direct Contact

- Inhalation of aerosols from infected animals (primary route in cattle).
- Ingestion of contaminated feed, water, or milk (common in calves).

2. Indirect Contact

- Contact with contaminated bedding or soil.

3. Vertical Transmission

- Rare but possible via transplacental or milk transmission.

4. Zoonotic Potential

- Humans can contract *M. bovis* through unpasteurized milk or direct contact with infected animals.

Clinical Signs

- Often subclinical in early stages.
- **Respiratory Signs**
 - Chronic cough.
 - Dyspnea and tachypnea.
 - Nasal discharge (sometimes with blood).
- **General Signs**
 - Weight loss and emaciation.
 - Decreased appetite and milk production.
- **Localized Lesions**
 - Enlarged lymph nodes, especially in the cervical region.
 - Abscesses or draining sinuses.
- **Other Signs**
 - Involvement of other organs (e.g., digestive, reproductive) depending on disease progression.

Diagnosis

1. Clinical Examination

- Physical signs, history of exposure.

2. Tuberculin Skin Test

- *Caudal fold test* or *comparative intradermal test*.

3. Serological Tests

- ELISA for detecting antibodies.

4. Direct Detection

- Acid-fast staining of samples.
- Culture on specialized media (e.g., Lowenstein-Jensen).

5. Molecular Methods

- PCR for specific *Mycobacterium* DNA.

6. Postmortem Examination

- Granulomas in affected organs.

Treatment

- Treatment in animals is generally **not recommended** due to:
 - Public health risks.
 - High costs and prolonged duration.
 - Risk of drug resistance.
- In pet or zoo animals:
 - Long-term antibiotic therapy with combinations such as rifampin, isoniazid, and pyrazinamide under strict veterinary supervision.

Prevention and Control

1. Herd Management

- Regular testing and culling of infected animals.
- Isolation of newly introduced or suspect animals.

2. Sanitation

- Clean and disinfect housing, feeding areas, and equipment.

3. Milk Pasteurization

- Prevents zoonotic transmission via milk.

4. Surveillance and Biosecurity

- Monitoring wildlife reservoirs.
- Controlling access to farm premises.

5. Vaccination

- BCG vaccine used experimentally in some wildlife and high-risk areas.
- Not widely implemented in livestock due to interference with diagnostic tests.

6. Public Health Measures

- Education on zoonotic risks.
- Screening of farm workers for TB.

Exercise

Choose the correct answer from the given alternatives.

1. What is the synonym for Hemorrhagic Septicemia?
a. Foot-and-mouth disease b. Blackleg
c. Pasteurellosis d. Malignant catarrhal fever
2. What is the causative agent of Hemorrhagic Septicemia?
a. *Escherichia coli* b. *Pasteurella multocida*
c. *Clostridium perfringens* d. *Mycobacterium bovis*
3. Which of the following is the mode of transmission for Hemorrhagic Septicemia?
a. Fecal-oral route
b. Direct contact with infected animals
c. Vector-borne transmission by ticks
d. Inhalation of aerosolized spores
4. Which clinical symptom is most commonly observed in animals infected with Hemorrhagic Septicemia?
a. Abdominal distension
b. Sudden high fever and swelling of neck
c. Paralysis of the limbs
d. Rashes on the skin
5. Which is the causative organism of Anthrax?
a. *Brucella abortus* b. *Clostridium botulinum*
c. *Bacillus anthracis* d. *Pasteurella multocida*
6. What is the common route of transmission for Anthrax?
a. Airborne spores

- b. Ingestion of contaminated feed or water
- c. Tick bites
- d. Fecal-oral transmission

7. Which of the following is not a symptom of Anthrax in cattle?

- a. Sudden death
- b. Difficulty in breathing
- c. Severe swelling in the neck and chest
- d. Excessive salivation

8. What is the causative agent of Black Quarter?

- a. *Clostridium tetani*
- b. *Clostridium perfringens*
- c. *Clostridium chauvoei*
- d. *Mycobacterium bovis*

9. How is Black Quarter typically transmitted?

- a. Ingestion of contaminated food
- b. Inhalation of spores
- c. Skin wounds or injuries, especially in anaerobic environments
- d. Direct contact with infected urine

10. Which clinical sign is the characteristic of black quarter in cattle?

- a. Swollen limbs with crepitus (crackling sound)
- b. Severe diarrhea
- c. Lameness in one leg
- d. Jaundice

11. Which of the following is the most common cause of Mastitis in dairy cattle?

- a. *Escherichia coli*
- b. *Brucella abortus*
- c. *Streptococcus agalactiae*
- d. *Listeria monocytogenes*

12. What is the primary mode of transmission for Mastitis?

- Direct contact with contaminated bedding
- Fecal-oral route
- Inhalation of infected aerosols
- Contaminated milking equipment and improper handling

13. What is a common clinical sign of Mastitis?

- Swollen, hot, and painful udders
- Coughing and nasal discharge
- Redness and fever in the skin
- Diarrhea and vomiting

14. Which is the causative organism of brucellosis in cattle?

a. <i>Brucella abortus</i>	b. <i>Brucella melitensis</i>
c. <i>Brucella suis</i>	d. <i>Brucella canis</i>

15. What is the primary mode of transmission for brucellosis?

- Fecal-oral route
- Contact with infected blood or aborted fetuses
- Inhalation of airborne bacteria
- Vector transmission through ticks

6. Which of the following is a common symptom of brucellosis?

a. Sudden death	b. Abortions in pregnant females
c. Severe diarrhea	d. Loss of appetite and dehydration

17. Which bacteria is most commonly associated with enterotoxaemia in calves?

a. <i>Clostridium perfringens</i>	b. <i>Escherichia coli</i>
c. <i>Salmonella enterica</i>	d. <i>Staphylococcus aureus</i>

18. What is the major symptom of enterotoxaemia in calves?
 - a. Severe vomiting
 - b. Sudden death and abdominal distension
 - c. Excessive salivation
 - d. Diarrhea with mucus
19. Which of the following is used for the prevention of enterotoxaemia in livestock?
 - a. Routine vaccination with *Clostridium* toxoid
 - b. Use of antibiotics in feed
 - c. Good sanitation and controlling feed contamination
 - d. Regular deworming
20. Which is the most common causative organism for pneumonia in cattle?

a. <i>Mycobacterium bovis</i>	b. <i>Pasteurella multocida</i>
c. <i>Escherichia coli</i>	d. <i>Clostridium botulinum</i>
21. What is a common clinical sign of pneumonia in cattle?
 - a. Labored breathing and coughing
 - b. Swollen joints and lameness
 - c. Excessive salivation and dehydration
 - d. Reddened eyes and nasal discharge
22. How is pneumonia in cattle typically diagnosed?
 - a. Blood smear examination
 - b. Fecal culture for *E. coli*
 - c. Clinical signs and lung auscultation, followed by radiography
 - d. Skin biopsy

23. Which is the causative organism of tuberculosis in cattle?
 - a. *Mycobacterium tuberculosis*
 - b. *Mycobacterium bovis*
 - c. *Mycobacterium avium*
 - d. *Brucella abortus*
24. What is the primary mode of transmission for tuberculosis in cattle?
 - a. Fecal-oral route
 - b. Airborne droplets from coughing and sneezing
 - c. Ingestion of contaminated water
 - d. Skin contact with infected soil
25. Which of the following is not a common symptom of tuberculosis in cattle?
 - a. Chronic cough
 - b. Weight loss and weakness
 - c. Enlarged lymph nodes
 - d. Sudden death

Write short answer to the following questions.

1. Write etiology, clinical signs and method of diagnosis of anthrax.
2. Write etiology, method of diagnosis and prevention and control measures of HS.
3. Write etiology, method of diagnosis and prevention and control measures of black quarter.
4. Write clinical signs and method of diagnosis of mastitis.
5. Is tuberculosis a zoonotic disease? How can it be prevented?
6. Write clinical signs and line of treatment of enterotoxaemia.
7. Write etiology and clinical signs of brucellosis.
8. Write clinical signs and method of diagnosis of pneumonia.

Write long answer to the following questions.

1. Write etiology, clinical signs, method of diagnosis and treatment of anthrax.
2. Write etiology, mode of transmission, method of diagnosis and prevention and control measures of HS.

3. Write etiology, clinical signs, method of diagnosis and prevention and control measures of Black quarter.
4. Write clinical signs, mode of transmission and method of prevention of mastitis.
5. Write etiology, clinical signs, method of diagnosis and prevention of tuberculosis.
6. Write clinical signs and line of treatment and prevention and control measures of enterotoxaemia.
7. Write etiology, mode of transmission clinical signs and line of treatment of brucellosis.
8. Write clinical signs and method of diagnosis of pneumonia.

6.1. Rabies

Rabies is a fatal viral zoonotic disease that affects all warm-blooded animals, including dogs and humans. It is caused by the *Rabies virus (RABV)*, primarily affecting the central nervous system and leading to encephalitis. Once clinical signs appear, the disease is almost invariably fatal.

Synonym

- Hydrophobia (in humans, due to fear of water).
- Lyssa or Rabid Disease.

Etiology

- Caused by the *Rabies virus (RABV)*, a member of the genus *Lyssavirus* in the family *Rhabdoviridae*.
- The virus is neurotropic, meaning it specifically targets nervous tissue.

Mode of Transmission

- 1. Direct Transmission**
 - Bite wounds: The virus is present in the saliva of infected animals and is transmitted through bites.
- 2. Non-bite Exposure**
 - Through scratches or abrasions contaminated with infected saliva.
- 3. Rare Modes**
 - Inhalation of aerosolized virus (e.g., in bat caves).
 - Organ transplantation from an infected donor (in humans).

Clinical Signs

1. Incubation Period

- Typically 1 to 3 months but can range from 10 days to over a year, depending on the site of the bite and viral load.

2. Prodromal Phase

- Behavioral changes (e.g., restlessness, anxiety, or aggression).
- Fever, itching, or pain at the bite site.

3. Excitative ("Furious") Phase

- Aggressiveness and hyperexcitability.
- Roaming and attacking objects, animals, or people without provocation.
- Excessive salivation ("foaming at the mouth").
- Hyperesthesia (sensitivity to stimuli).

4. Paralytic ("Dumb") Phase

- Progressive paralysis, beginning at the bite site.
- Dropped jaw and inability to swallow (causing drooling).
- Lethargy and coma, leading to death.

5. Outcome

- Death usually occurs within 7 to 10 days after the onset of clinical signs due to respiratory failure.

Diagnosis

1. Clinical Diagnosis

- Based on behavioral and neurological signs in endemic areas.

2. Laboratory Diagnosis

- **Direct fluorescent antibody test (DFA):** Gold standard for detecting viral antigens in brain tissue (postmortem).

- **RT-PCR:** Detects viral RNA in saliva, cerebrospinal fluid, or brain tissue.
- **Histopathology:** Identification of Negri bodies in brain tissues (postmortem).
- **Serology:** Rabies antibodies in unvaccinated animals indicate exposure.

Treatment

- No specific treatment is available once clinical signs appear.
- Supportive care is rarely effective, and euthanasia is recommended in animals with suspected rabies to prevent further suffering and transmission.

Prevention and Control Measures

1. Vaccination

- Dogs should receive routine rabies vaccination according to national or local guidelines.
- Puppies are vaccinated at 12 weeks of age, with boosters given annually or as per manufacturer recommendations.

2. Control of Stray Animals

- Reduce the population of stray dogs through humane programs, including sterilization and vaccination.

3. Quarantine

- Isolate dogs suspected of rabies for observation (10 days for domestic dogs).

4. Public Awareness

- Educate pet owners about the importance of vaccination and safe handling of animals.
- Inform about the risks of bites and the need for prompt post-exposure prophylaxis (PEP) in humans.

5. Post-exposure Measures

- Clean bite wounds immediately with soap and water.
- Seek medical attention and rabies post-exposure prophylaxis (PEP) if exposed.

6. Regulation of Pet Movement

- Enforce strict vaccination and health certification requirements for animal travel across borders.

7. Wildlife Management

- Limit contact between domestic animals and wildlife reservoirs like foxes, bats, and raccoons.

Public Health Significance

Rabies is a notifiable disease due to its zoonotic nature and fatal outcome in humans. Global efforts, led by organizations like WHO and OIE, aim to eliminate canine-mediated rabies through mass dog vaccination and public health campaigns.

6.2 Foot and Mouth Disease (FMD)

Foot and Mouth Disease (FMD) is a highly contagious viral disease affecting cloven-hoofed animals, including cattle, sheep, goats, pigs, and wild ruminants. It is caused by the *Foot-and-mouth disease virus (FMDV)*, a member of the genus *Aphthovirus* within the family *Picornaviridae*.

Etiology

The disease is caused by picorna virus, which has seven serotypes:

- O
- A
- C
- Asia 1

- SAT 1
- SAT 2
- SAT 3

Each serotype requires specific vaccines, as there is no cross-immunity between them.

Mode of transmission

1. Direct Contact

- With infected animals' saliva, vesicular fluid, feces, or urine.

2. Indirect Contact

- Contaminated equipment, clothing, vehicles, or feed.

3. Airborne Transmission

- Particularly over long distances in humid and windy conditions.

4. Carriers

- Recovered animals can carry the virus in their nasopharyngeal tissues, potentially spreading the disease.

Clinical Signs

1. In Cattle

- High fever (41°C or 105.8°F).
- Vesicles (blisters) on the tongue, gums, lips, teats, and coronary bands.
- Drooling of saliva ("stringy" saliva).
- Lameness due to painful vesicles on feet.
- Reduced milk production.
- Weight loss.
- Secondary infections and possible death in young calves due to myocarditis.

2. In Sheep and Goats

- Signs are usually milder and may include lameness and vesicles on the feet and mouth.

3. In Pigs

- Severe lameness.
- Vesicles on the snout and feet.

Diagnosis

1. Clinical Examination

- Observation of characteristic vesicles and associated clinical signs.

2. Laboratory Tests

- **Virus isolation:** Culture the virus in susceptible cell lines.
- **ELISA (enzyme-linked Immunosorbent assay):** Detects specific viral antigens or antibodies.
- **PCR (polymerase chain reaction):** Detects viral RNA.
- **Serotyping:** Determines the specific serotype.

Treatment

- There is no specific antiviral treatment for food and mouth disease.
- Symptomatic treatment includes:
 - Pain relief (analgesics).
 - Application of antiseptics or healing agents to lesions.
 - Supportive care, such as fluid therapy to prevent dehydration.

Prevention and Control Measures

1. Vaccination:

- Use of inactivated food and mouth disease vaccines specific to the prevalent serotypes.
- Regular vaccination schedules are critical in endemic regions.

2. Biosecurity Measures

- Restrict animal movement.
- Disinfect vehicles, equipment, and personnel entering farms.
- Implement quarantine for new or returning animals.

3. Eradication Strategies

- Culling infected and at-risk animals in outbreak zones.
- Proper disposal of carcasses (burning or burial).

4. Public Awareness

- Educate farmers and stakeholders about food and mouth disease signs and reporting procedures.

5. Monitoring and Surveillance

- Regular testing and monitoring of livestock.

6. International Measures

- Strict import and export regulations to prevent cross-border transmission.

6.3 Peste des Petits Ruminants (PPR)

Peste des Petits Ruminants (PPR), also known as "goat plague," is a highly contagious viral disease primarily affecting small ruminants like goats and sheep. It is characterized by fever, oral erosions, diarrhea, respiratory distress, and high morbidity and mortality rates. The disease is caused by the *Peste des Petits Ruminants Virus (PPRV)*, belonging to the genus *Morbillivirus* in the family *Paramyxoviridae*. The disease is also called "Pseudo rinderpest".

Etiology

The causative agent is Morbilli virus.

Mode of Transmission

1. Direct Contact

- Through secretions from the eyes, nose, and mouth of infected animals.
- Spread via saliva, respiratory droplets, feces, and urine.

2. Indirect Contact

- Via contaminated feed, water, bedding, or equipment.

3. Close Proximity

- The virus spreads most efficiently in situations where animals are densely housed, such as markets or shelters.

Clinical Signs

1. Incubation Period

- Typically 4-10 days.

2. Acute Symptoms

- High fever (40–41°C).
- Depression, lethargy, and loss of appetite.
- Nasal discharge (serous to mucopurulent).
- Conjunctivitis with matting of eyelids.
- Erosive lesions in the mouth, gums, and tongue leading to excessive salivation.
- Diarrhea, often profuse and foul-smelling, leading to dehydration.
- Coughing and respiratory distress in advanced stages.

3. Chronic Symptoms

- Poor growth, emaciation, and secondary bacterial infections.

4. Mortality Rate

- Can reach up to 90% in naive populations.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic signs such as oral lesions, diarrhea, and respiratory distress.

2. Laboratory Diagnosis

- **RT-PCR (reverse transcription polymerase chain reaction):** Detects viral RNA.
- **ELISA (enzyme-linked immunosorbent assay):** Identifies PPR-specific antigens or antibodies.
- **Virus isolation:** Using susceptible cell cultures.
- **Histopathology:** Erosive stomatitis and necrosis in lymphoid tissues.

Treatment

There is no specific antiviral treatment for PPR. Supportive care is essential:

1. **Antibiotics:** To control secondary bacterial infections.
2. **Rehydration Therapy:** To treat dehydration caused by diarrhea.
3. **Nutritional Support:** Including easily digestible feed.
4. **Anti-inflammatory Drugs:** To reduce fever and pain.

Prevention and Control Measures

1. Vaccination

- Use of live attenuated PPR vaccines, which provide long-lasting immunity.
- Regular mass vaccination campaigns in endemic regions.

2. Quarantine

- Isolate new or sick animals to prevent disease introduction and spread.

3. Biosecurity Measures

- Proper cleaning and disinfection of facilities, equipment, and transport vehicles.

- Control of animal movement, especially during outbreaks.

4. Culling

- In severe outbreaks, culling infected and at-risk animals may be necessary.

5. Awareness and Education

- Inform farmers about PPR clinical signs and the importance of early reporting.

6. Surveillance and Monitoring

- Continuous monitoring of susceptible populations to detect early outbreaks.

7. International Measures

- Strict import/export regulations to minimize cross-border transmission.

6.4 Swine Fever

Swine fever, also known as hog cholera, is a highly contagious viral disease affecting domestic pigs and wild boars. The disease is characterized by fever, hemorrhages, neurological signs, and immunosuppression.

Synonym

- Hog Cholera
- Classical Swine Fever (CSF)
- Plaque porcine

Etiology

The causative agent is the *Classical Swine Fever Virus (CSFV)*, a member of the genus *Pestivirus* in the family *Flaviviridae*.

Mode of Transmission

1. Direct Contact

- Through secretions and excretions (saliva, feces, urine, nasal discharge) from infected pigs.

2. Indirect Contact

- Contaminated feed, water, bedding, or equipment.
- Ingestion of raw or improperly cooked pork products containing the virus.

3. Vertical Transmission

- From infected sows to their fetuses, resulting in congenital infection or persistent carriers.

4. Fomites

- Spread through contaminated vehicles, clothing, and footwear.

The virus is stable in the environment and resistant to many disinfectants, enhancing its spread.

Clinical Signs

1. Incubation Period

- 3 to 10 days.

2. Acute Form

- High fever (41°C).
- Loss of appetite and lethargy.
- Conjunctivitis with ocular discharge.
- Cyanosis of extremities (ears, tail, and snout).
- Petechial hemorrhages on the skin and internal organs.
- Diarrhea or constipation.
- Neurological signs: Ataxia, tremors, and convulsions.
- Sudden death in severe cases.

3. Chronic Form

- Intermittent fever.
- Growth retardation and wasting.
- Chronic diarrhea.
- Mild neurological signs.

4. Congenital Infection

- Stillbirths or weak piglets.
- Persistently infected piglets may appear normal but shed the virus.

Diagnosis

1. Clinical Diagnosis

- Observation of characteristic signs such as fever, hemorrhages, and neurological symptoms.

2. Laboratory Diagnosis

- **RT-PCR (reverse transcription polymerase chain reaction):** Detects CSFV RNA.
- **ELISA (enzyme-linked immunosorbent assay):** Identifies viral antigens or antibodies.
- **Virus isolation:** In cell culture or animal models.
- **Histopathology:** Hemorrhages, necrosis in lymphoid tissues, and vascular damage.

Treatment

- There is no specific antiviral treatment for CSF.
- Supportive therapy may include:
 - **Antibiotics:** To control secondary bacterial infections.
 - **Electrolyte replacement:** To manage dehydration.

Infected pigs are usually culled to prevent further spread.

Prevention and Control Measures

1. Vaccination

- Use of live attenuated CSF vaccines in endemic areas.
- Regular vaccination campaigns are critical to maintain herd immunity.

2. Quarantine

- Isolate newly introduced pigs or pigs returning from markets.

3. Biosecurity Measures

- Restrict access to pig farms.
- Disinfect equipment, vehicles, and clothing.
- Ensure proper disposal of carcasses and waste.

4. Feeding Practices

- Prohibit feeding swill or untreated kitchen waste to pigs.

5. Culling

- Immediate slaughter of infected and exposed animals in outbreak situations.

6. Surveillance and Monitoring

- Regular testing and reporting of clinical cases.
- Traceback investigations during outbreaks.

7. International Measures

- Enforce strict import/export controls to prevent cross-border spread.

6.5 Canine Distemper

Canine distemper is a highly contagious and potentially fatal viral disease affecting domestic dogs and other carnivores, including foxes, wolves, and ferrets. It primarily affects the respiratory, gastrointestinal, and central nervous systems.

Synonym

- Hard pad Disease (due to characteristic thickening of footpads in chronic cases).

Etiology

- The disease is caused by *Paramyxo virus*
- CDV is closely related to measles and rinderpest viruses.

Mode of Transmission

1. Direct Transmission

- Inhalation of respiratory droplets from infected animals.

2. Indirect Transmission

- Contact with contaminated environments (food bowls, bedding, or water).

3. Vertical Transmission

- From infected pregnant dogs to their fetuses.

4. Viral Shedding

- Infected animals shed the virus through respiratory secretions, urine, feces, and saliva.

CDV is highly contagious but relatively fragile in the environment, being sensitive to heat, sunlight, and common disinfectants.

Clinical Signs

1. Incubation Period

- 1 to 2 weeks.

2. Acute Phase

- High fever (often biphasic).
- Nasal discharge (serous to mucopurulent).
- Conjunctivitis with ocular discharge.

- Coughing and respiratory distress.
- Vomiting and diarrhea.
- Loss of appetite and lethargy.

3. Neurological Phase

- Ataxia (lack of coordination).
- Muscle twitching or tremors.
- Seizures (chewing gum fits are characteristic).
- Paralysis or hyperesthesia (increased sensitivity to stimuli).

4. Chronic Phase

- Hyperkeratosis (thickening) of the nose and footpads ("hardpad disease").
- Enamel hypoplasia in teeth of young dogs that survive.

5. Mortality Rate

- Can exceed 50% in severe cases, especially in young, unvaccinated dogs.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic signs like fever, nasal discharge, neurological symptoms, and hardpads.

2. Laboratory Diagnosis

- **RT-PCR:** Detects viral RNA in blood, urine, or nasal swabs.
- **Immunofluorescence:** Demonstrates CDV antigens in conjunctival or nasal smears.
- **Serology:** Detects rising antibody titers in paired serum samples.
- **Histopathology:** Identifies inclusion bodies in epithelial or nervous tissues.

Treatment

There is no specific antiviral treatment for canine distemper. Management focuses on supportive care:

1. **Antibiotics:** To control secondary bacterial infections.
2. **Fluid Therapy:** To address dehydration due to diarrhea or vomiting.
3. **Nutritional Support:** Easily digestible diets and appetite stimulants if needed.
4. **Anticonvulsants:** For managing seizures (e.g., diazepam or phenobarbital).
5. **Anti-inflammatory drugs:** To reduce neurological inflammation.

Prevention and Control Measures

1. Vaccination

- Routine vaccination with modified-live virus vaccines is the most effective preventive measure.
- Puppies should receive their first dose at 6-8 weeks of age, followed by boosters every 3-4 weeks until 16 weeks, and annual revaccination thereafter.

2. Isolation

- Separate infected animals to prevent spread to healthy dogs.

3. Biosecurity Measures

- Disinfect the environment with effective agents like bleach solutions.
- Avoid sharing equipment between infected and healthy animals.

4. Hygiene Practices

- Regular cleaning of kennels, food bowls, and bedding.

5. Public Awareness

- Educate dog owners about the importance of vaccination and early signs of distemper.

6. Wildlife Management

- Limit contact between domestic dogs and wildlife reservoirs of CDV.

6.6 Rinderpest

Rinderpest, also known as "cattle plague," is a highly contagious and often fatal viral disease of cattle and other cloven-hoofed animals.

Synonym

- Cattle Plague.

Etiology

- Caused by the *Rinderpest virus (RPV)*, a member of the genus *Morbillivirus* in the family *Paramyxoviridae*.
- The virus is closely related to Peste des Petits Ruminants Virus (PPRV) and human measles virus.

Mode of Transmission

1. Direct Transmission

- Inhalation of respiratory droplets from infected animals.
- Contact with saliva, nasal discharge, or feces of infected animals.

2. Indirect Transmission

- Via contaminated feed, water, equipment, or environments.

3. Susceptible Hosts

- Domestic cattle were the primary hosts, but buffaloes, antelope, and other wild ungulates could also be infected.

Clinical Signs

1. Incubation Period

- Typically 3 to 15 days.

2. Acute Symptoms

- High fever (persistent).
- Depression, loss of appetite, and lethargy.
- Nasal and ocular discharge (serous to mucopurulent).
- Erosions and ulcers in the mouth, gums, and tongue, causing excessive salivation.
- Severe diarrhea, often bloody, leading to dehydration.
- Coughing and respiratory distress in some cases.

3. Later Symptoms

- Emaciation, weakness, and death within 6-12 days after the onset of symptoms in severe cases.

4. Mortality Rate

- Could reach up to 100%.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic signs such as fever, oral ulcers, and diarrhea in endemic areas.

2. Laboratory Diagnosis

- **RT-PCR:** Detects viral RNA in blood, nasal swabs, or tissue samples.
- **ELISA:** Identifies antibodies or viral antigens.
- **Virus Isolation:** In susceptible cell cultures.
- **Histopathology:** Lymphoid necrosis and mucosal erosions.

Treatment

- No specific antiviral treatment was available for rinderpest.
- Supportive care (hydration, antibiotics for secondary infections) could alleviate symptoms but rarely altered the course of the disease.

Prevention and Control Measures

1. Vaccination

- Mass vaccination campaigns with live attenuated vaccines were the cornerstone of rinderpest eradication.
- Immunity following vaccination lasted for life in most animals.

2. Quarantine and Movement Control

- Restrict movement of animals in outbreak areas to prevent disease spread.

3. Slaughter Policy

- Culling infected and in-contact animals during outbreaks was used to contain the virus.

4. Biosecurity Measures

- Disinfection of premises, equipment, and transport vehicles.
- Proper disposal of carcasses to prevent environmental contamination.

5. Surveillance

- Continuous monitoring for clinical signs in susceptible populations.
- Immediate reporting and investigation of suspected cases.

6. Awareness Programs

- Educating farmers and veterinary personnel about rinderpest and its signs.

Eradication Success

Rinderpest was officially declared eradicated in 2011 by the World Organization for Animal Health (OIE) and the Food and Agriculture Organization (FAO).

6.7 ORF

Orf, also known as contagious ecthyma or sore mouth, is a zoonotic, viral skin

disease primarily affecting sheep and goats. It is characterized by pustular and scabby lesions on the lips, muzzle, and other areas, particularly where the skin is damaged. Humans can contract the disease through direct contact with infected animals or contaminated materials.

Synonym

- contagious ecthyma
- sore mouth
- scabby mouth
- sheep pox

Etiology

- Caused by the *Orf virus*, a member of the genus *Parapoxvirus* in the family *Poxviridae*.
- The virus is resilient and can survive in the environment for months under favorable conditions.

Mode of Transmission

1. Direct Contact

- Contact with lesions or scabs from infected animals.
- Virus transmission through cuts, abrasions, or damaged skin.

2. Indirect Contact

- Via contaminated equipment, feed troughs, fences, or bedding.

3. Zoonotic Transmission

- Humans can contract the disease by handling infected animals or contaminated materials.

Clinical Signs

1. Incubation Period

- Typically 3 to 7 days.

2. In Sheep and Goats

- Pustular or vesicular lesions around the lips, nostrils, and mouth.
- Lesions may spread to the udder, feet, or other body parts.
- Difficulty eating, leading to reduced feed intake and weight loss.
- Secondary bacterial infections may complicate the lesions.
- Lambs and kids may develop severe infections, especially if immunocompromised.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic lesions around the mouth and nostrils in affected animals.

2. Laboratory Diagnosis

- **Electron Microscopy:** To visualize the virus in lesion material.
- **PCR (Polymerase Chain Reaction):** Detects *Orf virus* DNA.
- **Virus Isolation:** In susceptible cell cultures (rarely used).
- **Histopathology:** Reveals epidermal hyperplasia and viral inclusion bodies.

Treatment

1. In Animals

- The disease is usually self-limiting, resolving within 3 to 6 weeks.
- Apply topical antiseptics or antibiotics to prevent secondary bacterial infections.
- Provide supportive care, such as soft feeds for animals with severe oral lesions.

Prevention and Control Measures

1. Vaccination

- Live attenuated vaccines are available for sheep and goats in endemic

areas.

- Vaccinate lambs and kids around 6 to 8 weeks old.
- Only use vaccines in infected flocks, as vaccination introduces the virus into the environment.

2. Biosecurity Measures

- Isolate infected animals to prevent spread.
- Disinfect contaminated equipment, feeding troughs, and premises.
- Minimize skin injuries in animals, especially during shearing or handling.

3. Personal Protection

- Wear gloves and protective clothing when handling infected animals or contaminated materials.
- Practice good hygiene, such as washing hands thoroughly after contact.

4. Environmental Management

- Remove scabs and dispose of them safely to reduce environmental contamination.
- Maintain clean and dry housing for animals.

5. Education and Awareness

- Train farmers and animal handlers about the disease and its zoonotic potential.

Exercise

Choose the correct answer from the given alternatives.

1. What is the synonym for Rabies?
 - a. Hydrophobia
 - b. Malignant catarrhal fever
 - c. Equine encephalitis
 - d. Bovine viral diarrhea
2. Which of the following causative organism of Rabies?
 - a. *Canine parvovirus*
 - b. *Rabies virus* (Rhabdovirus family)
 - c. *Leptospira*
 - d. *Peste des Petits Ruminants virus*
3. How is Rabies transmitted?
 - a. Airborne transmission
 - b. Bite from an infected animal (usually dogs)
 - c. Fecal-oral route
 - d. Vector transmission by ticks
4. Which of the following is a common symptom of Rabies in animals?
 - a. Sudden paralysis of limbs
 - b. Excessive salivation, aggression, and difficulty swallowing
 - c. Diarrhea with blood
 - d. Lameness and joint swelling
5. How is Rabies diagnosed?
 - a. Blood culture
 - b. PCR on saliva or brain tissue
 - c. Serum ELISA for antibodies
 - d. Fecal examination for oocysts
6. What is the treatment for Rabies?
 - a. Antiviral drugs like acyclovir
 - b. No effective treatment once symptoms appear

- c. Antibiotics
- d. Fluid therapy and painkillers

7. How can Rabies be prevented?

- a. Vaccination of pets and livestock
- b. Use of acaricides
- c. Isolating infected animals
- d. Quarantine and culling of infected animals

8. What is the causative organism of Foot and Mouth Disease (FMD)?

- a. *Foot and Mouth Virus* (Picornaviridae)
- b. *Canine parvovirus*
- c. *Clostridium tetani*
- d. *Peste des Petits Ruminants virus*

9. Which of the following is the primary mode of transmission for FMD?

- a. Fecal-oral route
- b. Inhalation of aerosolized particles from infected animals
- c. Bite from infected ticks
- d. Contact with contaminated bedding

10. Which clinical sign is most commonly seen in FMD?

- a. Severe nasal discharge and pneumonia
- b. Sudden death with no clinical signs
- c. Fever, blisters, and lesions on the mouth, feet, and teats
- d. Jaundice and gastrointestinal bleeding

11. How is FMD diagnosed?

- a. Blood smear for *FMD* antibodies
- b. PCR to detect *FMD* virus

- c. ELISA for viral antigens
- d. Direct contact with symptomatic animals

12. What is the primary treatment for FMD?

- a. Vaccination during outbreaks
- b. Antibiotic therapy to control secondary infections
- c. Fluid therapy and anti-inflammatory drugs
- d. Antiviral medication

13. How can FMD be controlled?

- a. Mass culling of infected animals
- b. Regular deworming
- c. Use of vaccines and movement restrictions
- d. Antibiotic treatment

14. What is the causative organism of Peste des Petits Ruminants (PPR)?

- a. *Peste des Petits Ruminants virus* (Paramyxoviridae)
- b. *Foot and Mouth Virus*
- c. *Mycoplasma bovis*
- d. *Brucella abortus*

15. Which species is most affected by PPR?

- a. Cattle
- b. Sheep and goats
- c. Dogs
- d. Horses

16. What is the primary mode of transmission for PPR?

- a. Airborne droplets from coughing or sneezing
- b. Bite from infected ticks
- c. Fecal-oral route
- d. Contact with contaminated feed

17. What is a common clinical sign of PPR in sheep and goats?

- Severe coughing, nasal discharge, and diarrhea
- Sudden death with no signs
- Enlarged lymph nodes and skin rashes
- Lameness and joint swelling

18. How is PPR diagnosed?

- PCR to detect PPR virus
- Blood smear for *PPR* antibodies
- Serology for *Eimeria* oocysts
- ELISA for Bovine virus

19. How can PPR be prevented?

- Vaccination of susceptible animals
- Antibiotic treatment for all animals
- Isolation of sick animals only
- Improving housing conditions only

20. What is the causative organism of Swine Fever?

- Porcine reproductive and respiratory syndrome virus*
- Classical swine fever virus* (Hog cholera)
- Foot and Mouth Virus*
- Brucella suis*

21. What is the most common symptom of Swine Fever?

- Abdominal pain and vomiting
- Skin lesions, fever, and hemorrhages
- Sudden weight loss
- Jaundice and diarrhea

22. How is swine fever diagnosed?

- PCR to detect swine fever virus

- b. Fecal culture for bacteria
 - c. Blood smear for *E. coli*
 - d. ELISA for *Brucella* antibodies
- 23. How is swine fever controlled?
 - a. Vaccination and culling of infected animals
 - b. Regular deworming
 - c. Antibiotic therapy
 - d. Isolation of pigs from other species
- 24. What is the causative organism of canine distemper?
 - a. *Canine parvovirus*
 - b. *Canine distemper virus* (Paramyxoviridae)
 - c. *Brucella canis*
 - d. *E. coli*
- 25. How is canine distemper transmitted?
 - a. Direct contact with infected animal fluids
 - b. Airborne transmission
 - c. Vector-borne transmission by fleas
 - d. Fecal-oral route
- 26. What is a common clinical sign of canine distemper?
 - a. Nasal and ocular discharge, coughing, and convulsions
 - b. Diarrhea and vomiting
 - c. Skin lesions and abscesses
 - d. Sudden paralysis of limbs
- 27. What is the causative agent of rinderpest?
 - a. *Peste des Petits Ruminants virus*

- b. *Rinderpest virus* (Morbillivirus)
- c. *Brucella abortus*
- d. *Foot and Mouth Virus*

28. How is rinderpest transmitted?

- a. Airborne droplets from coughing and sneezing
- b. Vector-borne transmission via ticks
- c. Fecal-oral route
- d. Bites from infected mosquitoes

29. What is a common symptom of rinderpest?

- a. Fever, nasal discharge, and mouth ulcers
- b. Sudden weight loss and lethargy
- c. Swelling of limbs and joints
- d. Excessive salivation and abdominal pain

30. What is the causative agent of ORF (Sore Mouth)?

- a. *Orf virus* (Poxviridae)
- b. *Mycobacterium bovis*
- c. *Brucella melitensis*
- d. *Foot and Mouth Virus*

Write short answer to the following questions.

1. Write etiology, clinical signs and method of diagnosis of Rabies.
2. Write etiology, method of diagnosis and prevention and control measures of FMD.
3. Write etiology, method of diagnosis and prevention and control measures of PPR.
4. Write clinical signs and treatment of Canine distemper.

5. Write the synonym of Swine fever. How can swine fever be prevented?
6. Write clinical signs and line of treatment of Orf.
7. Write etiology and clinical signs of Rinderpest.

Write long answer to the following questions.

1. Write etiology, clinical signs, method of diagnosis and treatment of FMD.
2. Write etiology, mode of transmission, method of diagnosis and prevention and control measures of Rabies.
3. Write etiology, clinical signs, method of diagnosis and prevention and control measures of PPR.
4. Write clinical signs, mode of transmission and method of prevention of Canine distemper.
5. Write etiology, clinical signs, method of diagnosis and prevention of Swine fever.
6. Write clinical signs and line of treatment and prevention and control measures of Rinderpest.
7. Write etiology, mode of transmission clinical signs and line of treatment of Orf.

7.1 Ringworm

Ringworm, also known as dermatophytosis, is a zoonotic fungal infection affecting the skin, hair, and nails of animals and humans. It is characterized by circular, scaly lesions that may cause itching, hair loss, and crusting. The disease is caused by dermatophyte fungi and is common in cattle, sheep, goats, and other farm animals.

Synonym

- Dermatophytosis

Etiology

- Caused by dermatophyte fungi, primarily:
 - *Trichophyton verrucosum* (common in cattle).
 - *Microsporum canis* (more common in cats and dogs but can affect farm animals).
 - *Trichophyton mentagrophytes*.

Mode of Transmission

1. Direct Contact

- Contact with infected animals or humans.

2. Indirect Contact

- Via contaminated objects like grooming tools, feeders, bedding, or fencing.

3. Environmental Persistence

- Dermatophyte spores can survive in the environment for months, promoting transmission.

4. Susceptible Populations

- Young, stressed, or immunocompromised animals are more susceptible.

Clinical Signs

1. In Cattle, Sheep, and Goats

- Circular, hairless lesions with scaly or crusty skin.
- Lesions commonly occur on the head, neck, and shoulders.
- In mild cases, itching may be absent; severe cases may cause discomfort and secondary bacterial infections.

2. In humans (Zoonosis)

- Red, scaly, itchy, circular lesions on exposed skin areas.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic skin lesions in affected animals.

2. Laboratory Diagnosis

- **Microscopic examination:** Skin scrapings viewed under a microscope for fungal hyphae.
- **Fungal culture:** Growth of dermatophytes on Sabouraud dextrose agar.
- **Wood's lamp examination:** Some *Microsporum* species fluoresce under UV light.
- **PCR (polymerase chain reaction):** Rapid detection of dermatophyte DNA.

Treatment

1. Topical Antifungals

- Apply antifungal ointments or sprays containing miconazole, clotrimazole, or enilconazole to affected areas.

2. Systemic Antifungals

- Griseofulvin or itraconazole in severe or widespread infections (veterinary prescription required).

3. Environmental Management

- Disinfect contaminated equipment and premises with fungicidal solutions.

4. Supportive Care

- Ensure proper nutrition and minimize stress to boost immunity.

Prevention and Control Measures

1. Hygiene and Biosecurity

- Regularly clean and disinfect animal housing and equipment.
- Avoid overcrowding to reduce stress and spread.

2. Isolation

- Isolate infected animals to prevent transmission to healthy individuals.

3. Environmental Control

- Treat contaminated bedding, grooming tools, and fences with antifungal disinfectants.

4. Vaccination

- Vaccines for dermatophytosis are available in some regions for cattle.

5. Education

- Train farm workers on the zoonotic nature of ringworm and emphasize personal hygiene.

6. Personal Protection

- Use gloves and protective clothing when handling infected animals or cleaning contaminated areas.

7. Regular Monitoring

- Conduct regular inspections for signs of dermatophytosis, especially in young or newly acquired animals.

7.2 Mycotoxicosis

Mycotoxicosis refers to a group of diseases caused by the ingestion of feed or forage contaminated with toxic secondary metabolites (mycotoxins) produced by certain fungi, including *Aspergillus*, *Fusarium*, and *Penicillium* species. These toxins cause a variety of adverse health effects, including reduced productivity, organ damage, immunosuppression, and in severe cases, death.

Synonym

- Fungal Toxicosis
- Mycotoxin Poisoning

Etiology

Mycotoxicosis is caused by ingestion of mycotoxins produced by certain fungi under specific environmental conditions. Common mycotoxins include:

1. **Aflatoxins** (*Aspergillus flavus* and *Aspergillus parasiticus*).
2. **Ochratoxins** (*Aspergillus* and *Penicillium* species).
3. **Zearalenone** (*Fusarium* species).
4. **Fumonisins** (*Fusarium verticillioides* and *Fusarium proliferatum*).
5. **Trichothecenes** (e.g., deoxynivalenol or DON, T-2 toxin from *Fusarium*).
6. **Ergot Alkaloids** (*Claviceps purpurea* infecting grains).

Mode of Transmission

1. Ingestion

- Consuming contaminated feed, forage, or silage.
- Toxins are often heat-stable and persist even in processed feeds.

2. Environmental Factors

- High humidity and temperatures during storage or harvesting favor fungal growth.

3. Susceptibility

- Young, stressed, or immunocompromised animals are more affected.

Clinical Signs

1. General Signs

- Reduced feed intake and growth rate.
- Diarrhea or gastrointestinal disturbances.
- Reduced milk yield or egg production.
- Immunosuppression and increased susceptibility to infections.

2. Specific Signs Based on Toxin Type

- **Aflatoxins:** Liver damage, jaundice, reduced fertility, and carcinogenic effects.
- **Ochratoxins:** Kidney damage, reduced growth, and dehydration.
- **Zearalenone:** Reproductive disorders like infertility, abortions, or vulvar swelling.
- **Fumonisins:** Pulmonary edema in pigs; liver and kidney damage in other species.
- **Trichothecenes (DON):** Vomiting, feed refusal, and oral ulcers.
- **Ergot alkaloids:** Gangrene of extremities, hyperthermia, and reduced lactation.

Diagnosis

1. History and Clinical Signs

- Correlation with poor feed quality and characteristic symptoms.

2. Feed Analysis

- Testing feed or forage for specific mycotoxins using methods like:
 - Thin-layer chromatography (TLC).
 - Enzyme-linked immunosorbent assay (ELISA).
 - High-performance liquid chromatography (HPLC).

3. Postmortem Examination

- Liver or kidney damage and gastrointestinal lesions indicative of mycotoxin exposure.

4. Laboratory Tests

- Detection of specific mycotoxins in animal tissues or biological samples.

Treatment

1. Removal of Contaminated Feed

- Replace with fresh, uncontaminated feed.

2. Supportive Therapy

- **Activated charcoal:** Binds mycotoxins in the gastrointestinal tract.
- **Liver protectants:** Such as vitamins.
- **Electrolytes and fluids:** To manage dehydration and support metabolism.

3. Mycotoxin Binders

- Additives like bentonite clay, activated carbon, or synthetic binders to reduce toxin absorption.

4. Antibiotics and Anti-inflammatory Drugs

- For secondary bacterial infections or inflammatory damage.

Prevention and Control Measures

1. Feed Management

- Harvest crops at appropriate maturity.
- Ensure proper drying of grains and forages to prevent fungal growth.
- Use airtight silos or sealed storage to minimize contamination.

2. Regular Inspection

- Monitor feed and storage areas for fungal growth and contamination.

3. Mycotoxin Binders in Feed

- Incorporate binders in feed as a preventive measure.

4. Improved Storage Conditions

- Maintain low moisture levels and temperature in storage areas.
- Use preservatives like organic acids (propionic acid) to inhibit fungal growth.

5. Crop Rotation and Resistant Varieties

- Use crop rotation to reduce fungal spore buildup.
- Plant resistant crop varieties where available.

6. Education

- Train farmers and feed handlers on the risks and management of mycotoxins.

7. Testing and Quality Control

- Regularly test feed ingredients and finished feed for mycotoxins.

Exercise

Choose the correct answer from the given alternatives.

1. What is the causative organism of ringworm?
 - a. *Trichophyton*
 - b. *Aspergillus*
 - c. *Toxoplasma gondii*
 - d. *Brucella abortus*
2. Which of the following is the most common symptom of ringworm in animals?
 - a. Excessive salivation
 - b. Circular, scaly patches of hair loss with red borders
 - c. Sudden death
 - d. Diarrhea and weight loss
3. How is ringworm primarily transmitted?
 - a. Fecal-oral route
 - b. Contact with infected animals or contaminated surfaces
 - C. Airborne droplets
 - d. Ingestion of contaminated food
4. Which of the following is used for the diagnosis of ringworm?
 - a. Fecal culture
 - b. Skin scraping and fungal culture
 - c. PCR for bacterial pathogens
 - d. Blood smear
5. What is the primary treatment for ringworm?
 - a. Antifungal topical creams and systemic antifungal drugs
 - b. Antiviral medications
 - c. Antibiotics for secondary infections
 - d. Steroids

6. How can ringworm be prevented and controlled?
 - a. Proper sanitation and disinfection of contaminated surfaces
 - b. Regular deworming
 - c. Vaccination of animals
 - d. Isolation of infected animals only
7. **What is mycotoxicosis?**
 - a. Disease caused by fungal infections
 - b. Poisoning caused by toxic substances produced by fungi
 - c. Disease caused by bacterial toxins
 - d. Disease caused by parasitic infestations
8. **Which of the following is a common source of mycotoxins in livestock?**
 - a. Contaminated feed and crops
 - b. Airborne bacterial spores
 - c. Infected animal saliva
 - d. Water from contaminated wells

Write short answer to the following questions.

1. Write the etiology and clinical signs of Mycotoxicosis.

Write long answer to the following questions.

1. Write the etiology, clinical signs, diagnosis, treatment and preventive measures of Ringworm.

8.1 Milk Fever

Milk fever, also known as parturient paresis, is a metabolic disorder primarily affecting dairy cows around calving. It is caused by acute hypocalcemia (low blood calcium levels), which disrupts normal neuromuscular function, leading to muscle weakness, inability to stand, and, if untreated, potentially death.

Synonym

- Parturient Paresis
- Hypocalcemia

Etiology

1. Primary Cause

- Inadequate levels of calcium in the blood due to the sudden demand for calcium for milk production post-calving.

2. Predisposing Factors

- High-producing dairy cows, particularly older animals.
- Diets with excess potassium or low calcium during the dry period.
- Breed predisposition (e.g., Jersey cows are more susceptible).
- Imbalance in the parathyroid hormone and vitamin D response.

Clinical Signs

1. Stage 1 (early signs)

- Muscle tremors and restlessness.

- Staggering gait.
- Reduced appetite and mild depression.

2. Stage 2 (moderate signs)

- Inability to stand (recumbency).
- Cold ears and extremities due to poor circulation.
- Dry muzzle and slow heartbeat (50–70 bpm).
- Dilated pupils and sluggish reflexes.

3. Stage 3 (severe signs)

- Lateral recumbency and complete inability to rise.
- Severe depression or coma.
- Bloat due to impaired rumen motility.
- Death within hours if untreated.

Diagnosis

1. Clinical Diagnosis

- Based on history of calving and characteristic clinical signs.

2. Laboratory Diagnosis

- Blood calcium levels below 8.0 mg/dL confirm hypocalcemia.
- Blood tests may also reveal hypophosphatemia and hypomagnesemia.

3. Differential Diagnosis

- Must be differentiated from other post-calving conditions like ketosis, mastitis, or downer cow syndrome.

Treatment

1. Intravenous Calcium Therapy

- Administer calcium borogluconate intravenously (500 mL of a 23% solution) slowly to avoid cardiac complications.

- Monitor heart rate during administration for signs of bradycardia or arrhythmia.

2. Subcutaneous Calcium

- Can be given after IV treatment to maintain blood calcium levels.

3. Supportive Care

- Ensure the animal is kept in a comfortable position to prevent bloating.
- Provide access to water and easily digestible feed once the animal can stand.

4. Other Minerals

- Administer phosphorus or magnesium if deficiency is suspected.

Prevention and Control Measures

1. Dietary Management During the Dry Period

- Feed a low-calcium diet in the late dry period to stimulate the parathyroid gland and improve calcium mobilization.
- Use anionic salts to create a negative dietary cation-anion balance (DCAB), which enhances calcium absorption.

2. Supplementation

- Provide calcium supplements (oral or injectable) to high-risk cows at calving.

3. Monitor High-producing Cows

- Closely monitor older and high-producing cows for early signs of milk fever.

4. Breed Selection

- Consider the susceptibility of certain breeds and select animals with lower risk factors for hypocalcemia.

5. Regular Testing

- Test forage and feed to ensure an appropriate mineral balance.

6. Educating Farmers

- Train farmers to recognize early signs and manage pre-calving nutrition effectively.

8.2 Ketosis

Ketosis is a metabolic disorder occurring primarily in high-producing dairy cows during early lactation. It is characterized by elevated levels of ketone bodies in the blood, urine, and milk, resulting from negative energy balance. This occurs when energy demands for milk production exceed dietary intake, leading to excessive fat mobilization and incomplete oxidation in the liver.

Synonym

- Acetonemia
- Ketonemia

Etiology

1. Primary Ketosis

- Insufficient dietary energy intake to meet the energy demands of milk production.
- Commonly seen in early lactation.

2. Secondary Ketosis

- Result of underlying conditions (e.g., displaced abomasum, mastitis, metritis) that reduce feed intake.

3. Pathophysiology

- Negative energy balance → Mobilization of body fat → Excessive non-esterified fatty acids (NEFAs) → Accumulation of ketone bodies (β -hydroxybutyrate, acetone, acetoacetate) due to incomplete oxidation in the liver.

Clinical Signs

1. Subclinical Ketosis

- No obvious clinical signs but reduced milk yield.
- Poor reproductive performance.

2. Clinical Ketosis

- Reduced appetite, particularly for grain.
- Weight loss and body condition score (BCS) decline.
- Decreased milk production.
- Sweet or acetone-like odor on breath, milk, or urine.
- Lethargy and dullness.
- Constipation or reduced rumen motility.
- Neurological signs in severe cases (nervous ketosis):
 - Hyperexcitability.
 - Circling, head pressing, or ataxia.

Diagnosis

1. History and Clinical Signs

- Recent calving with reduced feed intake and milk production.

2. Laboratory Tests

- **Blood ketone levels:** Elevated β -hydroxybutyrate (>1.2 – 1.4 mmol/L in subclinical ketosis; >3 mmol/L in clinical ketosis).
- **Urine ketone tests:** Positive dipstick test for ketones.
- **Milk ketones:** Can be detected with specialized tests.

3. Differential Diagnosis

- Differentiate from other post-calving disorders such as hypocalcemia, displaced abomasum, or hepatic lipidosis.

Treatment

1. Restore Energy Balance

- Provide energy-dense feed such as propylene glycol (oral drench, 200–300 mL/day) to increase glucose precursors.
- Intravenous glucose (50% dextrose solution, 500 mL) for immediate energy.

2. Insulin Therapy

- May be used to enhance glucose utilization and reduce fat mobilization.

3. Corticosteroids

- Dexamethasone or prednisolone to stimulate gluconeogenesis.

4. Supportive Care

- Adequate hydration and correction of any secondary conditions.

5. Vitamin Supplementation

- Vitamin B12 to support liver function and energy metabolism.

Prevention and Control Measures

1. Nutritional Management

- Ensure a balanced ration during the transition period (3 weeks pre- and post-calving).
- Provide adequate energy and protein in the diet to meet lactational demands.
- Include rumen-protected fats to increase energy density.

2. Body Condition Management

- Avoid over-conditioning during the dry period (optimal BCS of 3–3.5 out of 5).

3. Feed Additives

- Use of niacin, monensin, or choline supplements to improve energy metabolism and reduce ketone production.

4. Monitoring

- Regular testing of blood, milk, or urine ketones in early lactation cows.

5. Reduce Stress

- Minimize environmental and social stressors during the transition period.

6. Early Detection and Treatment

- Educate farm personnel to recognize early signs of ketosis and take prompt action.

8.3 Vitamin and Mineral Deficiencies

8.3.1 Deficiency symptoms of Fat Soluble Vitamins

1. Vitamin A (Retinol)

- **Functions:** Essential for vision, immune function, reproduction, and maintenance of epithelial tissues.
- **Deficiency Symptoms**
 - **Eyes:** Night blindness, xerophthalmia (dryness of the cornea), corneal ulceration, and blindness.
 - **Skin and mucous membranes:** Dry, scaly skin; keratinization of epithelial tissues.
 - **Reproductive issues:** Infertility in both sexes; abortion or stillbirth in pregnant animals.
 - **Growth and development:** Stunted growth and increased susceptibility to infections.
 - **Neurological issues:** Incoordination and convulsions in severe cases.

2. Vitamin D (Cholecalciferol/D3 and Ergocalciferol/D2)

- **Functions:** Regulates calcium and phosphorus metabolism, supporting bone growth and maintenance.

- **Deficiency Symptoms**
- **Bone Disorders**
 - Rickets in young animals (soft, deformed bones, bowed legs).
 - Osteomalacia in adults (bone weakness and fractures).
- **Muscle issues:** Weakness and poor growth.
- **Milk fever:** Increased susceptibility in lactating animals.
- **Egg production (poultry):** Soft-shelled or misshapen eggs.

3. Vitamin E (Tocopherol)

- **Functions:** Antioxidant protecting cell membranes, supports immune function, and prevents oxidative damage.
- **Deficiency Symptoms**
- **Muscle Disorders**
 - Nutritional muscular dystrophy (white muscle disease) in calves and lambs.
 - Mulberry heart disease in pigs.
- **Reproductive issues:** Infertility or embryonic death.
- **Immune dysfunction:** Increased susceptibility to infections.
- **Neurological issues:** Ataxia or incoordination in severe cases.

4. Vitamin K (Phylloquinone/K1 and Menaquinone/K2)

- **Functions:** Essential for blood clotting and bone metabolism.
- **Deficiency symptoms**
 - **Hemorrhages:** Prolonged clotting time, leading to excessive bleeding from minor injuries.
 - **Poor bone health:** Weak bones and fractures.
 - **In poultry:** Subcutaneous hemorrhages, reduced hatchability, and poor egg quality.

8.3.2 Deficiency symptoms of Water Soluble Vitamins

1. Vitamin B Complex

Vitamin B1 (Thiamine)

- **Functions:** Essential for carbohydrate metabolism and nerve function.
- **Deficiency Symptoms**
- **Nervous Disorders**
 - Polioencephalomalacia (PEM) in ruminants (blindness, head pressing, incoordination, opisthotonus).
- **Appetite loss:** Anorexia and poor feed intake.
- **Muscle weakness:** Weakness and lethargy.
- **Cardiovascular issues:** Bradycardia or irregular heartbeat in severe cases.
- **Poultry:** Star gazing posture

Vitamin B2 (Riboflavin)

- **Functions:** Key for energy production and cellular respiration.
- **Deficiency Symptoms**
 - **Skin and mucous membranes:** Dermatitis, cracked hooves, and stomatitis.
 - **Growth issues:** Poor growth and feed efficiency.
 - **Eye problems:** Cataracts or conjunctivitis in severe cases.
 - **Poultry:** Curled toe paralysis in chicks.

Vitamin B3 (*Niacin*)

- **Functions:** Crucial for energy metabolism and maintaining healthy skin.
- **Deficiency Symptoms**
 - **Digestive issues:** Diarrhea and loss of appetite.
 - **Skin lesions:** Dermatitis and rough skin.

- **Growth issues:** Reduced weight gain.
- **Poultry:** Black tongue and enlargement of joints in chicks.

Vitamin B5 (*Pantothenic Acid*)

- **Functions:** Supports energy metabolism and hormone synthesis.
- **Deficiency Symptoms**
 - **Growth issues:** Stunted growth and reduced productivity.
 - **Skin and coat issues:** Rough hair coat and scaly skin.
 - **Poultry:** Dermatitis, crusty skin around the beak, and poor feather quality.

Vitamin B6 (*Pyridoxine*)

- **Functions:** Involved in amino acid metabolism and neurotransmitter synthesis.
- **Deficiency Symptoms**
 - **Neurological issues:** Convulsions, nervousness, and ataxia.
 - **Poor growth:** Reduced weight gain.
 - **Egg production (poultry):** Decreased egg quality and hatchability.

Vitamin B7 (Biotin)

- **Functions:** Vital for carbohydrate, fat, and protein metabolism.
- **Deficiency symptoms**
 - **Hoof disorders:** Cracked hooves and lameness in cattle.
 - **Skin issues:** Dermatitis and alopecia.
 - **Reproductive issues:** Reduced fertility.

Vitamin B9 (Folic Acid)

- **Functions:** Crucial for DNA synthesis and red blood cell formation.
- **Deficiency Symptoms**
 - **Anemia:** Macrocytic or megaloblastic anemia.

- **Poor growth:** Stunted growth and reduced productivity.
- **Reproductive issues:** Infertility or fetal abnormalities.

Vitamin B12 (*Cynacobalamin*)

- **Functions:** Important for red blood cell production and nerve function.
- **Deficiency Symptoms**
 - **Anemia:** Weakness and lethargy.
 - **Neurological Issues:** Incoordination and nerve damage.
 - **Poor Growth:** Reduced weight gain and productivity.

2. Vitamin C (*Ascorbic Acid*)

- **Functions:** Antioxidant and supports immune function, collagen synthesis, and iron absorption.
- **Deficiency Symptoms**
 - **Weak immune system:** Increased susceptibility to infections.
 - **Poor wound healing:** Delayed recovery from injuries.
 - **Scurvy:** Swollen joints, bleeding gums, and loose teeth (primarily in species that require dietary Vitamin C, such as guinea pigs, not typically in ruminants).

8.3.3 Deficiency Symptoms of Minerals

1. Calcium (Ca)

- **Functions:** Bone and teeth formation, muscle contraction, nerve function, and milk production.
- **Deficiency Symptoms**
 - **Skeletal issues:** Rickets in young animals; osteomalacia in adults.
 - **Milk fever (hypocalcemia):** Muscle tremors, recumbency, and inability to stand (common in lactating animals).
 - **Reduced productivity:** Poor growth and decreased milk production.
 - **Reproductive Issues:** Delayed estrus and retained placenta.

2. Phosphorus (P)

- **Functions:** Bone and teeth formation, energy metabolism (ATP), and reproduction.
- **Deficiency Symptoms**
 - **Bone disorders:** Rickets or osteomalacia, similar to calcium deficiency.
 - **Poor appetite:** Reduced feed intake and pica (eating non-food items).
 - **Reproductive issues:** Irregular estrus cycles, infertility, and poor conception rates.
 - **Growth issues:** Slow growth and reduced weight gain.

3. Magnesium (Mg)

- **Functions:** Enzyme activation, muscle and nerve function, and energy metabolism.
- **Deficiency Symptoms**
 - **Grass tetany (hypomagnesemia):** Muscle tremors, staggering, convulsions, and death (common in grazing animals).
 - **Poor growth:** Reduced feed efficiency and productivity.
 - **Nervousness:** Increased excitability and restlessness.

4. Sodium (Na)

- **Functions:** Maintains osmotic balance, nerve impulses, and muscle contractions.
- **Deficiency Symptoms**
 - **Reduced appetite:** Poor feed intake and growth.
 - **Pica:** Eating soil or other non-food materials.
 - **Reproductive issues:** Poor fertility and delayed estrus.
 - **Dehydration symptoms:** Weight loss and decreased milk production.

5. Potassium (K)

- **Functions:** Osmotic balance, muscle contractions, and nerve function.
- **Deficiency Symptoms**
 - **Muscle weakness:** Reduced muscle tone and stiffness.
 - **Reduced milk production:** Particularly in lactating animals.
 - **Nervous disorders:** Lethargy and incoordination.
 - **Poor appetite:** Reduced feed intake and slow growth.

6. Sulfur (S)

- **Functions:** Component of sulfur-containing amino acids (cysteine, methionine) and vitamins (biotin, thiamine).
- **Deficiency Symptoms**
 - **Poor coat and hoof quality:** Brittle hooves and dull, rough hair.
 - **Reduced microbial protein synthesis:** In ruminants, leading to poor feed utilization.
 - **Growth issues:** Slow weight gain and low productivity.

7. Chlorine (Cl)

- **Functions:** Maintains osmotic balance, acid-base regulation, and digestive function (HCl in the stomach).
- **Deficiency Symptoms**
 - **Dehydration symptoms:** Reduced water balance and growth.
 - **Metabolic alkalosis:** Respiratory distress and reduced feed intake.
 - **Digestive issues:** Poor digestion and low productivity.

8. Iron (Fe)

- **Functions:** Component of hemoglobin, involved in oxygen transport and cellular respiration.
- **Deficiency Symptoms**

- **Anemia:** Pale mucous membranes, lethargy, and reduced growth.
- **Weakness:** Reduced stamina and exercise intolerance.
- **Reproductive issues:** Poor fertility and decreased milk production.
- **Piglets:** Particularly susceptible, leading to "thumps" (rapid breathing).

9. Copper (Cu)

- **Functions:** Enzyme cofactor, important for melanin production, iron absorption, and connective tissue formation.
- **Deficiency Symptoms**
 - **Coat changes:** Dull, depigmented hair or wool (black turns reddish).
 - **Bone disorders:** Fragile bones and joint abnormalities.
 - **Anemia:** Microcytic anemia due to poor iron metabolism.
 - **Reproductive issues:** Infertility and poor estrus.
 - **Neurological signs:** Swayback (enzootic ataxia) in lambs.

10. Zinc (Zn)

- **Functions:** Skin integrity, immune function, enzyme activity, and reproduction.
- **Deficiency Symptoms**
 - **Skin disorders:** Parakeratosis (thick, crusty skin), especially in pigs.
 - **Poor growth:** Stunted growth and poor feed efficiency.
 - **Reproductive issues:** Reduced fertility and delayed testicular development in males.
 - **Hoof disorders:** Cracked hooves and lameness.

11. Manganese (Mn)

- **Functions:** Bone formation, reproduction, and enzyme activation.

- **Deficiency Symptoms**

- **Skeletal issues:** Lameness, limb deformities, and "slipped tendon" (perosis) in poultry.
- **Reproductive problems:** Delayed estrus, low conception rates, and reduced hatchability in poultry.
- **Poor growth:** Reduced feed intake and weight gain.

12. Selenium (Se)

- **Functions:** Antioxidant (with Vitamin E), involved in muscle function and immune response.

- **Deficiency Symptoms**

- **Muscle disorders:** White muscle disease (nutritional muscular dystrophy) in young animals.
- **Poor growth and weakness:** Reduced feed efficiency and lethargy.
- **Reproductive issues:** Retained placenta and infertility.
- **Immune dysfunction:** Increased susceptibility to infections.

13. Iodine (I)

- **Functions:** Synthesis of thyroid hormones (thyroxine, T3).

- **Deficiency Symptoms**

- **Goiter:** Enlarged thyroid gland.
- **Reproductive issues:** Stillbirths, abortions, and weak offspring.
- **Poor growth:** Stunted development and low weight gain.
- **Hair issues:** Dry, rough coat and alopecia.

Exercise

Choose the correct answer from the given alternatives.

1. What is the primary cause of milk fever in dairy cows?
a. Magnesium deficiency b. Calcium deficiency
c. Phosphorus excess d. Potassium toxicity

2. Which group of cows is most at risk of developing milk fever?
a. Young heifers b. Early lactation cows
c. Late lactation cows d. Pregnant cows

3. What is the most common clinical sign of milk fever?
a. Increased appetite
b. Hyperactivity
c. Muscle tremors and inability to stand
d. Increased milk production

4. What is the best method to prevent milk fever in dairy cows?
a. Increase dietary calcium before calving
b. Reduce dietary calcium before calving
c. Increase phosphorus intake
d. Provide extra vitamin A

5. Which treatment is most effective for milk fever?
a. Oral glucose b. Intravenous calcium borogluconate
c. Antibiotics d. Magnesium supplements

6. What is the primary cause of ketosis in dairy cows?
a. Excess energy intake
b. Negative energy balance due to high milk production
c. Calcium deficiency
d. Protein deficiency

7. Which of the following is a common sign of ketosis?
 - a. Diarrhea
 - b. Decreased appetite and weight loss
 - c. Fever
 - d. Lameness
8. Which substance accumulates in the blood during ketosis?
 - a. Calcium
 - b. Ketone bodies
 - c. Insulin
 - d. Urea
9. What is the best method to diagnose ketosis?
 - a. Measure milk yield
 - b. Blood or urine test for ketone levels
 - c. Physical examination
 - d. Body condition scoring
10. Which of the following is a treatment for ketosis?
 - a. Intravenous glucose and propylene glycol
 - b. Calcium injections
 - c. Magnesium sulfate
 - d. Antibiotics
11. Which vitamin deficiency causes night blindness in cattle?
 - a. Vitamin D
 - b. Vitamin A
 - c. Vitamin E
 - d. Vitamin B12
12. What is a common symptom of vitamin D deficiency in animals?
 - a. Rickets or osteomalacia
 - b. Night blindness
 - c. Muscle tremors
 - d. Poor appetite
13. White muscle disease in calves and lambs is caused by a deficiency of which vitamin?
 - a. Vitamin A
 - b. Vitamin E
 - c. Vitamin D
 - d. Vitamin B1

14. Which vitamin deficiency leads to polioencephalomalacia (PEM) in ruminants?

a. Vitamin B1 (Thiamine) b. Vitamin C
c. Vitamin K d. Vitamin B12

15. Grass tetany in cattle is caused by a deficiency of which mineral?

a. Calcium b. Magnesium
c. Potassium d. Phosphorus

16. Which mineral deficiency is linked to goiter in farm animals?

a. Iron b. Iodine c. Zinc d. Copper

17. What is the main clinical sign of selenium deficiency in livestock?

a. Goiter b. White muscle disease
c. Anemia d. Lameness

18. Which mineral deficiency causes pica (eating non-food items) in cattle?

a. Calcium b. Phosphorus
c. Copper d. Magnesium

19. Which mineral deficiency commonly caused Anemia in piglets?

a. Zinc b. Iron c. Selenium d. Iodine

20. Which mineral deficiency can result in swayback in lambs?

a. Magnesium b. Copper
c. Selenium d. Iodine

Write short answer to the following questions.

1. What is the primary cause of milk fever in dairy cattle? Write the clinical signs of milk fever.
2. Define ketosis. Write about the prevention and control measures of ketosis.
3. Write the treatment and prevention and control measures of milk fever.

4. Write short notes on

- Deficiency symptoms of Vitamin A
- Deficiency symptoms of Vitamin D
- Deficiency symptoms of Vitamin E
- Deficiency symptoms of Vitamin K
- Deficiency symptoms of Vitamin C
- Deficiency symptoms of Calcium
- Deficiency symptoms of Phosphorus
- Deficiency symptoms of Zinc
- Deficiency symptoms of Copper
- Deficiency symptoms of Magnesium
- Deficiency symptoms of Selenium
- Deficiency symptoms of Iron
- Deficiency symptoms of Iodine

Write long answer to the following questions.

- Explain the deficiency symptoms of fat soluble vitamins.
- Explain the deficiency symptoms of water soluble vitamins.
- Write the etiology, method of diagnosis, treatment and prevention and control measures of milk fever.
- Write the etiology, method of diagnosis, treatment and prevention and control measures of Ketosis.

9.1 Bacterial Diseases of Poultry

9.1.1 Fowl Cholera

Fowl cholera is a contagious bacterial disease affecting domestic and wild birds. It is caused by *Pasteurella multocida* and manifests as acute septicemia or chronic localized infections, leading to significant economic losses in poultry production.

Synonym: Avian pasteurellosis

Etiology:

- **Causative agent:** *Pasteurella multocida* (a Gram-negative, facultatively anaerobic bacterium).

Host range: Chickens, turkeys, ducks, geese, and wild birds.

Mode of Transmission

- **Direct contact:** Between infected and healthy birds through nasal secretions, saliva, or excreta.
- **Aerosol transmission:** Via respiratory droplets in close confinement.
- **Contaminated environment:** Feed, water, and equipment can harbor the bacteria.
- **Carriers:** Chronically infected birds can shed bacteria intermittently.
- **Wild birds and rodents:** Serve as reservoirs and vectors of infection.

Clinical Signs

1. Acute Form

- Sudden death without prior symptoms.

- Fever, depression, cyanosis of comb and wattles.
- Mucous discharge from the mouth or nostrils.
- Greenish or yellowish diarrhea.
- High mortality rates.

2. Chronic Form

- Swollen wattles, joints, and footpads.
- Lameness due to joint infections.
- Poor egg production in laying birds.
- Localized abscesses in the respiratory tract or other tissues.

Diagnosis

1. Clinical Diagnosis

- Based on sudden mortality, septicemia, and characteristic lesions.

2. Laboratory Diagnosis

- **Microscopy:** Gram-negative bipolar-staining bacteria observed in blood smears or tissue samples.
- **Culture:** Isolation of *P. multocida* on blood agar or other enriched media.
- **Biochemical tests:** Identifies bacterial strain.
- **Molecular methods:** PCR for detecting *P. multocida* DNA.
- **Serology:** ELISA or agglutination tests to detect specific antibodies.

Treatment

• Antibiotics

- Penicillins, tetracyclines, sulfonamides, and fluoroquinolones are effective if administered early.
- **Caution:** Resistance can develop; sensitivity testing is recommended.

- **Supportive Care**
 - Provide electrolytes and easily digestible feed to reduce stress.

Prevention and Control Measures

1. Vaccination

- **Inactivated vaccines:** Provide good protection for commercial flocks.
- **Live attenuated vaccines:** Offer long-lasting immunity but require careful handling.

2. Biosecurity Measures

- Isolate new birds before introduction to the flock.
- Maintain strict hygiene in poultry houses.
- Disinfect equipment, waterers, and feeders regularly.
- Control wild birds, rodents, and other potential carriers.

3. Management Practices

- Avoid overcrowding to reduce stress and disease spread.
- Ensure proper ventilation in poultry houses.
- Provide balanced nutrition to strengthen immunity.

4. Culling

- Remove and dispose of severely affected birds promptly to prevent further spread.

5. Surveillance

- Monitor flocks regularly for early detection and control of outbreaks.

9.1.2 Pullorum Disease

Pullorum disease is a highly contagious bacterial infection affecting poultry, primarily chicks, and is caused by *Salmonella pullorum*. It leads to septicemia, high mortality rates, and can affect chickens, turkeys, and other avian species. The disease is also referred to as bacillary white diarrhea due to the characteristic white droppings seen in infected birds.

Synonym: Bacillary white diarrhea, Salmonellosis

Etiology

- **Causative agent:** *Salmonella pullorum* (a Gram-negative, facultatively anaerobic bacterium).
- **Host range:** Primarily chickens, but also turkeys and other poultry species.

Mode of Transmission

- **Vertical Transmission**
 - Infected hens can transmit the bacteria to their offspring through eggs (transovarial transmission), which is the most common route of infection in young birds.
- **Horizontal Transmission**
 - Through contaminated feed, water, bedding, or equipment.
 - Direct contact between infected and healthy birds, including fecal-oral transmission.
 - Infected birds shedding the bacteria in their feces can contaminate the environment, infecting other birds.

Clinical Signs

1. In Chicks

- **Sudden mortality:** Death in young chicks within the first few days of life.
- **White diarrhea:** Characteristic watery, white droppings.
- **Weakness and lethargy:** Infected chicks may be weak and show signs of depression.
- **Dehydration:** Due to diarrhea and reduced feeding.
- **Stunted growth:** Chicks that survive may have poor growth rates.

2. In Adult Birds

- **Chronic carrier state:** Adult birds may carry the bacteria without showing symptoms, but they shed the bacteria, spreading the infection to others.
- **Decreased egg production:** Infected hens may experience a reduction in egg production and hatchability.
- **Respiratory signs:** Mild symptoms such as coughing or nasal discharge, though less common than in chicks.

Diagnosis

1. Clinical Diagnosis

- Based on the characteristic white diarrhea and high mortality in chicks.
- History of egg-borne transmission and outbreaks in young birds can be suggestive of pullorum disease.

2. Laboratory Diagnosis

- **Bacterial culture:** Isolation of *Salmonella pullorum* from feces, blood, or internal organs on selective media like MacConkey or Salmonella-specific agar.
- **PCR (polymerase chain reaction):** Molecular testing to detect *S. pullorum* DNA.
- **Serological tests:** Agglutination tests (such as the tube agglutination test) or ELISA to detect antibodies against *Salmonella pullorum*.
- **Histopathology:** Examination of tissue samples may reveal characteristic lesions of septicemia.

Treatment

- **Antibiotics**
 - *Salmonella pullorum* is susceptible to antibiotics such as tetracycline, streptomycin, or sulfonamides.
 - Treatment should be initiated as soon as the disease is suspected,

especially in the early stages of infection.

- Antibiotics can help reduce mortality but do not eradicate the bacteria from carrier birds.
- **Supportive Care**
 - Provide electrolytes and fluids to maintain hydration and reduce the effects of diarrhea.
 - Improve feed and water quality to support the health of affected birds.

Prevention and Control Measures

1. Vaccination

- **Live attenuated vaccines:** Available for pullorum disease and used to immunize breeder flocks to reduce the transmission of *S. pullorum* through eggs.
- **Bacterin vaccines:** Can be used to control the disease in areas where pullorum is endemic.

2. Biosecurity Measures

- **Quarantine:** New birds should be quarantined and tested for *Salmonella pullorum* before introducing them into a flock.
- **Egg disinfection:** Disinfect eggs before incubation to prevent transovarial transmission.
- **Environmental hygiene:** Regular cleaning and disinfection of poultry houses, feeders, waterers, and equipment to reduce bacterial load.
- **Control rodents and wild birds:** Prevent access by rodents and wild birds to poultry facilities, as they can act as vectors.

3. Culling

- Infected birds, particularly those showing high mortality, should be culled to prevent the spread of the disease.

- Culling may also be necessary for chronic carriers that are shedding the bacteria and affecting the flock.

4. Screening and Monitoring

- Regular screening of flocks, especially in breeding and hatcheries, for *S. pullorum* using serological or bacteriological tests.
- Implement strict monitoring to detect early signs of outbreaks and control them quickly.

5. Sanitation and Management

- Ensure proper sanitation in hatcheries, brooding, and rearing facilities.
- Implement good management practices to reduce stress, which can make birds more susceptible to infections.

9.1.3 Chronic Respiratory Disease

Chronic respiratory disease (CRD) in poultry is a common infectious disease characterized by respiratory symptoms, including nasal discharge, coughing, and wheezing. It is primarily caused by *Mycoplasma gallisepticum* (MG), a bacterium that affects the respiratory system of chickens, turkeys, and other poultry species. CRD is often complicated by secondary infections, leading to chronic disease and production losses.

Synonym: Infectious Sinusitis, Mycoplasmosis, *Mycoplasma Gallisepticum* Infection

Etiology

- **Causative agent:** *Mycoplasma gallisepticum* (MG), a bacterium belonging to the genus *Mycoplasma*.

Mode of Transmission

- **Vertical transmission:** Infected hens can pass *Mycoplasma gallisepticum* to their offspring via eggs (transovarial transmission).
- **Horizontal Transmission**

- **Direct contact:** Infected birds shed the bacteria in nasal secretions, saliva, and feces, which can spread to healthy birds.
- **Aerosol transmission:** The bacteria can be spread via respiratory droplets when infected birds cough or sneeze.
- **Contaminated equipment:** Spread through contaminated feed, water, and equipment.
- **Vector transmission:** Wild birds, rodents, or insects can carry the bacteria into poultry houses.

Clinical Signs

- **Early Stage**
 - Nasal discharge (clear to mucopurulent).
 - Sneezing and coughing.
 - Swelling of the head, neck, and sinuses (especially in turkeys).
 - Mild conjunctivitis or eye discharge.
 - Decreased appetite and lethargy.
- **Advanced Stage**
 - Severe nasal discharge (thick, yellow-green).
 - Labored breathing, wheezing, and nasal congestion.
 - Increased mortality in young birds due to secondary bacterial infections.
 - Decreased egg production in laying hens.
 - Growth retardation in chicks.
 - Severe lung congestion and sinusitis in chronic cases.

Diagnosis

1. Clinical Diagnosis

- Based on respiratory symptoms (coughing, nasal discharge, and labored breathing), especially when occurring in flocks with a history

of respiratory disease.

- Swelling of the head and neck is common in turkeys.

2. Laboratory Diagnosis

- **Bacterial culture:** Isolation of *Mycoplasma gallisepticum* from respiratory tract samples (nasal swabs, tracheal swabs, or lung tissue).
- **PCR (polymerase chain reaction):** Used for detecting *Mycoplasma* DNA in clinical samples.
- **Serology:** ELISA (Enzyme-Linked Immunosorbent Assay) to detect antibodies against *Mycoplasma gallisepticum*.
- **Histopathology:** Lesions in the respiratory system (sinusitis, bronchitis, pneumonia).
- **Immunofluorescence or immunohistochemistry:** Detection of bacterial antigens in tissue samples.

Treatment

1. Antibiotics

- **tetracyclines** (e.g., doxycycline, oxytetracycline) are commonly used to treat *Mycoplasma gallisepticum* infections.
- **Macrolides** (e.g., tylosin, lincomycin) and fluoroquinolones may also be effective.
- **Treatment duration:** Prolonged antibiotic therapy is required to control the infection, as *Mycoplasma* infections are chronic and require several days of medication.

2. Supportive Care

- Provide proper nutrition and hydration to help affected birds recover.
- Reduce stress by providing adequate space, ventilation, and a calm environment.

Prevention and Control Measures

1. Vaccination

- **Mycoplasma gallisepticum vaccine:** Vaccination is available for *Mycoplasma gallisepticum* to reduce the severity of clinical signs and transmission.
- **Vaccine strains:** Inactivated and live attenuated vaccines can be used to control infection in breeding flocks.

2. Biosecurity Measures

- **Isolation:** Quarantine new birds before introducing them into the flock to avoid introducing *Mycoplasma*.
- **Contaminated equipment:** Disinfect all equipment, waterers, and feeders regularly to prevent the spread of the infection.
- **Personnel hygiene:** Ensure that personnel handling infected and uninfected birds follow strict hygiene protocols to prevent cross-contamination.

3. Management Practices

- **Control of stress:** Implement good management practices to reduce stress, which can worsen respiratory diseases.
- **Culling:** Infected birds with severe disease or those with chronic infections should be culled to prevent further spread of *Mycoplasma* in the flock.
- **Environmental control:** Provide good ventilation, reduce overcrowding, and maintain clean and dry housing to minimize respiratory stress.

4. Secondary Infections

- Treat secondary bacterial infections, often caused by *Escherichia coli*, using appropriate antibiotics (e.g., amoxicillin, enrofloxacin).

5. Surveillance

- Regular monitoring of poultry flocks for respiratory signs and ongoing testing for *Mycoplasma gallisepticum* to detect early outbreaks and reduce spread.

9.1.4 Fowl Typhoid

Fowl typhoid is a systemic bacterial infection caused by *Salmonella enterica* serovar Gallinarum, which primarily affects chickens and other avian species. It leads to septicemia, high mortality, and can result in reduced egg production and hatchability in poultry.

Synonym: Salmonellosis, *Salmonella Enterica* Infection

Etiology

- Causative agent:** *Salmonella enterica* serovar Gallinarum (S. Gallinarum).

Mode of Transmission

1. Oral Transmission

- The primary mode of transmission is through the ingestion of contaminated feed, water, or litter that contains *Salmonella*.
- Salmonella* bacteria are shed in the feces of infected birds, contaminating the environment.

2. Vertical Transmission

- Infected hens can transmit *Salmonella* to their eggs, leading to the spread of the infection to chicks upon hatching.

3. Fomites (indirect transmission)

- Contaminated equipment, clothing, and vehicles can spread the bacteria between farms.

4. Direct Contact

- Birds in close proximity to infected birds can contract the disease through direct contact, especially in crowded conditions.

Clinical Signs

1. Acute Form

- **High mortality rate:** Often sudden death in young and adult chickens.
- **Depression:** Infected birds exhibit lethargy and loss of activity.
- **Anorexia:** Loss of appetite and reduced feeding.
- **Diarrhea:** Watery or greenish diarrhea, often with a foul smell.
- **Dehydration:** Visible signs of dehydration, including dry, wrinkled skin.
- **High fever:** Temperature rise, often accompanied by a pale comb and wattle.

2. Chronic Form

- **Reduced egg production:** Laying hens may experience decreased egg production or poor hatchability.
- **Poor growth:** Stunted growth in chicks.
- **Emaciation:** Birds lose weight despite normal feeding.
- **Enlarged liver and spleen:** Palpation may reveal organ enlargement.
- **Swelling of joints:** Joint inflammation and lameness may occur in chronic cases.

3. Post-mortem Findings

- Pale, swollen liver with necrotic areas.
- Enlarged spleen.
- Hemorrhages in the intestines and other organs.

Diagnosis

1. Clinical Diagnosis

- Based on symptoms such as sudden mortality, diarrhea, and lethargy in a flock.

- Post-mortem examination may reveal characteristic lesions such as liver and spleen enlargement.

2. Laboratory Diagnosis

- **Bacterial culture:** Isolation of *Salmonella enterica* serovar *Gallinarum* from feces, liver, spleen, or other affected tissues.
- **PCR (polymerase chain reaction):** A rapid method for detecting *Salmonella* DNA in clinical samples.
- **Serological tests:** ELISA (Enzyme-Linked Immunosorbent Assay) for detecting *Salmonella* antibodies in the bird's blood.
- **Antibiotic sensitivity testing:** Determines the most effective antibiotics to treat the infection.

Treatment

1. Antibiotics

- **Broad-spectrum antibiotics:** Such as enrofloxacin, ampicillin, or gentamicin may be used to treat *Salmonella* infections.
- **Tetracycline or sulfonamides:** These can be effective, but antibiotic resistance can be an issue.
- **Supportive care:** Include fluids to combat dehydration, electrolytes to restore balance, and proper nutrition to support recovery.

2. Treatment Duration

- Antibiotics may need to be administered for 5-7 days depending on the severity of the infection and the response of the birds to treatment.

Prevention and Control Measures

1. Vaccination

- **Salmonella vaccines:** Vaccination of poultry against *Salmonella Gallinarum* can help reduce the incidence of fowl typhoid. Live attenuated vaccines are often used in breeder flocks to prevent vertical transmission.

2. Biosecurity Measures

- **Isolation:** New birds should be quarantined before being introduced to the flock to ensure they are free from *Salmonella*.
- **Control of movement:** Limit the movement of poultry and equipment to prevent cross-contamination between farms.
- **Hygiene:** Regular cleaning and disinfection of housing, waterers, feeders, and other equipment to reduce bacterial load.
- **Proper waste management:** Proper disposal of manure and contaminated bedding to prevent environmental contamination.

3. Environmental Management

- **Reduce crowding:** Overcrowded conditions promote the spread of infection, so adequate space should be provided.
- **Control rodents and wild birds:** These animals can be carriers of *Salmonella*, so measures should be taken to control their presence around poultry housing.

4. Monitoring and Surveillance

- Regularly monitor the flock for symptoms of *Salmonella* infection.
- Conduct routine testing of fecal samples to detect asymptomatic carriers.

5. Culling Infected Birds

- In severe outbreaks, infected birds, especially those showing clinical signs, should be culled to prevent the spread of infection.

6. Control of Secondary Infections

- Secondary bacterial infections may complicate the disease. These should be treated with appropriate antibiotics to prevent further morbidity and mortality.

9.2 Viral Diseases of Poultry

9.2.1 Newcastle Disease

Newcastle disease (ND) or Ranikhet disease is a highly contagious viral infection affecting poultry, caused by the Newcastle disease virus (NDV). It can cause severe respiratory, digestive, and nervous system symptoms, leading to high mortality rates, especially in unvaccinated flocks. NDV affects various bird species, including chickens, turkeys, ducks, and pigeons.

Synonym: Avian Paramyxovirus Type 1 (APMV-1), Avian Newcastle Disease (AND), Avian Distemper

Etiology

- **Causative agent:** Newcastle Disease Virus (NDV), a member of the *Avian paramyxovirus 1* (APMV-1) in the family *Paramyxoviridae*.
- NDV has multiple strains, ranging from lentogenic (mild) to velogenic (highly virulent). The virulence of the virus depends on its strain and the species infected.

Mode of Transmission

1. Direct Transmission

- NDV is mainly spread through direct contact with infected birds' secretions, such as saliva, nasal discharge, and feces.

2. Indirect Transmission (Fomites)

- The virus can be transmitted via contaminated equipment, vehicles, clothing, and feed.

3. Airborne Transmission

- NDV can be transmitted over short distances through the aerosolization of viral particles, particularly in crowded conditions.

4. Vertical Transmission

- Infected hens can pass the virus to their eggs, leading to vertical transmission to the chicks.

5. Vector Transmission

- Insects, rodents, and other animals can serve as mechanical vectors.

Clinical Signs

1. Respiratory Symptoms

- Sneezing, coughing, nasal discharge, and gasping for air.
- Labored breathing and swelling of the neck or head.

2. Nervous System Symptoms

- Tremors, twisting of the neck (torticollis), paralysis, and incoordination.
- Drooping wings and legs, inability to stand.

3. Digestive Symptoms

- Diarrhea that may be greenish in color.
- Dehydration and loss of appetite.

4. General Symptoms

- **Sudden death** in severe cases.
- **Reduced egg production** and poor hatchability.
- **Swelling of the head, neck, and eyes** (often seen in virulent forms).

5. Post-mortem Findings

- **Hemorrhages** in various organs, particularly the intestines and liver.
- **Enlarged and congested spleen**, liver, and kidneys.
- **Congestion and edema** in the respiratory system.

Diagnosis

1. Clinical Diagnosis

- Based on the clinical signs of respiratory, nervous, and digestive involvement, especially in an outbreak situation.
- Post-mortem findings, such as hemorrhagic lesions in internal organs, are also suggestive.

2. Laboratory Diagnosis

- **Virus isolation:** Isolation of NDV from swabs or organ tissues using embryonated chicken eggs or cell cultures.
- **PCR (polymerase chain reaction):** Detects viral RNA in clinical samples.
- **Serological tests:** ELISA (Enzyme-Linked Immunosorbent Assay) for detecting antibodies or antigens.
- **Hemagglutination inhibition test (HI test):** For detecting the presence of antibodies against NDV.

Treatment

- **No specific antiviral treatment** exists for Newcastle Disease.
- **Supportive care** to manage symptoms and secondary infections:
 - **Antibiotics:** To treat secondary bacterial infections.
 - **Fluid therapy:** To address dehydration from diarrhea and respiratory distress.
 - **Anti-inflammatory drugs:** To reduce fever and inflammation.

Prevention and Control Measures

1. Vaccination

- Vaccination is the most effective way to control ND. Both live attenuated and inactivated vaccines are available.
- **Vaccination programs** should begin early in life, with booster doses as required.
- Regular vaccination of breeder flocks is essential to ensure immunity in offspring.

2. Biosecurity Measures

- **Isolation of new birds:** Quarantine new arrivals for at least 30 days to prevent introducing the virus.

- **Control of movement:** Limit the movement of poultry, equipment, and personnel between farms.
- **Disinfection:** Regularly clean and disinfect poultry housing, waterers, feeders, and equipment.

3. Environmental Management

- **Reduce overcrowding:** Ensure adequate space for birds to minimize stress and viral transmission.
- **Vector control:** Control rodents, insects, and other animals that may carry the virus.

4. Surveillance

- Conduct routine monitoring and testing for NDV, particularly during outbreaks or in regions with known risk.
- Report any suspected cases to local veterinary authorities.

5. Culling Infected Birds

- In severe outbreaks, affected birds should be culled to limit the spread of the disease.

6. Travel and Trade Restrictions

- Infected regions may enforce restrictions on the movement of poultry and poultry products to prevent further spread.

9.2.2 Marek's Diseases

Marek's disease (MD) is a highly contagious viral disease in poultry, primarily affecting chickens. It is characterized by the formation of lymphomas (tumors) in various organs, nerve degeneration, and immune suppression. The disease is caused by the Marek's disease virus (MDV), which leads to various clinical forms, including cutaneous, ocular, and neurological manifestations.

Synonym: Avian Neoplastic Disease, Range paralysis

Etiology

- **Causative agent: Herpes virus**

Mode of Transmission

1. Aerosol Transmission

- MDV is primarily spread through the inhalation of dust or dander from infected birds, which contain virus particles shed from the skin, feathers, or feces.

2. Direct Contact

- Infected birds shed the virus in feather follicles, saliva, and feces, transmitting it to other birds through direct contact.

3. Vertical Transmission

- Though rare, MDV can sometimes be transmitted vertically from hens to their offspring via eggs.

4. Fomites

- The virus can be transmitted through contaminated equipment, clothing, and housing that comes into contact with infected birds.

Clinical Signs

1. Acute Form

- **Paralysis or incoordination:** Weakness in legs, wings, and neck due to nerve damage.
- **Unilateral or bilateral paralysis** of the legs, wings, and sometimes the neck.
- **Weight loss and poor growth** in young birds.
- **Enlarged, irregularly shaped pupils** (early sign of ocular form).
- **Tremors or ataxia** (loss of coordination).

2. Chronic Form

- **Tumor formation:** Lymphomas (tumors) can develop in various organs, including the liver, spleen, kidneys, and heart.
- **Visceral tumors:** Enlarged organs, such as liver, spleen, and heart.
- **Cutaneous form:** Tumors appear on the skin, especially near the feathers.
- Depression and lethargy.

3. Ocular Form

- Grey eyes or irregular pupils caused by tumors in the eye, leading to blindness.
- Vision impairment can be one of the first signs of the disease in infected birds.

4. Post-mortem Findings

- Tumors in various organs, including the liver, spleen, kidneys, and lungs.
- Enlarged nerves (nerve lesions) are often observed in the sciatic or vagus nerves.
- Hepatomegaly (enlarged liver) and splenomegaly (enlarged spleen) due to lymphoma.
- Inflammation in affected organs.

Diagnosis

1. Clinical Diagnosis

- Based on characteristic clinical signs such as paralysis, eye abnormalities, and the presence of tumors. Affected birds may show weight loss, incoordination, and other neurological symptoms.

2. Laboratory Diagnosis

- **Virus Isolation:** MDV can be isolated from feather follicles, blood, or

tissues using cell cultures or embryonated eggs.

- **Polymerase chain reaction (PCR):** PCR is used to detect MDV DNA in tissues or feathers.
- **Histopathology:** Examination of tissue samples under a microscope can reveal characteristic lesions, such as lymphomas or nerve damage.
- **Serological tests:** ELISA or indirect fluorescent antibody tests can detect antibodies to MDV, although they are not typically used for diagnosing acute infections.

3. Post-mortem Examination

- Tumors and nerve lesions, particularly in the sciatic nerve, along with organ enlargement, help confirm the diagnosis.

Treatment

- No specific antiviral treatment is available for Marek's disease. Once clinical signs appear, the disease is typically fatal.
- Supportive care: To alleviate symptoms, some supportive care like providing clean water, proper nutrition, and treatment for secondary bacterial infections may help improve the condition of affected birds.
- Culling: Infected birds are often culled to prevent further spread of the disease within a flock.
- Control of secondary infections with antibiotics may be necessary in some cases.

Prevention and Control Measures

1. Vaccination

- **Vaccination is the most effective control method** for Marek's disease. Vaccines based on Serotype 2 or 3 are commonly used to prevent the disease.
- Vaccination is typically given to chicks as day-old or at 1-day-old to ensure immunity before exposure to the virus.

- Vaccines do not completely prevent infection but can reduce the severity and occurrence of clinical disease and tumors.

2. Biosecurity

- **Quarantine and isolation** of new birds before introducing them to a flock.
- **Hygiene and sanitation:** Regular cleaning and disinfection of housing, equipment, and tools to prevent cross-contamination.

3. Environmental Management

- **Reduce overcrowding:** Overcrowded conditions promote the spread of the virus.
- **Control dust and dander:** Minimize dust in poultry houses, as the virus spreads through inhalation of dust particles containing the virus.

4. Genetic Resistance

- Some poultry breeds are more resistant to Marek's disease. Selective breeding for genetic resistance may reduce the disease's impact.

5. Monitoring and Surveillance

- Regular monitoring for clinical signs of Marek's disease and testing of new or affected birds to detect the virus early.

6. Culling Infected Birds

- Infected birds should be culled to limit virus spread and prevent further outbreaks.

9.2.3 Infectious Bursal Disease

Infectious Bursal Disease (IBD), commonly known as Gumboro disease, is a highly contagious viral disease that primarily affects young chickens. The disease targets the bursa of Fabricius, an important immune organ in birds, leading to immunosuppression. This condition makes the affected birds more susceptible to secondary infections and other diseases.

Synonym: Gumboro Disease, Avian AIDS

Etiology: Birna virus

Mode of Transmission

1. Fecal-oral Transmission

- The primary route of transmission is through the ingestion of feces or contaminated feed and water. Infected birds shed the virus in their feces, contaminating the environment and infecting other birds.

2. Vertical Transmission

- Infected hens can pass the virus to their offspring via eggs, leading to early infections in chicks.

3. Fomites (indirect transmission)

- The virus can be spread via contaminated equipment, clothing, shoes, and vehicles that come into contact with infected birds or their excretions.

4. Direct Contact

- Close contact between infected and healthy birds can lead to transmission of the virus.

Clinical Signs

1. Acute Form

- **Sudden death:** Often occurs without prior clinical signs, particularly in young birds.
- **Depression and lethargy:** Infected birds appear weak and lethargic.
- **Diarrhea:** Watery or greenish diarrhea is commonly seen.
- **Dehydration:** Visible signs of dehydration such as dry skin and sunken eyes.
- **Loss of appetite:** Infected birds exhibit anorexia (loss of appetite).
- **Immunosuppression:** Reduced ability to fight off secondary infections, leading to increased susceptibility to other diseases.

2. Subclinical Form

- **Immunosuppression:** While the birds may appear healthy, their immune system is compromised, making them more vulnerable to secondary infections.
- **Poor growth and weight loss:** Affected birds may show stunted growth and reduced weight gain.

3. Post-mortem Findings

- **Enlarged and hemorrhagic bursa of fabricius:** The bursa appears swollen, pale, and may contain hemorrhages.
- **Atrophy of the bursa:** In chronic cases, the bursa may appear atrophic and pale.
- **Hemorrhages in other organs:** Such as the thymus, kidneys, and liver, can sometimes be observed.
- **Enlarged spleen and liver:** These organs may show signs of inflammation or congestion.

Diagnosis

1. Clinical Diagnosis

- Diagnosis is often based on the observation of sudden death in young chickens, along with clinical signs of diarrhea, lethargy, and dehydration.

2. Laboratory Diagnosis

- **Virus isolation:** IBDV can be isolated from bursal tissue, feces, or other affected organs using cell cultures.
- **Polymerase chain reaction (PCR):** A molecular method used to detect IBDV DNA in clinical samples, such as bursal tissue or feces.
- **Enzyme-linked immunosorbent assay (ELISA):** This test detects antibodies or viral antigens in the serum, tissues, or feces of infected birds.

- **Histopathology:** Tissue samples (e.g., bursa, spleen) can be examined for characteristic lesions, such as bursal atrophy or hemorrhages.
- **Immunohistochemistry (IHC):** Can be used to detect viral antigens in tissue sections.

Treatment

- **No specific antiviral treatment** is available for IBD. Once the disease occurs, supportive care is often used to manage the symptoms.
- **Supportive Care**
 - Hydration therapy to counteract dehydration.
 - Electrolytes and nutritional support to aid in recovery and reduce mortality.
- **Control of Secondary Infections**
 - Secondary bacterial or viral infections may complicate IBD, and treatment with appropriate antibiotics or other medications may be required.
- **Culling:** Infected birds may be culled to reduce the spread of the virus and prevent further economic losses.

Prevention and Control Measures

1. Vaccination

- **Vaccination is the most effective control method** for IBD. There are various live and inactivated vaccines available for IBD.
- Vaccines can be administered through drinking water, eye drops, or intramuscular injection.
- Vaccination should be done early, typically at 1-7 days of age, to ensure protection before exposure to the virus.
- **Booster vaccines** are recommended for breeder flocks to ensure maternal antibody protection in chicks.

2. Biosecurity Measures

- **Isolation:** New birds should be quarantined for at least 2-3 weeks to prevent the introduction of the virus into the flock.
- **Cleaning and disinfection:** Regular cleaning and disinfection of poultry housing, equipment, and vehicles are essential to prevent cross-contamination.
- **Control of visitors:** Restrict access to the poultry farm to essential personnel and ensure they follow biosecurity protocols.

3. Environmental Management

- **Minimize Stress:** Stress factors such as overcrowding, poor nutrition, or temperature fluctuations can make birds more susceptible to infection.
- **Proper waste disposal:** Fecal matter should be removed regularly and disposed of properly to reduce environmental contamination.
- **Rodent and insect control:** Rodents and insects can spread the virus, so measures should be taken to control these pests.

4. Surveillance and Monitoring

- Regular monitoring of flocks for symptoms of IBD is important for early detection.
- Testing for the virus in flocks with suspicious clinical signs should be conducted to confirm the diagnosis.

5. Culling and Quarantine

- Infected birds should be culled immediately to prevent the spread of the disease.
- Quarantine affected areas and birds to limit viral transmission within the farm.

9.2.4 Infectious Bronchitis

Infectious Bronchitis is a highly contagious viral disease of chickens that primarily affects the respiratory system but may also involve the reproductive and renal systems. It leads to decreased egg production, poor egg quality, and significant economic losses in the poultry industry.

Etiology: Corona virus

Mode of Transmission

1. Aerosol Transmission

- The virus spreads through respiratory droplets released when infected birds cough or sneeze.

2. Direct Contact

- Close contact between infected and healthy birds facilitates transmission.

3. Fomites

- Contaminated equipment, feed, water, or human clothing can carry the virus.

4. Vertical Transmission

- Rare but possible if infected hens pass the virus to their eggs.

Clinical Signs

1. Respiratory Signs (primary presentation)

- Nasal discharge
- Coughing and sneezing
- Gurgling or rattling sounds in the throat
- Open-mouth breathing
- Conjunctivitis (inflammation of the eyes)

2. Reproductive System Effects (in layers)

- Reduced egg production
- Poor egg quality, including thin, misshapen, or rough-shelled eggs
- Watery egg whites

3. Renal Signs (in nephropathogenic strains)

- Increased water intake (polydipsia)
- Wet droppings or diarrhea
- Swollen kidneys on post-mortem examination

4. General Signs

- Depression and lethargy
- Reduced feed intake
- Weight loss or poor growth in young birds

5. Post-mortem Findings

- Inflammation and congestion of the trachea and lungs
- Cheesy exudate blocking the trachea or bronchi
- Swollen and pale kidneys with urate deposits (in nephropathogenic strains)

Diagnosis

1. Clinical Diagnosis

- Based on respiratory signs, decreased egg production, and egg abnormalities in the flock.

2. Laboratory Diagnosis

- **Virus isolation:** Isolation of IBV from tracheal swabs, feces, or kidney tissue in embryonated chicken eggs or cell cultures.
- **Polymerase chain reaction (PCR):** Rapid detection and identification of IBV RNA in clinical samples.

- **Serology:** Detection of IBV antibodies using ELISA, hemagglutination inhibition (HI), or virus neutralization tests.
- **Histopathology:** Examination of respiratory, renal, or reproductive tissues for characteristic lesions.

Treatment

- **No specific antiviral treatment** exists for IBV. Management is focused on supportive care and preventing secondary infections.
- **Supportive Care**
 - Provide adequate heat and ventilation to reduce stress on affected birds.
 - Ensure access to clean water and high-quality feed to support recovery.
- **Antibiotics**
 - Broad-spectrum antibiotics (e.g., tetracyclines, sulfonamides) may be used to control secondary bacterial infections.

Prevention and Control Measures

1. Vaccination

- **Live attenuated vaccines:** Commonly used for initial protection in young birds. Administered via drinking water, spray, or eye drops.
- **Inactivated vaccines:** Used as a booster in layers and breeders to enhance immunity.
- Vaccination schedules should be tailored based on the prevalent serotypes in the region.

2. Biosecurity Measures

- **Isolation:** Quarantine new birds before introducing them into the flock.
- **Hygiene:** Regular cleaning and disinfection of housing, equipment, and vehicles to reduce viral load.

- **Controlled access:** Restrict access to poultry farms to essential personnel.
- **Rodent and wild bird control:** Prevent contact with potential carriers of the virus.

3. Environmental Management

- Maintain optimal ventilation to reduce respiratory stress.
- Minimize overcrowding to limit the spread of the virus.

4. Monitoring and Surveillance

- Conduct regular health checks and diagnostic testing to detect IBV early.
- Monitor vaccination efficacy by testing antibody levels in the flock.

5. Management of Outbreaks

- Isolate affected birds to prevent the spread of the virus.
- Enhance environmental hygiene and biosecurity measures during outbreaks.

9.2.5 Fowl Pox

Fowl pox is a contagious viral disease affecting chickens, turkeys, and other avian species. It is caused by the **avian poxvirus**, a member of the family **Poxviridae**, and can present in two forms: **cutaneous (dry form)** and **diphtheritic (wet form)**.

Synonyms

- Avian pox

Etiology: Avian poxvirus (various strains)

Mode of transmission

1. **Direct contact:** Through abrasions or wounds on the skin.
2. **Indirect contact:** Via contaminated equipment, waterers, feeders, or

housing.

3. **Mechanical transmission:** By blood-feeding insects such as mosquitoes, mites, and lice.

Clinical Signs

1. Cutaneous Form (dry form)

- Nodular lesions (wart-like) on the unfeathered areas such as comb, wattles, eyelids, and legs.
- Lesions progress to scabs and may result in mild discomfort.

2. Diphtheritic Form (wet form)

- Yellowish-white plaques in the oral cavity, throat, and upper respiratory tract.
- Difficulty breathing, eating, or drinking.

3. General Signs

- Reduced appetite
- Weight loss
- Drop in egg production in laying birds
- Mortality is higher in the diphtheritic form.

Diagnosis

1. **Clinical observation:** Typical lesions in affected birds.

2. **Histopathology:** Detection of **bollinger bodies** (inclusion bodies) in epithelial cells.

3. Laboratory Confirmation

- Virus isolation
- PCR to detect avian poxvirus DNA
- Electron microscopy to identify viral particles

Treatment

- No specific antiviral treatment for fowl pox.
- **Supportive care**
 - Provide clean water and easily digestible feed.
 - Treat secondary bacterial infections with antibiotics.
- **Topical application:** Iodine or antiseptic solutions to lesions to prevent secondary infections.

Prevention and Control Measures

1. Vaccination

- Effective vaccines are available and administered via wing-web stab method (usually live attenuated vaccines).
- Vaccinate chicks at 6–10 weeks of age.

2. Vector Control

- Minimize mosquito breeding by eliminating standing water.
- Use insecticides or repellents in the poultry environment.

3. Biosecurity

- Maintain good hygiene and sanitation practices.
- Disinfect equipment, housing, and materials regularly.

4. Isolation: Quarantine affected birds to prevent spread.

5. Culling: Remove severely affected birds if necessary to protect the flock.

9.2.6 Bird Flu

Bird flu, also known as avian influenza, is an infectious disease caused by influenza viruses that primarily affect birds but can also infect humans and other animals. The most notable subtypes are H5N1 and H7N9, which are highly pathogenic and can cause severe illness in both birds and humans.

Synonyms

- Avian Influenza
- Fowl Plague
- Avian Flu

Etiology

Bird flu is caused by **avian influenza viruses** from the Orthomyxoviridae family. These viruses are classified into:

1. **Low Pathogenic avian influenza (LPAI)** – It cause mild illness in birds.
2. **Highly pathogenic avian influenza (HPAI)** – It cause severe disease and high mortality rates in birds.

Common Subtypes

- **H5N1**: High fatality rate in humans.
- **H7N9**: Recently emerged, capable of human infection.
- **H9N2**: Mild symptoms but widespread in poultry.

Mode of Transmission

1. **Direct contact**: With infected birds (live or dead), secretions, or droppings.
2. **Indirect contact**: Through contaminated surfaces, equipment, water, or feed.
3. **Aerosols**: Inhalation of virus particles from infected birds.
4. **Zoonotic transmission**: From infected birds to humans, especially during close handling.

Clinical Signs

In Birds

- Sudden death with no prior signs (HPAI).
- Respiratory distress (coughing, sneezing).

- Swelling of the head, comb, and wattles.
- Cyanosis of comb and wattles.
- Reduced egg production or misshapen eggs.
- Diarrhea.
- Neurological signs (tremors, incoordination).

Diagnosis

- PCR (Polymerase Chain Reaction) for viral RNA.
- Virus isolation in embryonated eggs.
- Serology: Hemagglutination Inhibition (HI) or ELISA.

Treatment

- No specific treatment.
- Supportive care for mild cases (LPAI).

Prevention and Control

1. Biosecurity

- Restrict movement of birds.
- Disinfect equipment and facilities regularly.

2. Vaccination: For LPAI in endemic areas.

3. Culling: Immediate culling of infected flocks to prevent spread.

4. Surveillance: Regular monitoring and reporting outbreaks.

9.3 Protozoal Diseases of Poultry

9.3.1 Coccidiosis

Coccidiosis is a parasitic disease affecting the intestinal tract of poultry, caused by protozoa of the genus *Eimeria*. It leads to poor growth, reduced productivity, and mortality, especially in young birds.

Synonyms

- Intestinal Coccidiosis
- Eimeriosis

Etiology

The disease is caused by protozoa from the genus *Eimeria*. The most common species affecting poultry include:

1. ***Eimeria tenella***: It causes severe cecal coccidiosis.
2. ***Eimeria acervulina***: It infects the upper small intestine.
3. ***Eimeria maxima***: It affects the mid-small intestine.
4. ***Eimeria necatrix***: It is highly pathogenic, affecting the small intestine.
5. ***Eimeria brunetti***: It targets the lower small intestine and rectum.

Mode of Transmission

1. **Fecal-oral route**: Birds ingest sporulated oocysts from contaminated feed, water, or litter.
2. **Environment**: Oocysts survive in moist, warm conditions and can remain infective for months.

Clinical Signs

Mild Cases

- Decreased feed intake.
- Poor growth and feed conversion.

Severe Cases

- Diarrhea (may be bloody in cases like *E. tenella*).
- Dehydration.
- Ruffled feathers.
- Depression and lethargy.
- Pale combs and wattles due to anemia.
- High mortality in acute cases.

Diagnosis

1. **Clinical signs and history:** Observation of bloody diarrhea and poor growth in flock settings.
2. **Post-mortem Examination**
 - Lesions in the intestines specific to *Eimeria* species.
 - Cecal cores in *E. tenella*.
3. **Microscopic Examination**
 - Identification of oocysts in feces or intestinal scrapings.
4. **PCR and molecular tests:** Species-specific identification of *Eimeria*.

Treatment

1. **Anticoccidial Drugs**
 - **Sulfa drugs:** Sulfamethazine, Sulfaquinoxaline.
 - **Amprolium:** Effective in blocking parasite metabolism.
2. **Supportive Therapy**
 - Provide clean water and electrolytes to combat dehydration.
 - High-quality feed to promote recovery.

Prevention and Tontrol

1. **Good Tanagerment Practices**
 - Clean and disinfect poultry houses regularly.
 - Avoid wet litter to prevent oocyst sporulation.
2. **Biosecurity**
 - Restrict access to the farm and control movement of equipment.
3. **Vaccination**
 - Live attenuated *Eimeria* vaccines are available for protection.
4. **Anticoccidial Medications in Feed**
 - Use ionophores (e.g., Monensin, Salinomycin) or synthetic drugs (e.g., Diclazuril) as preventive measures.

5. Rotation of Anticoccidials

- To prevent drug resistance, rotate between different classes of anticoccidials.

6. Litter Management

- Frequent removal or treatment of litter to reduce oocyst buildup.

9.4 Fungal Diseases of Poultry

9.4.1 Brooder's Pneumonia

Brooder's pneumonia is a respiratory disease in poultry caused by the fungus *Aspergillus fumigatus*. It primarily affects young birds (broilers and chicks) in brooding conditions and is characterized by respiratory distress and high mortality.

Synonyms

- Aspergillosis
- Fungal Pneumonia

Etiology

- **Causative agent:** *Aspergillus fumigatus* is the most common, but other *Aspergillus* species (e.g., *A. flavus*, *A. niger*) may also cause the disease.
- The fungus thrives in damp, poorly ventilated, or moldy environments and produces air borne spores that infect birds.

Mode of Transmission

1. **Inhalation of spores:** Birds inhale fungal spores from contaminated litter, feed, or environment.
2. **Egg infection:** Spores can penetrate eggshells, infecting embryos during incubation.
3. **Contact transmission:** Direct contact with contaminated surfaces.

Clinical Signs

Acute Form (young birds)

- Rapid onset of respiratory distress (open-mouth breathing, gasping).
- Cyanosis (bluish discoloration of skin and comb).
- Lethargy and inappetence.
- High mortality within days of infection.

Chronic Form (older birds)

- Weight loss and poor growth.
- Nasal discharge and coughing.
- Nodular lesions in the lungs and air sacs.
- Decreased egg production.

Diagnosis

1. **Clinical signs:** Respiratory symptoms in young birds with high mortality.
2. **Necropsy Findings**
 - Yellowish-white nodules or plaques in lungs, air sacs, and other organs.
 - Thickened air sacs (airsacculitis).
3. **Microscopic Examination**
 - Fungal hyphae in tissue samples.
4. **Fungal culture:** Isolation of *Aspergillus* species on Sabouraud Dextrose Agar.
5. **Histopathology:** To confirm fungal involvement in tissues.

Treatment

1. Antifungal Drugs

- **Copper sulfate** (added to drinking water).

- **Itraconazole or voriconazole** (limited use in poultry).
- **Nystatin**: Effective for surface infections.

2. Supportive Care

- Provide clean water and improve ventilation.

3. **Culling**: Severely affected birds may need to be culled to prevent further spread.

Prevention and Control

1. Environmental Management

- Use dry, clean, and well-ventilated housing.
- Avoid wet or moldy litter and feed.
- Disinfect poultry houses with antifungal agents.

2. Egg and Incubator Hygiene

- Clean and fumigate eggs before incubation.
- Maintain proper incubation hygiene.

3. Biosecurity Measures

- Restrict access to the farm and implement quarantine for new birds.

4. Preventive Medications

- Use antifungal additives in feed or water in high-risk environments.

9.4.2 Mycotoxicosis

Mycotoxicosis is a group of diseases in poultry caused by the ingestion of feed or grains contaminated with **mycotoxins**, which are toxic secondary metabolites produced by certain molds (fungi). These toxins can affect the liver, kidneys, immune system, and productivity of birds.

Synonyms:

- Fungal Toxin Poisoning
- Mold Poisoning

Etiology

Mycotoxicosis is caused by mycotoxins produced by molds. Common molds and their associated toxins include:

1. **Aspergillus flavus** and **A. parasiticus**: Produce *aflatoxins*.
2. **Fusarium species**: Produce *trichothecenes* (e.g., T-2 toxin), *zearalenone*, and *fumonisins*.
3. **Penicillium species**: Produce *ochratoxins*.
4. **Claviceps species**: Produce *ergot alkaloids*.

Mode of Transmission

1. **Ingestion**: Consumption of feed or grains contaminated with mycotoxins.
2. **Feed storage**: Poor storage conditions promote fungal growth and toxin production.
3. **Water contamination**: In some cases, water sources can harbor molds.

Clinical Signs

General Signs

- Reduced feed intake and growth rate.
- Poor feather quality.
- Decreased egg production and hatchability.

Toxin-specific Signs

- **Aflatoxins**
 - Hepatotoxicity (enlarged, pale liver).
 - Jaundice and reduced immunity.
- **Trichothecenes (T-2 toxin)**
 - Oral lesions and necrosis.
 - Reduced egg production.

- **Zearalenone**
 - Estrogenic effects: Swollen cloaca and reproductive disorders.
- **Ochratoxins**
 - Kidney damage, increased water intake, and urination.
- **Fumonisins**
 - Neurological signs and respiratory issues in severe cases.

Diagnosis

1. Feed Analysis

- Laboratory testing of feed samples for mycotoxin contamination (HPLC, ELISA).

2. Histopathology

- Tissue changes in the liver, kidneys, or other affected organs.

3. Clinical History and Signs

- Correlation of symptoms with feed quality and storage practices.

4. Mycological Examination

- Identification of molds in feed or storage areas.

Treatment

1. Removal of Contaminated Feed

- Replace with clean, toxin-free feed.

2. Mycotoxin Binders

- Add binders (e.g., bentonite clay, activated charcoal, or yeast cell walls) to contaminated feed to reduce toxin absorption.

3. Supportive Therapy

- Provide vitamins (A, E, and selenium) to counteract oxidative stress.
- Improve hydration and electrolyte balance.

- 1. Feed Storage**
 - Store feed in dry, cool conditions to prevent fungal growth.
 - Use preservatives or antifungal agents (e.g., propionic acid) in stored feed.
- 2. Regular Testing**
 - Periodically test feed for mycotoxins.
- 3. Use of Mycotoxin Binders**
 - Incorporate binders in poultry diets as a preventive measure.
- 4. Proper Feed Handling**
 - Avoid prolonged storage of feed or grains.
 - Ensure thorough cleaning of feed silos and equipment.
- 5. Crop Management**
 - Minimize pre-harvest fungal contamination through good agricultural practices.
- 6. Biosecurity Measures**
 - Control pests and rodents that may carry fungal spores.

Exercise

Choose the correct answer from the given alternatives.

1. What is the causative agent of fowl cholera?
a. *Pasteurella multocida* b. *Mycoplasma gallisepticum*
c. *Salmonella pullorum* d. *Salmonella gallinarum*
2. Which of the following is a synonym for fowl cholera?
a. Avian pasteurellosis b. Bird flu
c. Newcastle disease d. Coryza
3. **How can fowl cholera be prevented and controlled?**
a. Routine vaccination and sanitation
b. Avoiding overcrowding
c. Quarantining new birds
d. All of the above
4. What is the causative agent of pullorum disease?
a. *Salmonella pullorum* b. *Pasteurella multocida*
c. *Salmonella gallinarum* d. *Escherichia coli*
5. Which of the following is a synonym for pullorum disease?
a. Avian typhoid b. Bacillary white diarrhea
c. Chronic respiratory disease d. Coryza
6. What is the primary mode of transmission of pullorum disease?
a. Aerosol transmission b. Vertical transmission through eggs
c. Contaminated water d. Tick bites
7. Which of the following symptoms is typical of pullorum disease?
a. Blindness and lameness

- b. White diarrhea and high mortality in chicks
- c. Swollen joints and nasal discharge
- d. Sudden death with no signs

8. What is the causative agent of chronic respiratory disease?

- a. *Mycoplasma gallisepticum*
- b. *Pasteurella multocida*
- c. *Salmonella gallinarum*
- d. *Escherichia coli*

9. Which of the following is a synonym for chronic respiratory disease?

- a. Infectious coryza
- b. Air sac disease
- c. Bacillary white diarrhea
- d. Newcastle disease

10. What is the most common mode of transmission of CRD?

- a. Aerosol transmission
- b. Contaminated water
- c. Vertical transmission through eggs
- d. Both a and c

11. Which of the following is a key symptom of CRD?

- a. Watery white diarrhea
- b. Coughing, nasal discharge, and reduced egg production
- c. Swollen wattles
- d. Sudden death

12. Which of the following is a synonym for fowl typhoid?

- a. Avian paratyphoid
- b. *Salmonella enteritis*
- c. Salmonellosis of adult poultry
- d. Avian cholera

13. What is the primary cause of mycotoxicosis?

- Toxins produced by molds such as *Aspergillus* and *Fusarium*
- Infection with fungal spores
- Inhalation of toxic gases
- Viral contamination

14. What is the causative agent of brooder's pneumonia?

- Aspergillus fumigatus*
- Candida albicans*
- Fusarium* spp.
- Clostridium* spp.

15. Which of the following clinical signs is commonly associated with coccidiosis?

- Bloody diarrhea, weight loss, and poor growth
- Nasal discharge and coughing
- Paralysis and lameness
- Nodular lesions on skin

16. Which disease is also known as fowl plague?

- Ranikhet
- IBD
- Mareks
- Bird flu

17. Which disease is also known as avian distemper?

- Ranikhet
- IBD
- Mareks
- Bird flu

18. Which disease is also known as avian AIDS?

- Ranikhet
- IBD
- Mareks
- Bird flu

19. Which disease is also known as range paralysis?

- Ranikhet
- IBD
- Mareks
- Bird flu

20. What is the causative agent of Fowl Pox?

- a. Avipoxvirus
- b. Paramyxovirus
- c. Herpesvirus
- d. Reovirus

Write short answer to the following questions.

1. Write the etiology, clinical signs and preventive measures of Pullorum disease.
2. Write the etiology, clinical signs and methods of diagnosis of CRD.
3. Write the clinical signs and preventive measures of fowl typhoid.
4. Write the mode of transmission and clinical signs of ranikhet disease.
5. Write the etiology, clinical signs, and methods of diagnosis of bird flu.
6. Write the synonym of Gumboro disease. Write its clinical signs and mode of transmission.
7. How can spread of Bird flu be prevented? write.
8. Write the etiology, clinical signs and methods of diagnosis of Infectious Bronchitis.
9. Write the clinical signs and symptoms and preventive measures of Brooder's Pneumonia.
10. Write etiology and clinical signs of Mycotoxicosis in poultry.

Write long answer to the following questions.

1. Write the etiology, clinical signs and preventive measures and methods diagnosis of CRD.
2. Write the etiology, clinical signs, diagnosis and line of treatment of Pullorum disease.
3. Write the clinical signs, mode of transmission, diagnosis and preventive measures of Fowl cholera.
4. Write the mode of transmission, clinical signs, diagnosis and treatment of Fowl typhoid.

5. Write the etiology, clinical signs, diagnosis and prevention and control measures of Ranikhet disease.
6. Write the synonym of Bird flu. Writ its clinical signs, mode of transmission and methods of diagnosis
7. Write the etiology, clinical signs, line of treatment and control measures of Infectious Bronchitis.
8. Write the etiology, mode of transmission, clinical signs and methods of diagnosis of IBD.
9. Write the clinical signs, diagnostic methods, treatment and preventive measures of Brooders Pneumonia.
10. Write etiology and clinical signs, method of diagnosis and control measures of Mycotoxicosis in poultry.
11. Write etiology and clinical signs, method of diagnosis and control measures of Mareks disease in poultry.
12. Write etiology and clinical signs, line of treatment and control measures of Fowl pox.
13. Write etiology and clinical signs, line of treatment and control measures of Coccidiosis in poultry.

10.1 Definition and Uses of Vaccine

A **vaccine** is a biological preparation designed to stimulate the immune system to recognize and combat specific pathogens (such as bacteria or viruses). It typically contains an agent that resembles a disease-causing microorganism, often in a weakened, killed, or inactivated form, or as fragments of the microorganism (such as proteins or genetic material).

Uses of Vaccine

1. Prevention of Infectious Diseases

- Vaccine protects animals from diseases caused by bacteria, viruses, fungi, or parasites (e.g., rabies, foot-and-mouth disease, brucellosis).

2. Zoonotic Disease Control

- Reduces the risk of transmission of diseases from animals to humans (e.g., rabies, leptospirosis, anthrax).

3. Improved Animal Health and Productivity

- Ensures healthier livestock, leading to increased productivity in terms of milk, meat, eggs, and wool production.

4. Disease Control in Wildlife

- Helps manage diseases in wild animal populations, preventing their spread to domestic animals or humans.

5. Economic Benefits in Agriculture

- Reduces losses caused by disease outbreaks in livestock, minimizing treatment costs and improving yields for farmers.

6. Enhanced Trade and Export

- Ensures compliance with international health standards, enabling safe trade and export of animals and animal products.

7. Epidemic and Andemic Control

- Used in mass vaccination programs during outbreaks to control diseases (e.g., avian influenza, Newcastle disease).

8. Animal Welfare

- Reduces suffering by preventing painful or life-threatening diseases in pets, farm animals, and wildlife.

9. Food Safety and Public Health

- Prevents diseases in food-producing animals, ensuring safer meat, milk, and egg supplies for human consumption.

10.2 Vaccine Handling and Storage

Proper handling and storage of vaccines are crucial to maintaining their efficacy and safety. Below are the key points regarding vaccine handling and storage:

Vaccine Handling

1. Cold Chain Maintenance

- Ensure vaccines are kept at the correct temperature (typically **+2°C to +8°C** for most vaccines) from manufacture to administration.
- Avoid freezing vaccines unless specified (some vaccines like oral polio can tolerate freezing).

2. Inspection on Delivery

- Check vaccine vials for damage, expiration dates, and any signs of contamination (e.g., discoloration, particles, cracks in vials).

3. Minimize Exposure to Light

- Many vaccines are light-sensitive and should be kept away from direct sunlight or fluorescent light to prevent degradation.

4. Careful Handling

- Handle vaccines gently to avoid shaking or damage to the vial, especially for vaccines with adjuvants or live attenuated forms.

5. Documentation

- Maintain records of vaccine batches, expiration dates, and storage conditions.

Vaccine Storage

1. Refrigerator Requirements

- Use a dedicated medical or vaccine refrigerator, not a domestic refrigerator.
- Ensure temperature monitoring with a calibrated thermometer or a data logger.

2. Temperature Monitoring

- Check and log the refrigerator temperature at least twice daily to ensure it stays within the recommended range.
- Install an alarm system to alert in case of temperature deviations.

3. Organization in the Refrigerator

- Place vaccines in the central shelves of the refrigerator, not in doors or near the back walls where temperatures can fluctuate.
- Keep vaccines in their original packaging to protect them from light and damage.

4. Avoid Overcrowding

- Ensure adequate air circulation inside the refrigerator. Do not overfill or block air vents.

5. Power Backup

- Have an alternative power source (e.g., generator) in case of power outages to maintain storage temperature.

6. Separate Storage for Opened Vials

- Keep opened vials separated from unopened stock and follow strict guidelines for their use.

7. Expiry Management

- Rotate stock using the **First Expired, First Out (FEFO)** method to ensure older vaccines are used first.

8. Emergency Plan

- Have a contingency plan for vaccine storage in case of equipment failure or power outages.

10.3 Vaccination Schedule for Livestock and Pet

Vaccination schedule for cattle

S.N.	Vaccine	Disease	Age	Dose	Booster	Interval	Season
1	FMD (Hectus)	FMD	3-8 wk	10 ml S/C	After 3 month	6 month	Bhadra- Falgun
	Rakshya FMD	FMD	4 month	3 ml S/C	After 1 month		
2	HS broth	HS	All age	5 ml S/C	After 6 month	Annual	Before start of rain
	HS oil adjuvent	HS	All age	3 ml S/C	After 3 month	Annual	
3	Polyvalent BQ	BQ	All age	5 ml S/C	After 6 month	Annual	Before start of rain
4	HS and BQ	HS+BQ	All age	1 ml S/C	After 6 month	Annual	Before start of rain
5	Anthrax spore	Anthrax	All age	1 ml S/C	After 6 month	Annual	Before start of rain

6	Tet-vac	Tetanus	All age	2 ml S/C	After 6 week	Annual	At anytime
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Vaccination Schedule for Goat

Months	Vaccine	Goat	Kids (Above 6 months)
January	CCPP (Contagious Caprine Pleuro Pneumonia)	0.2 ml I/D	0.2 ml I/D
March	HS	5 ml S/C	0.2 ml S/C
April	Goat pox	Scratch method	Scratch method
May	Enterotoxaemia, FMD	5 ml S/C	2.5 ml S/C
July	BQ	5 ml S/C	2.5 ml S/C
August	F.M.D	5 ml S/C	0.5 ml S/C
September	Enterotoxaemia	5 ml S/C	2.5 ml S/C

Vaccination Schedule for Sheep

Disease	Vaccine	Age	Dose	Booster	Interval	Season
F.M.D	F.M.D vaccine	Adult	5 ml S/C	-	Annual	Winter/ Autumn
B.Q	B.Q vaccine	Adult	2 ml S/C	-	Annual	All season
Lamb dysentery	Lamb dysentery vaccine	Lamb	2 ml S/C	-	Annual	All season
B.Q	B.Q polyvalent vaccine	Lamb Adult	2 ml S/C 5 ml S/C	-	Annual	All season
Enterotoxaemia	Enterotoxaemia vaccine	Lamb Adult	2.5 ml S/C 5 ml S/C	7-10 days	Annual	Lambing season
HS	HS adjuvant vaccine	Adult	2 ml S/C	-	Annual	March/June

Sheep pox	Sheep pox vaccine	Lamb Adult	3 ml S/C 5 ml S/C	Repeat after 6 months	Annual	December/ March
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Vaccination Schedule for Pig

S.N.	Age	Vaccine	Time
1.	Adult pig	Leptospirosis, parvo viral enteritis, Swine erysepalous	Before mating 2 times a year
2.	Gilt and Sow	E. coli, Atropic rhinitis	Before parturition 2 times a year
3.	Growers	Erysepalous	New one
4.	Growers and adult	F.M.D	Bhadra- Falgun
5.	After 6-8 weeks	Swine fever vaccine	Repeat annually

Vaccination Schedule for Dog

S.N.	Age	Vaccination
1.	6-8 weeks	DHPPL (CD, Canine hepatitis, parvovirus, Leptospirosis, Parainfluenza)
2.	9-12 weeks	Booster dose of DHPPL
3.	13 weeks	Rabies
4.	4 month	Repeat DHPPL and rabies

10.4 Vaccination Schedule for Layers and Broilers

Vaccination Schedule for Layers

S.N.	Days	Vaccine	Strain	Dose	Route
1.	1	Marek's disease	HVT 126	0.1 ml	S/C
2.	5-7	Ranikhet disease	F1 or R2B	1 drop	I/O or I/N
3.	10	Leetchy heart disease	-	0.25 ml	S/C or I/M

4.	12-13	Gumboro (IBD)	Intermediate plus	1 drop	I/O or I/N
5.	18-19	Gumboro (IBD)	Intermediate	1 drop	I/O or I/N
6.	25-27	Ranikhet disease	Lasota	1 drop	D/W, SMP
7.	30	IBD	Intermediate		DW, SMP
8.	42	Fowl pox	Fowl pox		Wing web
9.	7 weeks	ND	Lasota		DW, SMP
10.	9 - 10 weeks	ND	R2B	0.5 ml	DW, SMP
11.	13 weeks	Fowl pox	Fowl pox		Wing web
12.	14 weeks	IB	IBH120		DW, SMP
13.	16 weeks	IB+ND	0.5 ml		I/M
14.	25 - 26 weeks	IB+ND			DW, SMP

Vaccination Schedule for Broiler

S.N.	Days	Vaccine	Strain	Dose	Route
1.	1	Marek's disease	HVT 126	0.1 ml	S/C
2.	5-7	Ranikhet disease	F1 or R2B	1 drop	I/O or I/N
3.	10	Leetchy heart disease	-	0.25 ml	S/C or I/M
4.	12-13	Gumboro (IBD)	Intermediate plus	1 drop	I/O or I/N
5.	18-19	Gumboro (IBD)	Intermediate	1 drop	I/O or I/N
6.	25-27	Ranikhet disease	Lasota	1 drop	D/W, SMP

Exercise

Choose the correct answer from the given alternatives.

1. What is a vaccine?
 - a. A medicine that treats bacterial infections
 - b. A preparation that stimulates immunity against a specific disease
 - c. A chemical that kills pathogens in the environment
 - d. A dietary supplement for enhancing growth
2. What is the primary purpose of vaccination?
 - a. To treat infections
 - b. To prevent disease by inducing immunity
 - c. To kill external parasites
 - d. To improve feed conversion ratio
3. Which of the following is an example of a live attenuated vaccine?
 - a. Newcastle disease vaccine
 - b. Tetanus toxoid
 - c. Rabies inactivated vaccine
 - d. Foot-and-mouth killed vaccine
4. Which type of vaccine is safest for immunocompromised animals?
 - a. Live attenuated vaccine
 - b. Inactivated (killed) vaccine
 - c. Recombinant vaccine
 - d. DNA vaccine
5. At what temperature should most vaccines be stored?

a. -20°C	b. 2–8°C
c. Room temperature (25°C)	d. Above 30°C

6. What is the main reason for maintaining the cold chain during vaccine storage?
 - a. To prevent contamination
 - b. To preserve the efficacy of the vaccine
 - c. To ensure the vaccine becomes active
 - d. To save transportation costs
7. Which of the following is not a recommended practice for vaccine handling?
 - a. Shaking the vaccine vigorously before use
 - b. Protect vaccines from direct sunlight
 - c. Using a sterile syringe and needle for administration
 - d. Using reconstituted vaccines within a specified time
8. What should be done if a vaccine is accidentally frozen?
 - a. Warm it to room temperature and use immediately
 - b. Discard it and do not use
 - c. Mix it thoroughly to reactivate
 - d. Add a stabilizer before administration
9. Which disease is commonly vaccinated against in cattle?

a. Foot-and-mouth disease	b. Canine distemper
c. Coccidiosis	d. Marek's disease
10. At what age is the first dose of rabies vaccine generally administered to dogs?

a. At birth	b. 6–8 weeks
c. 12 weeks	d. 6 months

11. Which vaccine is part of the standard vaccination schedule for goats?
 - a. Peste des petits ruminants (PPR) vaccine
 - b. Gumboro vaccine
 - c. Newcastle disease vaccine
 - d. Infectious bursal disease vaccine
12. What is the typical vaccination schedule for Newcastle disease in broilers?
 - a. Day 1 and day 14
 - b. Day 7 and day 21
 - c. Day 1 and day 7
 - d. Day 14 and day 28
13. **At what age should layers be vaccinated against Marek's disease?**
 - a. 1 day
 - b. 1 week
 - c. 2 weeks
 - d. 4 weeks

Write short answer to the following questions.

1. Define vaccine. Write about its uses.
2. How is vaccine handled and stored?
3. Write the vaccination schedule of cattle and buffalo.
4. Write the vaccination schedule of goat.
5. Write the vaccination schedule of sheep.
6. Write the vaccination schedule of pig.
7. Write the vaccination schedule of dog.
8. Write the vaccination schedule of broilers.
9. Write the vaccination schedule of layers.

Project work

1. Make a vaccination schedule of farm animals and discuss about importance of vaccination schedule in your class. Also present in the class.

11.1 Introduction of Zoonotic Disease and Awareness Towards Zoonotic Disease

Zoonotic diseases, also known as zoonoses, are diseases that can be transmitted between animals and humans. These diseases are caused by various pathogens, including bacteria, viruses, fungi, and parasites, and can spread through direct contact with infected animals, their secretions, or contaminated environments. Some zoonoses are also transmitted through vectors such as mosquitoes, ticks, or fleas.

Zoonotic diseases pose significant public health challenges, as they can lead to outbreaks and epidemics with substantial economic and health impacts. Many emerging infectious diseases are zoonotic in origin, often linked to changes in human-animal interactions, environmental changes, and global trade and travel. Some examples of zoonotic diseases are Rabies, Anthrax, Leptospirosis, Brucellosis, Lyme disease, Toxoplasmosis, Listeriosis, Swine flu, SARS, Bird flu, Ranikhet, Parrot fever etc.

Importance of Zoonotic Diseases

Zoonotic diseases are critically important to human and animal health, as well as the economy and global ecosystems. Understanding and addressing zoonoses is essential for preventing outbreaks, protecting public health, and promoting sustainable interactions between humans and animals.

1. Public Health Significance

- **High disease burden:** Zoonoses contribute significantly to global

morbidity and mortality. Diseases like rabies, avian influenza, and leptospirosis continue to affect millions worldwide.

- **Emerging and re-emerging diseases:** More than 60% of emerging infectious diseases are zoonotic, including COVID-19, Ebola, and SARS. These diseases often arise from increased human-animal interaction and environmental changes.
- **Global pandemics:** Zoonotic pathogens like the H1N1 influenza virus have caused widespread pandemics, with devastating health and economic consequences.

2. Economic Impact

- **Loss of livestock and productivity:** Zoonotic diseases like brucellosis and anthrax lead to significant economic losses in agriculture due to livestock illness, reduced productivity, and culling of infected animals.
- **Healthcare costs:** Treating zoonotic diseases imposes a heavy financial burden on healthcare systems, particularly in resource-limited settings.
- **Trade restrictions:** Outbreaks of zoonoses can disrupt international trade, particularly in the livestock and food industries (e.g., bans on meat exports during foot-and-mouth disease outbreaks).

3. Food Security and Safety

- **Contaminated animal products:** Diseases like salmonellosis and toxoplasmosis can spread through infected meat, milk, or eggs, compromising food safety.
- **Impact on agriculture:** Zoonoses can reduce livestock productivity, threatening the livelihoods of farming communities and global food supply chains.

4. Biodiversity and Ecosystem Health

- **Wildlife conservation:** Many zoonotic diseases originate from wildlife (e.g., bats, rodents). Balancing conservation efforts with disease

prevention is essential for maintaining biodiversity.

- **Ecosystem disruption:** Disease outbreaks can alter predator-prey dynamics, disrupt ecosystems, and lead to cascading environmental consequences.

5. One Health Perspective

- **Interconnected health:** The health of humans, animals, and the environment is interconnected. Addressing zoonotic diseases requires a holistic approach, involving collaboration between medical, veterinary, and environmental professionals.
- **Global collaboration:** Zoonoses necessitate international cooperation for surveillance, research, and response, given their transboundary nature.

6. Threat to Vulnerable Populations

- **Rural and low-income communities:** These populations are often in close contact with animals and lack access to healthcare, making them more vulnerable to zoonotic diseases.
- **Occupational risks:** Farmers, veterinarians, and wildlife handlers face increased exposure to zoonotic pathogens.

7. Pandemic Preparedness

- **early warning systems:** Monitoring zoonotic diseases in animals provides an early warning for potential human outbreaks.
- **Vaccine Development:** Studying zoonotic pathogens enhances our ability to develop vaccines and therapeutics for both animal and human use.

Common Meat Borne Diseases

Meat-borne diseases are illnesses caused by consuming contaminated meat or meat products. These diseases are typically associated with bacterial, viral, parasitic, or chemical contaminants present in improperly handled, stored, or

cooked meat. Such diseases pose significant risks to public health globally. Some of the common meat borne diseases are:

Salmonellosis	Cholera	Swine flu
E. coli	Toxoplasmosis	Giardiasis
Listeriosis	Anthrax	Leptospirosis
Campylobacteriosis	Avian influenza	Hydatidosis
Taeniasis	Erysipelas	Hepatitis E

Common Milk Borne Diseases

Milk-borne diseases are illnesses caused by consuming raw or improperly processed milk or dairy products contaminated with pathogens. These diseases are a significant public health concern, especially in areas where raw milk consumption is common or dairy hygiene practices are inadequate. Contamination can occur from infected animals, unhygienic milking practices, or improper storage.

E. coli	Q fever	Toxoplasmosis
Brucellosis	Anthrax	FMD
Salmonellosis	Cryptosporidiosis	Tuberculosis

Exercise

Choose the correct answer from the given alternatives.

1. What are zoonotic diseases?
 - a. Diseases that affect only humans
 - b. Diseases that are transmitted from animals to humans
 - c. Diseases transmitted through air
 - d. Diseases caused by fungi

2. Which of the following is a zoonotic disease?

a. Malaria	b. Tuberculosis
c. Brucellosis	d. Diabetes

3. What is the primary mode of transmission for zoonotic diseases?
 - a. Direct contact with infected animals
 - b. Contaminated food and water
 - c. Vectors like mosquitoes and ticks
 - d. All of the above

4. Which organization monitors zoonotic diseases globally?
 - a. WHO (World Health Organization)
 - b. FAO (Food and Agriculture Organization)
 - c. OIE (World Organisation for Animal Health)
 - d. All of the above

5. Which of the following is a key step in preventing zoonotic diseases?
 - a. Vaccination of animals and humans
 - b. Avoiding direct contact with animals
 - c. Proper hygiene and sanitation practices
 - d. All of the above

6. Which of the following is a common milk-borne zoonotic disease?
 - a. Brucellosis
 - b. Rabies
 - c. Anthrax
 - d. Foot-and-mouth disease
7. Which of the following is a meat-borne zoonotic disease?
 - a. Trichinellosis
 - b. Listeriosis
 - c. Toxoplasmosis
 - d. All of the above
8. Which zoonotic disease is also known as "undulant fever" in humans?
 - a. Brucellosis
 - b. Anthrax
 - c. Leptospirosis
 - d. Q fever

Write short answer to the following questions.

1. Define zoonotic disease. Write about importance of zoonotic disease.
2. List the name of any five milk borne and meat borne disease.

Write long answer to the following questions.

1. Write any four examples of zoonotic disease. As a veterinary student what will be your role to prevent transmission of zoonotic disease in the society. Discuss with some of the points.

Project work

1. Prepare awareness pamphlet for zoonotic disease. And display in the class.

12.1 Introduction, Importance and Scope

Artificial Insemination (AI) is a reproductive biotechnology technique where semen, collected from a male animal, is processed and introduced into the reproductive tract of a female animal using specialized instruments. This is done without the natural act of mating, aiming to achieve pregnancy. AI is widely used in livestock, wildlife conservation, and human fertility treatments.

1. Genetic Improvement

- Selective Breeding**

AI enables farmers to use semen from genetically superior sires, improving desirable traits such as milk yield, growth rate, meat quality, and disease resistance.

- Global Genetic Exchange**

Facilitates access to high-quality semen from top-performing sires worldwide, promoting genetic diversity.

- Breed Preservation**

Helps in conserving and propagating native or endangered livestock breeds by ensuring controlled reproduction.

2. Increased Reproductive Efficiency

- Enhanced Fertility Management**

AI allows precise timing of insemination to coincide with the female's optimal fertility period, increasing conception rates.

- **Use of Frozen Semen**

Cryopreserved semen can be stored and used long after collection, ensuring year-round breeding independent of the male's availability.

- **Overcoming Natural Mating Barriers**

Useful for animals with physical or behavioral issues that prevent natural mating.

3. Disease Prevention

- **Control of Sexually Transmitted Diseases (STDs)**

AI minimizes the risk of spreading diseases such as brucellosis, leptospirosis, and trichomoniasis that occur during natural mating.

- **Hygienic Practices**

Semen is collected and processed under controlled conditions, reducing contamination risks.

4. Cost-Effective Breeding

- **Reduced Male Maintenance Costs**

Eliminates the need to maintain large numbers of breeding males, saving space, feed, and veterinary costs.

- **Efficient Use of Sires**

A single ejaculate can be processed into multiple doses, allowing one sire to inseminate hundreds of females.

5. Increased Production

- **Higher Milk Yield**

In dairy animals, AI contributes to breeding cows with superior genetics, enhancing milk production.

- **Better Meat Quality**

In beef cattle, AI is used to propagate traits like faster growth and higher carcass quality.

- **Wool and Fiber Improvement**

In sheep and goats, AI is applied to improve wool quality and fiber yield.

6. Crossbreeding Programs

- AI facilitates controlled crossbreeding to produce hybrid animals with superior performance, combining traits like higher productivity, better adaptability, and disease resistance.

7. Conservation of Rare Breeds

- Helps preserve genetic material from rare or endangered livestock breeds through semen collection, cryopreservation, and strategic breeding.

8. Synchronization of Breeding Programs

- **Timed Artificial Insemination (TAI)**

Allows simultaneous insemination of multiple animals in large herds, streamlining management.

- **Seasonal Breeders**

Ensures optimal use of breeding seasons, especially in sheep and goats.

9. Addressing Ethical and Practical Concerns

- **Reduced Stress on Animals**

AI eliminates the physical stress of repeated natural mating for both males and females.

- **Controlled Breeding**

Prevents unwanted pregnancies and ensures planned reproduction in farm animals.

10. Research and Development

- **Reproductive Studies**

AI is a valuable tool for studying animal reproductive biology, physiology, and endocrinology.

- **Advancements in Biotechnology**

Supports innovations like sexed semen, embryo transfer, and in vitro fertilization (IVF).

12.2 Advantages and Disadvantages

Advantages

1. There is no need of maintenance of breeding bull for a herd; hence the cost of maintenance of breeding bull is saved.
2. It prevents the spread of certain diseases and sterility due to genital diseases example contagious abortion and vibriosis.
3. By regular examination of semen after collection and frequent checking on fertility make early detection of inferior males and better breeding efficiency is ensured.
4. The progeny testing can be done at an early age.
5. The semen of a desired sire can be used even after the death of that particular sire.
6. The semen collected can be taken to the urban areas or rural areas for insemination.
7. It makes possible the mating of animals with great differences in size without injury to either of the animals.
8. It is helpful to inseminate the animals that refuse to accept the male at the time of oestrus.
9. It helps in maintaining the accurate breeding and calving records.
10. Semen can be used on tens of thousands of females a year instead of the actual bull being only able to cover 30 females in a breeding season.

11. Job is less dangerous because there is not a potentially dangerous bull to handle after every breeding season
12. The rate of genetic development and production gain can be increased, by using semen from males of high genetic merit for superior females.
13. It enables breeding between animals in different geographic locations.
14. Artificial Insemination can be used in conservation of rare breeds or endangered species.

Disadvantages

1. It requires well-trained operators and special equipment.
2. It requires more time than natural services.
3. Improper cleaning of instruments and insanitary conditions may lead to lower fertility.
4. If the bull is not properly tested, the spreading of genital diseases will be increased.
5. There is only a 60 to 70% conception rate for artificial insemination than with using a fertile herd bull
6. The operator needs to have adequate knowledge of heat periods of female stock and know what to look for to see if any stock is in heat and then judge the time to artificially inseminate them. There may be added stress involved when females have to be restrained
7. Artificial insemination isn't a job that can be learned by trial and error; it has to be taught first by a trained professional before it can be carried out in the field.
8. Semen has to be stored properly otherwise it will become no longer viable; improper handling will also render it in viable.
9. There has been a decline in fertility in dairy cattle and horses associated with an increase in Artificial Insemination.
10. The focus on certain individuals may result in loss of genetic variation.

12.3 Insemination Techniques

The techniques of artificial insemination are briefly described below:

1. Vaginal Speculum Techniques

The vaginal speculum technique is another commonly used to facilitate artificial insemination procedure in livestock, particularly in cattle. It is a relatively simple procedure that involves the use of a vaginal speculum to visualize the cervix and deposit semen directly into the reproductive tract. This technique is often preferred for its less invasive nature compared to other techniques such as recto-vaginal or uterine insemination.

Procedure of Vaginal Speculum AI Techniques

1. Preparation

- **Insemination Equipment**

The necessary equipment includes a sterile vaginal speculum, AI gun (a syringe-like device), and sterile gloves. Semen is either fresh or thawed if it is frozen.

- **Cleaning and Sanitation**

Before starting the procedure, it is essential to clean the vulva and surrounding area of the female to avoid contamination.

2. Positioning of the Animal

- The animal is typically restrained in a standing position, either in a stanchion or a similar device that allows for easy access to the vaginal and cervical area. This minimizes movement and reduces stress during the procedure.

3. Insertion of the Vaginal Speculum

- The operator uses the speculum to open the vagina and provide a clear view of the cervix. The speculum is a cylindrical device that is gently inserted into the vaginal canal. It is designed to hold open the vaginal walls and allow the operator to see the cervix.

- **Lighting**

Good lighting is crucial for visualizing the cervix accurately, as the speculum reflects light to illuminate the area.

4. Semen Insemination

- Once the cervix is visible through the speculum, the inseminator uses an AI gun to introduce the semen into the cervix. The AI gun is inserted through the speculum and directed toward the cervical opening.
- The semen is deposited just below the cervix into the uterine body or the cervical canal, depending on the species and reproductive goals.

5. Post-insemination

- After the semen is deposited, the speculum is carefully withdrawn, and the animal is allowed to stand quietly for a few minutes to ensure that the semen is properly distributed in the reproductive tract.
- The inseminator may monitor the animal for signs of pregnancy in the following weeks.

2. Recto-vaginal Techniques of Artificial Insemination (AI)

The recto-vaginal techniques of artificial insemination (AI) is one of the most common techniques used in livestock, especially in cattle, sheep, goats, and horses. This method allows the inseminator to guide semen into the reproductive tract of the female animal using a combination of manual rectal and vaginal manipulation. It is a precise technique that requires skilled handling to ensure successful semen deposition in the proper location.

Procedure of Recto-vaginal AI Techniques

1. Preparation

- **Insemination Equipment**

The equipment includes an AI gun (a syringe-like tool for depositing semen), a sterile catheter, and gloves. The semen is collected (fresh or

frozen) from the male animal (sire) and is either used immediately or thawed if it is frozen.

- **Cleaning and Sanitation**

The reproductive area of the female is cleaned to prevent infection or contamination. It is essential to maintain sterile conditions during the procedure.

2. Positioning of the Animal

- The female animal is often restrained in a standing position (for cattle) or placed in a position that allows easy access to both the rectum and vagina. Sometimes, the animal may be restrained in a stanchion or holding pen for proper control.

3. Rectal Palpation

- The inseminator puts a gloved hand into the rectum of the animal. Through rectal palpation, the cervix is located. The operator uses their fingers to identify the location and orientation of the cervix, which helps in guiding the AI gun through the vaginal canal and cervix.

4. Vaginal Insertion

- With the other hand, the operator inserts the insemination gun (with the semen) into the vagina. The gun is directed towards the cervix, which is felt through the rectal hand. The inseminator carefully guides the gun through the cervix and into the uterus.

5. Insemination

- Once the insemination gun is in position, the semen is deposited into the uterus or just below the cervix (depending on the species and reproductive cycle). The semen is typically released slowly to allow for proper distribution.

6. Post-insemination

- After semen deposition, the AI gun is withdrawn, and the female is monitored for signs of pregnancy. The operator may also check for any signs of trauma or infection.

Exercise

Choose the correct answer from the given alternatives.

1. What is artificial insemination (AI)?
 - a. Natural mating between animals
 - b. Placement of sperm into the female reproductive tract without natural mating
 - c. Surgical removal of reproductive organs
 - d. Genetic modification of livestock
2. Which of the following is an important advantage of AI in farm animals?
 - a. Increased transmission of diseases
 - b. Controlled use of superior genetics
 - c. Decreased efficiency in breeding
 - d. Reduced reproductive rates
3. In which species is Artificial insemination is widely used in Nepal?

a. Dairy cattle	b. Poultry
c. Sheep	d. Buffalo
4. Why artificial insemination is considered as important in modern livestock management?
 - a. It eliminates the need for male animals on farms
 - b. It ensures the use of high-quality semen from genetically superior sires
 - c. It increases the likelihood of twin births
 - d. It allows for random genetic selection
5. Who is credited with the development of AI as a scientific practice?

a. Robert Koch	b. Ilya Ivanovich Ivanov
c. Louis Pasteur	d. Norman Borlaug

6. Which of the following is a sign that indicates a cow is in heat and ready for AI?
 - a. Decreased appetite
 - b. Stand still when mounted by another animal
 - c. Constant lying down
 - d. Aggression toward other animals
7. What is the main benefit of using frozen semen in AI?
 - a. Longer storage and transport to distant locations
 - b. Reduced genetic diversity
 - c. No need for estrus detection
 - d. Elimination of all technical challenges
8. In poultry, in what part of the reproductive tract is semen is deposited during artificial insemination?

a. Cervix	b. Cloaca
c. Uterus	d. Ovary
9. What is the most common method of artificial insemination in cattle?
 - a. Cervical insemination
 - b. Recto-vaginal technique
 - c. Intrauterine laparoscopic insemination
 - d. Natural insemination
10. What is the ideal temperature for thawing frozen semen in cattle?

a. 25°C	b. 35°C–37°C
c. 15°C–20°C	d. 40°C–45°C

Write short answer to the following questions.

1. Define artificial insemination. Write about its scope in Nepal.

2. Define thawing. What are the advantages of artificial insemination?
3. Write the disadvantages of artificial insemination.

Write long answer to the following questions.

1. Describe the different techniques of artificial insemination.
2. Write about advantages and disadvantages of artificial insemination.

Project work

1. Make a list of advantages and disadvantages of artificial insemination and present it in your class.
2. Discuss various techniques of artificial insemination and present it in a chart.

References

ABS (American Breeders Service Inc.). 1972. *A.I. management manual*. De Forest, Wisconsin, USA. 197 pp.

Akinboade O A. 1980. Incidence of bovine trichomonisis in Nigeris. *Revue d'Elevage et de Médecine Vétérinaire des Pays Tropicaux* 33: 381-384.

Alton G G. 1981. The control of bovine brucellosis; recent developments. *World Animal Review* 39: 17-24.

Arthur G H. 1964. *Wright's veterinary obstetrics*. 3rd edition. Bailleire Tindall and Cassell, London, UK. 549 pp.

Arthur G H. 1982. *Veterinary reproduction and obstetrics*. 5th edition. Bailleire Tindall, London, UK. 616 pp.

Ball M G. 1966. Animal hosts of Leptospires in Kenya and Uganda. *American Journal of Tropical Medicine and Hygiene* 15: 523-530.

Banerjee A K. 1966. A study of the action of Terramycin on the bacterisl flora of the uterus in cattle following retained placenta. Thesis, University of Utrecht, Utrecht, The Netherlands. 118 pp.

Basile J R and Megale F. 1974. Developmental abnormalities of the genitalis of Zebu cows in the state of Minas Geriss. *Argentina Biologica e Tecnologica* 17: 136-150 (*Animal Breeding Abstracts* 44: 3133).

Bell R A. 1984. The most significant genital diseases of cattle in Great Britain. In: *Proceedings of the 11th Conference of the OIE Regional Commission for Europe, 25-28 September 1984, Vienna, Austris*. OIE (Office international des épizooties), Paris, France. pp. 223-241.

Brinker WO: Types of fractures and their repair. In Archibald J (ed): *Canine*

Surgery, 2nd ed, pp 957- 960. Santa Barbara, American Veterinary Publications, 1974

Bhatt G N. Vyas K K, Purohit S K and Jatkar R P. 1979. Studies on immuno-infertility in repeat breeder cows. *Indian Veterinary Journal* 56: 184-188.

Binemo-Madi C and Mposhy M. 1982. Study of sterility in cows on ranches in Shoa, Zaire. *Revue d'Elevage et de Médecine Vétérinaire des Pays Tropicaux* 35: 281-284.

Blood D C, Henderson J A and Radostits O M. 1979. *Veterinary medicine*. 5th edition. Bailliere Tindall, London, UK.

Bolinder A, Seguin B, Kindahl H, Bouley D and Otterby D. 1988. Retained fetal membranes in cows: Manual removal versus nonremoval and its effect on reproductive performance. *Theriogenology* 30: 45-56.

Britt J H, Harrison D S and Morrow D A. 1977. Frequency of ovarian follicular cysts, reasons for culling, and fertility in Holstein-Friesian cows given gonadotrophin-releasing hormone at two weeks after parturition. *American Journal of Veterinary Research* 38: 749.

Boyd H. 1977. Anoestrus in cattle. *Veterinary Record* 100: 150-153.

Carroll E J. 1972. Control of diseases affecting reproduction in beef cattle. Proceedings of the 21st and 22nd Beef Cattle Short Course, Texas A and M University, August 1972. College Station, Texas, USA. pp. 32-49.

Carroll E J. 1973. Disease and reproduction in beef cattle. Lecture notes. Colorado State University, Fort Collins, Colorado, USA.

Casagrande J F and Goes N F. 1977. Use of PGF₂ for the treatment of Nellore repeat breeders. *Científica, Brazil* 5: 344-347 (Veterinary Bulletin 48: 6276).

Casida L E and Chapman A B. 1951. Factors affecting the incidence of cystic

ovaries in a herd of Holstein cows. *Journal of Dairy Science* 34: 1200.

Casida L E, McShan W H and Meyer R K. 1944. Effects of an unfractionated pituitary extract upon cystic ovaries and nymphomania in cows. *Journal of Animal Science* 3: 273-282.

Chauhan F S, Mgongo F O K and Kessey B M. 1984. Recent advances in normal therapy in bovine reproductive disorders: A review. *Veterinary Bulletin* 54: 991-1009.

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Chenna R M C. 1980. A case of uncommon voluminous vaginal fibroma in a nondescript cow. *Indian Veterinary Journal* 57: 861.

Chukwu C C. 1985. Brucellosis in Africa. Part 1. The prevalence. *Bulletin of Animal Health and Production in Africa* 33: 193-198.

Chukwu C C. 1987. Brucellosis in Africa. Part 2. The importance. *Bulletin of Animal Health and Production in Africa* 35: 92-98.

Clark B L. 1971. Review of bovine vibriosis. *Australian Veterinary Journal* 47: 103-107.

Clark B L and Dufty J H. 1978. Isolation of *Campylobacter fetus* from bulls. *Australian Veterinary Journal* 54: 262-263.

Dafalla E N. 1962. Incidence of animal and human brucellosis in the Sudan. *Sudan Journal of Veterinary Science and Animal Husbandry* 3: 80-88.

De B N, Chatterjee A, Bidyanta J, Chakraborty M, Deb S K, Mondal P and Sen G. P. 1982. Note on the problems of breeding cows with special reference to common coital infections. *Indian Journal of Animal Sciences* 52: 700-702.

Deas W. 1981. Non-brucella abortion in cattle. *In Practice* 3: 14-19.

Denny HR: A Guide to Canine Orthopaedic Surgery. Oxford, Blackwell Scientific Publications, 1980

DePalma AF: The Management of Fractures and Dislocations, vol 1 and 2. Philadelphis, WB Saunders, 1959

Disz R. Garatea P. Jones L M and Moriyani I. 1979. Radisl immunodiffusion test with a Brucella polysaccharide antigen for differentisting infected from vaccinated cattle. *Journal of Clinical Microbiology* 10: 3 7-41.

Dufty J H and McEntee K. 1969. Evaluation of some culture medis and sampling techniques for the disgnsis of vibriosis in the bull. *Australisn Veterinary Journal* 45: 140-144.

Eduvie L O. Osori D I K, Addo P B and Njoku C O. 1984. Bacteriological investigation of the postpartum uterus: Relationship to involution and histopathological findings. *Theriogenology* 21: 733-745.

El-Azab M A, Kakoma I, Brodie B O. McKenna D J and Gustafusson K. 1988. Bacteriology of the postpartum bovine uterus with retained placenta and induced metritis: Specisl reference to minimum inhibitory concentration (MIC). In: *Proceedings of the 11th International Congress on Animal Reproduction and Artificisl Insemination, 26-30 June 1988*. University College, Dublin, Ireland. p. 513.

Ellis W A and Little T W A (eds). 1986. *The present state of leptospirosis disgnsis and control*. Proceedings of the Seminar of the EEC Programme of Coordination of Research on Animal Pathology, 10-11 October 1984, Belfast, Northern Ireland. Martinus Nijhoff Publishers, Dordrecht/ Boston/Lancaster, for the Commission of the European Communities. 247 pp.

Elmore R G. Bierschwal C J. Youngquist R S. Cantley T C, Kesler D J and

Garverick H A. 1975. Clinical responses of dairy cows with ovarian cysts following treatment with 10,000 HCG or 100 MCG GnRH. *Veterinary Medicine/Small Animal Clinicism* 70: 1346-1349.

Enkhis K L, Kohli I S and Bhatis J S. 1983. Electrolytes of cervico-vaginal mucus and blood during oestrus in normal and repeat-breeding Rathi cows. *Indian Journal of Animal Science* 53: 66-68.

Erb H N and Martin S W. 1980. Interrelationships between production and reproduction diseases in Holstein cows. *Journal of Dairy Science* 63: 1911-1917.

Erb R E, Hinze P M and Gildow E M. 1959. Factors influencing prolificacy of cattle. II. Some evidence that certain reproductive traits are additively inherited. *Washington Agricultural Experimental Station Bulletin* No. 30.

Esoruoso G O. 1974. Bovine brucellosis in Nigerians. *Veterinary Record* 95: 54-58.

Fazeli M, Ball L and Olson J. D. 1980. Comparison of treatment of pyometra with oestradiol cypionate or cloprostenol followed by infusion or non-infusion with nitrofurozone. *Theriogenology* 14: 339-347.

Fensterbank R. 1986. Brucellosis in cattle, sheep and goats: Diagnosis, control and vaccination. *Revue scientifique et technique de l'Office international des épizooties* 5: 605-618.

Florent A. 1963. Viral infertility. In: *Livestock infertility*. Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 36-43.

Foley J W, Bryner J H, Hughes D E and Bastard R D. 1979. Improved method for diagnosis of *Campylobacter fetus* infection in cattle using selective

enrichment transportation medium. *Proceedings of the American Association of Veterinary Laboratory Diagnosis* 22: 367-372.

Garcis M M, Eaglesome M D and Rigby C. 1983. Campylobacters important in veterinary medicine. *Veterinary Bulletin* 53: 793-818.

Gartland JJ: Fundamentals of Orthopaedics, Philadelphis, WB Saunders, 1965

Gawade A F A, Nada S M and Micheal D G. 1981. Incidence of *Trichomonas foetus* infection in bulls in Sharkya governorate, Egypt. *Assiut Veterinary Medicine Journal* 8: 89-91.

Gledhill B L. 1968. Viral infertility in cattle. *Cornell Veterinaris* 57: 466-479.

Grunert E. 1984. Placental separation/retention in the bovine. *Proceedings of the 10th International Congress on Animal Reproduction and Artificial Insemination, 10-14 June 1984, University of Illinois, UrbanaChampaign, Illinois, USA.* IV: XI 17-24.

Hernendez-Ledezma J J, Arenas P. Dominquez L F and de Fernandez C L. 1984. Factors associsted with the occurrence of ovarisn cysts in dairy cattle. *Tecnica Pecuris en Mexico* 1984(47): 88-94.

Hoerlein A B. 1970. Vibriosis. In: W J Gibbons, E J Catcott and J F Smithcors (eds), *Bovine medicine and surgery*. American Veterinary Publications, Wheaton, Illinois, USA. p. 91.

Hoerlein A B. Carroll E J. Kramer T and Beckenhaeur. 1965. Bovine vibriosis immunization. *Journal of the American Veterinary Medical Association* 146: 828.

Hughes D E. 1953. A study of the disgnois of bovine vibriosis with specisl reference to the detection of agglutinins in the vaginal secretions. *Cornell Veterinaris* 43: 431-444.

Hussain P M and Muniraju L. 1984. Study in the incidence of reproductive

disorders of bovines in a part of Southern Kartanaka. *Livestock Adviser* 9: 1316.

Jackson P S. 1977. Treatment of chronic postpartum endometritis in cattle with cloprostenol. *Veterinary Record* 101: 441-443.

Johannson I. 1960. Genetic causes of faulty germ cells and low fertility. *Journal of Dairy Science* 43 (Supplement):

JennyJ: Orthopaedic Notes. Unpublished, University of Pennsylvanis, 1970

Kagumba M and Nandhoka E. 1978. A survey of the prevalence of bovine brucellosis in East Africa. *Bulletin of Animal Health and Production in Africa* 26: 224-229.

Kaikini A S. Chikalikar G K and Dindorkar C V. 1983. Reproductive disorders in Holstein-Friesian x Gir F₁ crossbred cows. *Indian Journal of Animal Sciences* 53: 556-558.

Kaneene J M B. Nicoletti P. Anderson R K, Muscoplat C C and Johnson D W. 1979. Cell-mediated immune responses in adult cattle vaccinated with *Brucella abortus* Strain 19 and in cattle infected with *Brucella abortus* field strain. *American Journal of Veterinary Research* 40: 1503.

Kesler D J and Garverick H A. 1982. Ovarian cysts in dairy cattle: A review. *Journal of Animal Science* 55: 1147-1159.

Kesler D J. Garverick H A, Candle A B. Bierschwal C J. Elmore R G and Youngquist R S. 1978. Clinical and endocrine responses of dairy cows with ovarian cyst to GnRH and PGF_{2α}. 46: 719-725.

Klastrup N O and Halliwell R W. 1977. Infectious causes of infertility/abortion of cattle in Malawi. *Nordisk Veterinaermedicin* 29: 325-330.

Kodagali S B. 1974. Report on study of infertility in cattle 1965-69. *Veterinary Bulletin* 45: 1285 (Animal Breeding Abstracts 43: 3369).

Kumi-Diska J, Ogwu D and Osori D I K. 1981. Significance of atrophic ovaries in livestock production in northern Nigeris. *Veterinary Record* 108: 277278.

Lagerlof N. 1963. Hereditary factors in infertility in cattle. In: *Infertility in livestock*. Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 63-77.

Laing J A (ed). 1960. *Vibrio fetus* infection of cattle. FAO Agricultural Study Publication No. 51. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy.

Laing J A. 1963. *Vibrio fetus* infection. In: *Infertility in livestock* Animal Health Branch Monograph No. 5. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. pp. 10-17.

Lambert G, Deyoe B L and Painter G M. 1964. Postvaccinal persistence of *Brucella abortus* strain-19 in bulls. *Journal of the American Veterinary Medical Association* 145: 909.

Lamming G E. 1977. Annual Report of the Milk Marketing Board of England and Wales, Thames Ditton, Surrey, UK.

Leonard EP: Orthopaedic Surgery of the Dog and Cat, 2nd ed, pp 90-94. Philadelphis, WB Saunders, 1971

Little D A. 1976. Assessment of several pasture species, particularly tropical legumes, for oestrogenic activity. *Australisn Journal of Agricultural Research* 27: 681-686.

Marinov P and Boehnel H. 1976. A viral infection connected with infertility in cattle in Tanzanis. *Bulletin of Animal Health and Production in Africa*] 24: 19-28.

Mathei C A and Deyoe B L. 1970. Brucellosis. In: W J Gibbons, E J Catcott and

J F Smithcors (eds), *Bovine medicine and surgery*. American Veterinary Publications, Wheaton, Illinois, USA. p. 104.

McKercher D G. 1969. Relationship of viruses to reproductive problems. *Journal of the American Veterinary Medical Association* 154: 1184-1191.

Menge A C, Mares S E, Tyler W J and Casida L E. 1962. Variation and association among postpartum reproduction and production characteristics in Holstein-Friesian cattle. *Journal of Dairy Science* 45: 233.

Merck Veterinary Manual <http://www.merckvetmanual.com/digestive-system/intestinal-diseases-in-ruminants/disrrhoea-in-neonatal-ruminants>

Meyer C E. 1980. Report on veterinary activities. Institute of Agricultural Research, Ethiopia. FAO Report No. AG: DP/ETH/78/004. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. 24 pp.

Moch R W, Ebner E E, Barsoum I S and Botros B A M. 1975. Leptospirosis in Ethiopia: A serological survey in domestic and wild animals. *The Journal of Tropical Medicine and Hygiene* 78: 38-42.

Mohanty B N, Parihar N S and Luktude S N. 1980. Tuberculosis of genital organs causing repeat breeding in cattle. *Indian Veterinary Journal* 57: 859-860.

Munoz de Cote J C, Velazquez E A, Garza R J and Valencis M J. 1980. Immunological aspects of infertility in cows and its effects on breeding. *Veterinaria (Mexico City)* 11: 63-70.

Mustafa A A and Nur B M. 1968. Bovine brucellosis in the Sudan: A survey in the Gash and Tokar areas of Kasala Province. In: *Proceedings of the 3rd Veterinary Conference. Sudan Veterinary Association, Khartoum, 5-7 January 1968*. Khartoum, Sudan.

Mustafa A A, Ghalib H W and Sinnary K A B. 1976. Incidence of bovine brucellosis in Southern Kordofan province. *Sudan Journal of Veterinary Science and Animal Husbandry* 16: 75-80.

Namboothripad T R B and Raja C K S V. 1976. A study of the aetiology of repeat breeding in cows. *Kerala Journal of Veterinary Science* 7: 195.

NCSR (National Council for Scientific Research). 1970. Factors affecting the calving rate in local breeds of cattle in Zambis. Animal Production Research Report NCSR/TR 7. NCSR, Lusaka, Zambis. 20 pp.

Nuru S and Dennis S M. 1976. Abortion and reproductive performance of cattle in Northern Nigeris: A questionnaire survey. *Tropical Animal and Health and Production* 8: 213-219.

Osmanu S T. 1979. Studies on bovine infertility at the Agricultural Research Station (Legon) over half a decade (1972-77). Ghana University, Department of Animal Science Studies, Legon, Ghana. 82 pp.

Paisley L G. Mickeesen W D and Anderson P B. 1986. Mechanisms and therapy for retained fetal membranes and uterine infections of cows: A review. *Theriogenology* 25: 353-381.

Pandey S K, Pandit R K and Chandary R A. 1982. Note on productive and reproductive efficiency in Tharparkar cows and their causes. *Indisn Journal of Animal Sciences* 52: 691-692.

Perkins J R. Olds D and Seath D M. 1954. Study of 1000 bovine genitalis. *Journal of Dairy Science* 37: 1158-1163.

Polydorou K. 1984. The most important genital diseases of cattle (control, treatment and hygiene of semen collection). In: *Proceedings of the 11th conference of the OIE Regional Commission for Europe. 25-28 September 1984, Vienna, Austris*. OIE (Office international des épizooties), Paris, France. pp. 219-221.

Rao RA, Rao N P and Rao A S P. 1965. Some observations on genital abnormalities of cattle. *Indian Veterinary Journal* 42: 751-754.

Rao P R, Rakha B S and Parihar N S. 1977. Pathology of repeat breeding cows. *Indian Journal of Animal Sciences* 45: 943-948.

Roberts S J. 1955. Clinical observations on cystic ovaries in dairy cattle. *Cornell Veterinarian* 45: 497-513.

Roberts S J. 1956. *Veterinary obstetrics and genital diseases*. 1st edition. Edwards Brothers, Ann Arbor, Michigan, USA.

Roberts S J. 1971. *Veterinary obstetrics and genital diseases*. 2nd edition. Published by the author. Distributed by Edwards Brothers, Ann Arbor, Michigan, USA. 776 pp.

Schurig G D, Hall C E, Burda K, Corbeil L B, Duncan J R and Inter A J. 1973. Persistent genital tract infection with *Vibrio fetus intestinalis* associated with serotypic alteration of the infecting strain. *American Journal of Veterinary Research* 34: 1399-1403.

Segel C L, Lank R B and Levy H E. 1966. Dihydrostreptomycin for treatment of genital vibriosis in the bull. *Journal of the American Veterinary Medical Association* 149: 1634.

Singh C S P, Singh S K and Singh B. 1981. Studies on the incidence of infertility in cows. *Indian Veterinary Journal* 58: 909-912 (Veterinary Bulletin 51: 7266).

Singh A, Taylor C M and Singh B N. 1983. Factors affecting calving interval in Malvi cattle. *Livestock Adviser* 8: 9-11.

Smith T. 1918. Spirilla associated with disease of the foetal membranes in cattle (infectious abortion). *Journal of Experimental Medicine* 28: 701-719.

Stemshorn B W, Nielsen K H, Samagh B S, Forbes L B and Ingram D G. 1980.

Evaluation of an enzyme-labelled antiglobulin test for anti-Brucella immunoglobulin G among three cattle populations. *American Journal of Veterinary Research* 41: 1779.

Stoenner H G. 1968. Bovine leptospiral abortion. In: L C Faulkner (ed.), *Abortion diseases of livestock*. Charles C Thomas, Springfield, Illinois, USA.

Tedesco L, Errico F and Del Boulglivi L P. 1977. Comparison of three sampling methods for the diagnosis of genital vibriosis in the bull. *Australian Veterinary Journal* 53: 470-472.

Tekelye Bekele, Kasali O B. Mukasa-Mugerwa E, Scholtens R G and Tamrat Yigzaw. 1989a. The prevalence of brucellosis in indigenous cattle in central Ethiopia. *Bulletin of Animal Health and Production in Africa* 37: 97-98.

Tekelye Bekele, Kasali O B. Scholtens R G and Mukasa-Mugerwa E. 1989b. Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis (IBR/IPV) in cattle in central Ethiopia. *Bulletin of Animal Health and Production in Africa* 37: 31-33.

Tennant B and Peddicord R G. 1968. The influence of delayed uterine involution and endometritis on bovine fertility. *Cornell Veterinarism* 58: 158-192.

Terblanche J. 1979. Bovine vibriosis. *Rhodesia Agricultural Journal* 76: 43-45.

Todd JD, Volnec F J and Paton I M. 1971. Intranasal vaccination against infectious bovine rhinotracheitis: Studies on early onset of protection and use of vaccine in pregnant cows. *Journal of the American Veterinary Medical Associstion* 159: 1370.

Trichard C J V and Jacobsz E P. 1985. Mycoplasmas recovered from bovine genitalis, aborted foetuses and placentas in the Republic of South Africa. *Ondersterpoort Journal of Veterinary Research* 52: 105-110.

Vale W G, Ohashi O M, Ribiero H F L and Sousa J S. 1984. Causes and incidence of infertility and subfertility in zebu crossbred cows in the Amazon region of Brazil. *Veterinary Medical Review* 2: 133-143 (*Veterinary Bulletin* 55: 3059).

Vandeplassche M. 1982. *Reproductive efficiency in cattle: A guideline for projects in developing countries*. FAO Animal Production and Health Paper No. 25. FAO (Food and Agriculture Organization of the United Nations), Rome, Italy. 118 pp.

Waghela S. 1976. Animal brucellosis in Kenya: A review. *Bulletin of Animal Health and Production in Africa* 24: 53.

Watson P F. 1979. The preservation of semen in mammals. *Oxford Review of Biology* 1: 283.

Whittick WG: Canine Orthopaedics, pp 127-130. Philadelphia, Lea and Febiger, 1974 http://www.lsuagcenter.com/en/crops_livestock/livestock/animal_health/beef_cattle/Bovine+Viral+disrrhoea+Virus+B+VD+of+Cattle.htm for more information on BVD.