Common Zoonotic and Animal Diseases in Oromia, Amhara, SNNPR and Tigray Regions

Training of Trainers for CIGs

Technical Manual

November 2020

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MINISTRY OF AGRICULTURE

AGRICULTURAL GROWTH PROGRAM

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ABBREVIATIONS

AHA	Animal Health Assistant
CIG	Common Interest Group
CBPP	Contagious Bovine Pleuropneumonia
CCPP	Contagious Caprine Pleuropneumonia
CRD	Chronic Respiratory Disease
MG	Mycoplasma gallisepticum
ND	Newcastle Diseas
PPR	Peste des petits ruminants
PTT	Participative Training Techniques
TNA	Training Need Assessment
ToT	Training of Trainer

Introduction

In an attempt to deliver standard and appropriate training to CIGs, this ToT manual concentrates on the technical content to support the participative approaches and methods for CIGs trainers. Trainers also need to receive guidance on how to plan and evaluate training sessions which will be provided in separate accompanying training materials. The manual concentrates on the technical content based on the identified training needs of the CIG and is presented in the format of training sessions to complement the ToT training.

The principles and tools set out are based on practical situations and best-practice recommendations. Once veterinarians are trained on this ToT manual, they will train CIGs using the facilitator's guide and this technical manual. The objective of the Training of Trainers is to provide CIG trainers with the necessary practical skills that will enable them to facilitate CIGs training programmes on zoonotic and animal diseases.

Session 1. Zoonotic Diseases Prevention and Control

Objective: At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control and prevention of locally important zoonotic diseases

Time: 6 hours

It is essential that control and prevention of locally important zoonotic disease and public health hazards are practiced by all trainees. The control method of zoonotic diseases varies according to the disease in question. Therefore, where a specific zoonotic disease is a problem, trainees should be provided sufficient training on the problems and the massage and its extension approach. This may include: hygienic measures while dealing with livestock and information on locally important zoonotic diseases such as milk-borne zoonosis, consumption of meat from sick animals; disposal of anthrax carcasses and control of stray dogs and rabies.

Session 1.1 Brucellosis

Objective: At the end of the training the trainees should be able to describe clinical signs, transmission mechanism, prevention and control of brucellosis.

Time 1 hour

Cause and Transmission of Brucellosis:

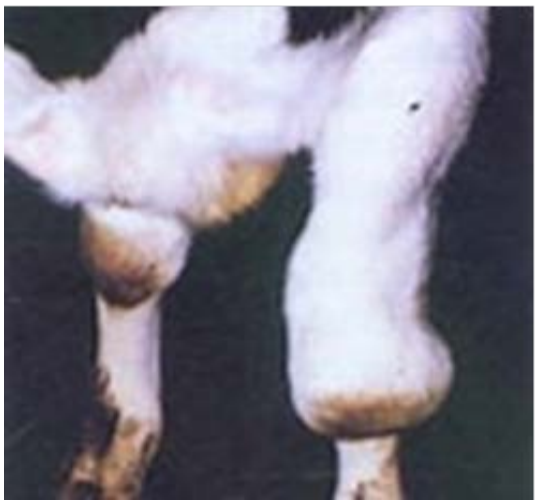
- Brucellosis is caused by Brucella Melitensis in /goats, Brucella ovis in sheep, and Brucella Abortus in cattle.
- Large and small ruminants and humans are susceptible to Brucella.
- Infection is usually introduced into a herd through latently or acutely infected animals.
- Infection occurs mostly by ingestion of materials which has been contaminated with the excretion of a female which has been aborted but may also have had normal birth.
- Contaminated feed (hay) can spread the disease from infected pastures over large distances.
- Infection occurs mostly during kidding, lambing and calving seasons.
- Brucella is excreted in milk, urine and feces, fetus, placenta and vaginal discharge for 2-3 months after abortion or parturition.

Symptoms and disease in people

- People can contract the disease from milk, milk products and meat, as well as urine, feces, fetus, placenta, vaginal discharge or other contaminated materials.
- Infection takes place through mucosa or the injured and/or intact skin, through the oral ingestion of contaminated feed and food.
- Brucella infection is readily transmissible to humans, causing acute febrile illness (undulant fever) which may progress to a more chronic form
- People can contract the disease from milk, milk products and meat, as well as urine, feces, fetus, placenta, vaginal discharge during calving/lambing/kidding or other contaminated materials.
- Infection takes place through mucosa or the injured and/or intact skin, through the oral ingestion of contaminated feed and food.
- Intermittent fever over months or years accompanied by debilitating anorexia, weight loss, abdominal and joint pain, headache, backache, weakness, irritability, insomnia, depression, and emotional instability may occur.
- Humans are treated with antibiotics, however, relapse are possible.

Clinical Signs in animals:

- Significant reduction in productivity such as late first calving age, long inter-calving time, below 60% herd fertility and comparatively low milk production.
- Abortion occurs in the last 2 months of pregnancy in most of the cases.
- A mid-to late abortion storm when the disease is first introduced.
- Placental retention.
- If the infection is introduced into a non-infected herd in which all animals are immunologically naïve abortion storms may occur.
- Inflammation of testes (orchitis).
- Joint swelling.



Diagnosis of Brucellosis:

- Laboratory isolation of the organisms from fetus, placenta, or vaginal discharges.
- Rose Bengal plate test (RBT) for milk is useful survey test that can be used in farm and at market.
- Laboratory serological test from blood samples

Prevention and Control of brucellosis:

- In an infected herd it is important to decrease natural challenge by appropriate hygiene at kidding and milking time.
- Assist with kidding/lambing with gloves if possible
- Placenta and aborted fetuses should be handled carefully and buried deeply or burned.
- Purchase of unknown breeding animals should be avoided.
- Pasteurization of milk and the usual disinfectants can destroy Brucella.
- In Ethiopia there is no government vaccination program. However, vaccination especially of kids and lambs 3 to 8 months of age using live attenuated B. melitensis Rev 1 vaccine is possible where available. The vaccine will cause abortion, and thus is to be avoided in pregnant and or those within 1 month before mating. Immunity from a single dose is considered to be lifelong, but is not absolute.
- Test and slaughter infected animals where possible.

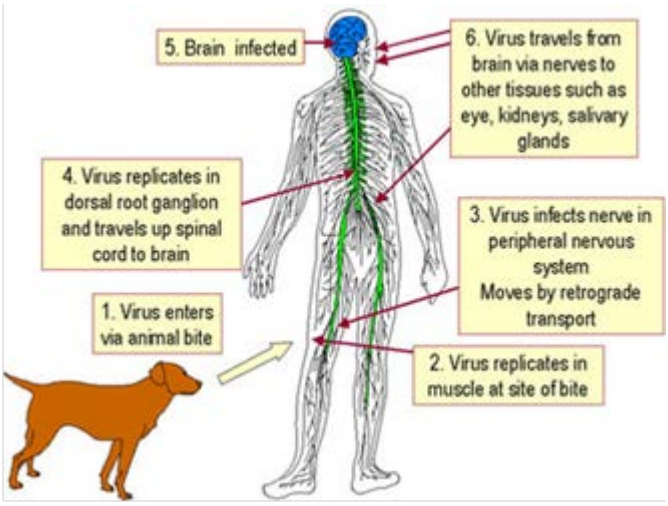
Session 1.2 Rabies

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism, prevention and control of rabies.

Time ***1 hour***

Causes and Transmission of Rabies:

- Rabies infection is caused by a virus.
- Rabies is a deadly virus spread to people from the saliva of infected animals.
- The rabies virus is usually transmitted through a bite through the saliva of infected animals.
- Infected animals can spread the virus by biting another animal or a person.
- Once a person begins showing signs and symptoms of rabies, the disease nearly always causes death. For this reason, anyone who may have a risk of contracting rabies should receive rabies vaccinations for protection.
- Any mammal (an animal that suckles its young) can transmit the rabies virus.
- The animals most likely to transmit the rabies virus to people include: Cats, Cows, Dogs, Goats, Donkey and Horses, Wild animals, Bats, etc.
- **Signs in dogs**: strange behavior, agitation, seizure, difficulty in drinking, hydrophobia (afraid of water), spasms of pharynx produces choking and death in 1-6 days



Symptoms of Rabies in people:

- In rare cases, rabies can be spread when infected saliva gets into an open wound or the mucous membranes, such as the mouth or eyes. This could occur if an infected animal were to lick an open cut on a person's skin.
- Signs and symptoms may include:
  - Fever
  - Headache
  - Nausea Vomiting, Agitation, Anxiety, Confusion, Hyperactivity, Difficulty swallowing, Excessive salivation, Fear brought on by attempts to drink fluids because of difficulty swallowing water, Hallucinations, Insomnia,
  - Partial paralysis, Death

Diagnosis and Treatment of Rabies in people:

- If an animal bites you, go to the doctor for the wound immediately.
- Wash your wound gently and thoroughly with soap and generous amounts of water.
- If you've been bitten by an animal that is known to have rabies, you'll receive a series of streptomycin shots to prevent the rabies virus from infecting you.
- If the animal that bit someone can't be found, it may be safest to assume that the animal has rabies depending on the type of animal, its history and the situation in which the bite occurred.
- The doctor may ask: What animal bit you? Was it a wild animal or a pet? If it was a pet, do you know who owns the pet? Was it vaccinated? Can you describe the animal's behavior before it bit you? Was the animal provoked? Were you able to capture or kill the animal after it bit you?
- If the animal that bit you can be contained or captured without causing more injury, do so. Do not kill the animal with a blow or a shot to the head, as the resulting injuries may make it difficult to perform laboratory tests to determine whether the animal has rabies.
- Tell your doctor that you have captured the animal that bit you. Your doctor may then contact the local health department to determine what to do with the animal.
    - Seek immediate medical care if you're bitten by any animal, or exposed to an animal suspected of having rabies.
    - Based on your injuries and the situation in which the exposure occurred, you and your doctor can decide whether you should receive treatment to prevent rabies.
    - Even if you aren't sure whether you've been bitten, seek medical attention.

Prevention and control of rabies:

To reduce the risk of coming in contact with rabid animals:

- Vaccinate all cats and dogs against rabies, usually annually. Ask your veterinarian how often your pets should be vaccinated.
- Keep cats and dogs confined. Keep your pets inside and supervise them when outside. This will help keep your pets from coming in contact with wild animals.
- Report stray animals especially those suspected of having rabies to local authorities.
- Don't approach wild animals. Wild animals with rabies may seem unafraid of people. It's not normal for a wild animal to be friendly with people, so stay away from any wild animals or stray dogs that seems unafraid.
- Keep bats out of your home. Seal any cracks and gaps where bats can enter your home.
- If you know you have bats in your home, work with a local expert to find ways to keep bats out.

Session 1.3 Anthrax Disease in Human

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control of anthrax disease.

Time ***1 hour***

Cause of Anthrax Disease:

- The infection is caused by bacteria (Bacillus anthracis) commonly found in the soil.
- Humans can get anthrax through:
  - exposure to infected domestic or wild grazing animals
  - exposure to infected animal products, such as wool or hides
  - inhalation of spores, typically during the processing of contaminated animal products (inhalation anthrax)
  - consumption of undercooked meat from infected animals (gastrointestinal anthrax)
- Anthrax can be transmitted when spores are inhaled, ingested or touched.
- It can spread when:
  - Infected farm animals or their products such as meat and hide are touched.
  - Undercooked meat from animals infected with anthrax are eaten.

Symptoms of Anthrax in people:

- There are four common routes of anthrax infection, each with different signs and symptoms.
- In most cases, symptoms develop within **six days** of exposure to the bacteria. However, it's possible for inhalation anthrax symptoms to take more than six weeks to appear.

Symptoms vary depending on the route of entry of bacterial spores:

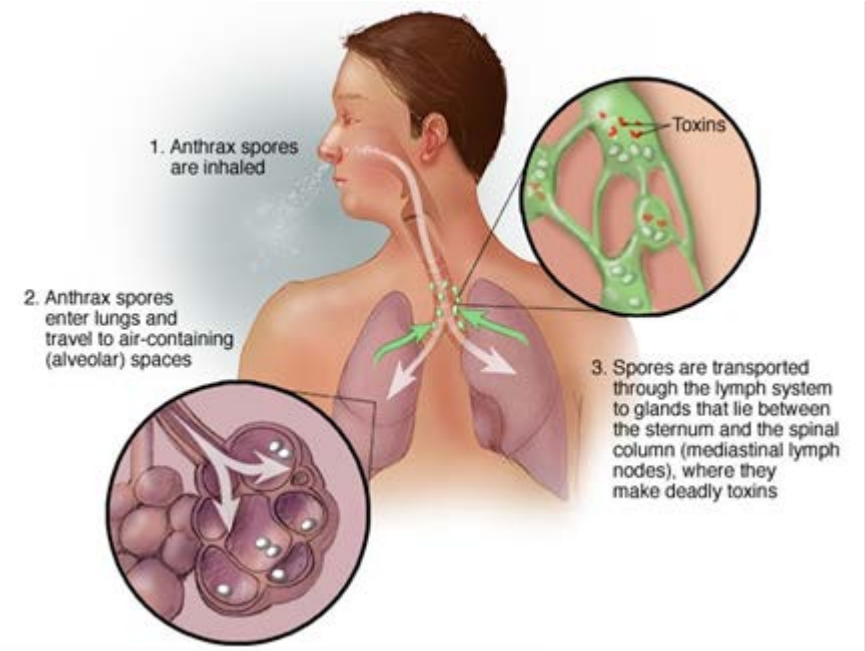
a) Cutaneous anthrax: Infection where bacteria enters the body through a cut or sore on skin:

- Raised, itchy bump which develops into a painless sore with a black center and swollen lymph nodes.
- The cutaneous (skin) form of anthrax starts as a red-brown raised spot that enlarges with considerable redness around it, blistering, and hardening.
- It causes an ugly, dark sore. Humans and animals can ingest anthrax from carcasses of dead animals that have been contaminated with anthrax.

b) Gastrointestinal anthrax: Infection which results due to consumption of undercooked meat of an infected animal:

- Nausea, Vomiting, Abdominal pain, Headache, Loss of appetite, Fever, Severe, bloody diarrhea in the later stages of the disease and Swollen neck.
- Ingestion of anthrax can cause serious, sometimes fatal disease.
- Inhalation (pulmonary) anthrax: Infection develops when the patient breathes in anthrax spores:
- Sore throat, mild fever, fatigue and muscle aches, Chest discomfort, Shortness of breath, Nausea, Coughing up blood and Painful swallowing.

c) Respiratory anthrax: The most deadly form is inhalation anthrax. If the spores of anthrax are inhaled, they migrate to lymph glands in the chest where they proliferate, spread, and produce toxins that often cause death.



Active TB is a stage where immediate medications and care is required, the common symptoms include:

- Persistent cough (which lasts for more than 2 - 3 weeks)
- Cough with blood in sputum
- Fever for more than 2 -3 weeks
- Sudden weight loss
- Night sweats
- Loss of appetite

Treatment and Prevention of Anthrax Disease:

- Anthrax can be treated with antibiotics if it's caught early. The problem is that many people don't seek treatment until it's too late. Without treatment, the chances of death from anthrax increase.
- Treatment for anthrax depends if you've developed symptoms or not.
- If you're exposed to anthrax but you have no symptoms, your doctor will begin preventive treatment.
- If you've been exposed to anthrax and have symptoms, your doctor will treat you with antibiotics.
- Do not open carcass of animal died from Anthrax Carcasses including aborted fetuses should be buried deeply or burned.
- Vaccinate your animals once per year.

Prevention and control of anthrax in livestock

- Regular vaccination on annual basis
- Burn or bury carcasses of animals died of anthrax.

- Mark contaminated site such as through fencing can help herders to keep animals away from such site.
- It is essential that affected animals are isolated for treatment with antibiotics such as intravenous (IV) injection of oxytetracycline.

Session 1.4 Echinococcosis

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control of Echinococcosis

Time ***1 hour***

Cause of Echinococcosis:
Echinococcosis is a parasitic disease caused by tapeworms that occurs in two main forms in humans: Cystic echinococcosis (also known as hydatidosis) and alveolar echinococcosis. Dogs act as definitive hosts for the parasite, and host the mature tapeworm in their intestine and evacuate the parasite eggs in their faeces. They are infected through the consumption of viscera of intermediate hosts that harbor the parasite.

Cystic echinocccosis (CE), likewise called hydatid disease, is brought on by infection with the larval stage of Echinococcus granulosus, a ~ 2-7 millimeter long tapeworm found in dogs (definitive host) and sheep, livestock and goats (intermediate hosts). Although the majority of infections in humans are asymptomatic, CE causes damaging, slowly enlarging cysts in the liver, lungs, and other organs that frequently grow undetected and overlooked for many years.

Alveolar echinococcosis (AE) disease is caused by infection with the larval stage of Echinococcus multilocularis, a ~ 1-4 millimeter long tapeworm discovered in foxes, coyotes, and dogs (conclusive hosts). Little rodents are intermediate hosts for E. multilocularis. Although cases of AE in animals in endemic areas are relatively typical, human cases are rare. AE positions a much greater health hazard to people than CE, causing parasitic tumors that can form in the liver, lungs, brain, and other organs. If left untreated, AE can be deadly. Humans are infected through ingestion of parasite eggs in contaminated food, water or soil, or after direct contact.

The genotype triggering the great majority of cystic echinococcosis infections in people is principally kept in a dog — sheep — dog cycle, yet numerous other domestic animals may likewise be included, consisting of goats, cattle and camels. A number of herbivorous and omnivorous animals act as intermediate hosts of Echinococcus. They end up being infected by consuming the parasite eggs in contaminated food and water, and the parasite then turns into larval stages in the viscera.

Diagnosis of Echinococcosis:

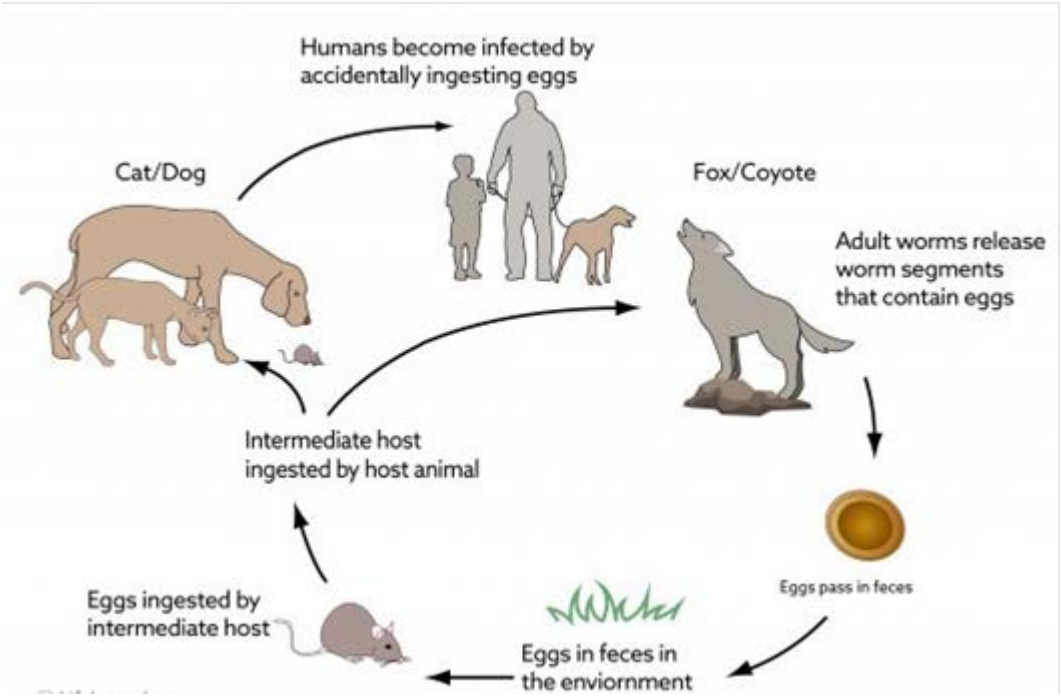
- Ultrasonography imaging is the technique of choice for the diagnosis of both cystic echinococcosis and alveolar echinococcosis in human beings. This strategy is generally complemented or confirmed by computed tomography (CT) and/or magnetic resonance imaging (MRI) scans.
- Cysts can be found by radiography.
- Particular antibodies are discovered by various serological tests and can support the medical diagnosis.
- Biopsies and ultrasound-guided punctures might likewise be performed for differential diagnosis of cysts from tumours and abscesses.

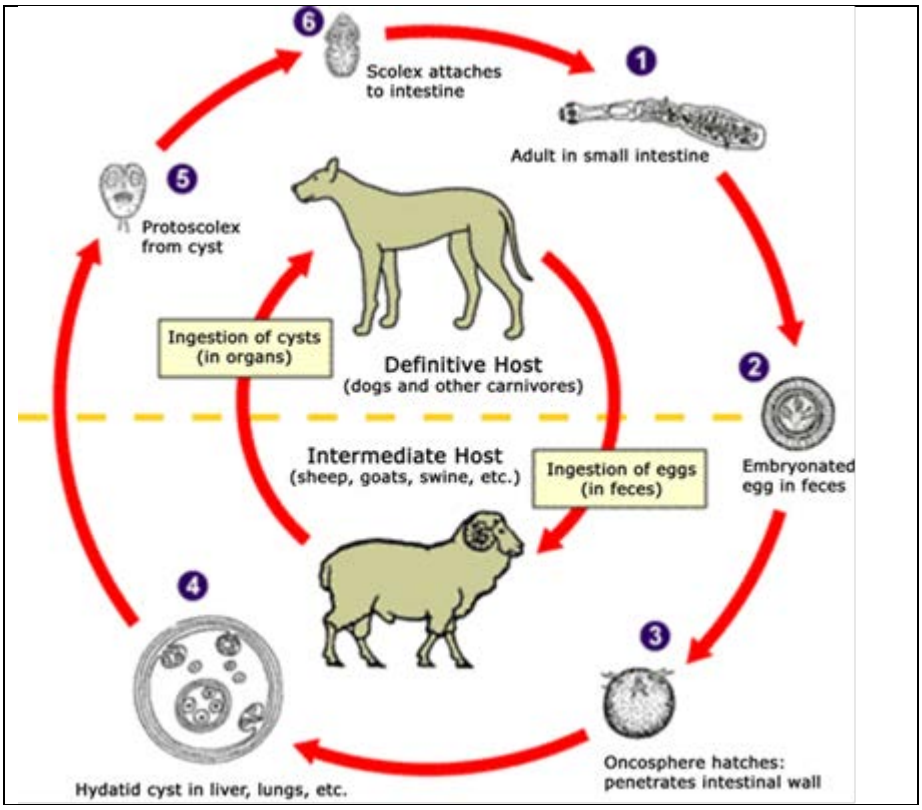
Treatment of Echinococcosis:

- Both cystic echinococcosis and alveolar echinococcosis are typically expensive and complicated to treat, often needing substantial surgery and/or extended drug therapy.
- There are 4 choices for the treatment of cystic echinococcosis:
  - percutaneous treatment of the hydatid cysts with the PAIR (Puncture, Aspiration, Injection, Re-aspiration) technique;
  - Surgery
  - Anti-infective drug treatment
  - “Watch and wait”.
- The option should mainly be based on the ultrasound pictures of the cyst, following a stage-specific technique, as well as on the medical facilities and personnel available.
- For alveolar echinococcosis, early diagnosis and radical (tumour-like) surgery followed by anti-infective prophylaxis with albendazole remain the key elements. If the sore is confined, extreme surgery can be curative.
- Regrettably in many patients the disease is diagnosed at a sophisticated stage. As a result, if palliative surgery is performed without complete and efficient anti-infective treatment, regular regressions will happen.
- Early detection of E. granulosus and E. multilocularis infections, especially in low-resource settings, is still needed in addition to the assessment of clinical treatment alternatives. This could supplement control measures such as the treatment of dogs.

Prevention of Echinococcosis:

- Cystic echinococcosis is a preventable disease as it includes domestic animal types as definitive and intermediate hosts.
- Regular deworming of dogs, enhanced hygiene in the slaughtering of livestock (including the proper damage of infected offal), and public education campaigns have actually been found to lower and prevent transmission and minimize the problem of human disease.


Humans become infected by accidentally ingesting eggs
Cat/Dog
Fox/Coyote
Adult worms release worm segments that contain eggs
Intermediate host ingested by host animal
Eggs pass in feces
Eggs ingested by intermediate host
Eggs in feces in the enviornment


6
Scolex attaches to intestine
1
Adult in small intestine
5
Protoscolex from cyst
Ingestion of cysts (in organs)
Definitive Host
(dogs and other carnivores)
2
Embryonated egg in feces
Intermediate Host
(sheep, goats, swine, etc.)
Ingestion of eggs (in feces)
4
3
Oncosphere hatches: penetrates intestinal wall
Hydatid cyst in liver, lungs, etc.

Session 1.5 Tuberculosis

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control of Tuberculosis

Time ***1 hour***

Cause and Risk Factors of Tuberculosis:

- Zoonotic tuberculosis (TB) is a form of tuberculosis in people caused by Mycobacterium bovis, which belongs to the M. tuberculosis complex.
- It often affects sites other than the lungs (extra-pulmonary, gastro-intestinal), but in many cases is clinically indistinguishable from TB caused by M. tuberculosis.
- Within animal populations, M. bovis is the causative agent of bovine TB.
- M. bovis mainly affects cattle as well as range of wild animal species.
- It results in important economic losses and trade barriers with a major impact on the livelihoods of poor and marginalized communities.
- The most common route of transmission of M. bovis to humans is through food (mainly untreated dairy products or, less commonly, untreated meat products).
- Airborne transmission also poses an occupational risk to people in contact with infected animals or animal products, including farmers, veterinarians, slaughterhouse workers and butchers.

Challenges:

- The laboratory procedures most commonly used to diagnose TB do not differentiate M. tuberculosis from M. bovis. This leads to under diagnosis of zoonotic TB.
- Zoonotic TB poses challenges for patient treatment and recovery.
- M. bovis is naturally resistant to pyrazinamide, one of the four medications used in the standard first-line anti TB treatment regimen.
- As most healthcare providers initiate treatment without drug susceptibility testing, patients with zoonotic TB may receive inadequate treatment.
- Zoonotic TB in humans is often initially extra-pulmonary and may be misdiagnosed, and therefore initiation of treatment can be delayed.

Control and Prevention of Tuberculosis:

- Use separate house for livestock especially cattle.
- The World Health Organization (WHO), the World Organisation for Animal Health (OIE), the Food and Agriculture Organization of the UN (FAO) and the International Union Against Tuberculosis and Lung Disease (The Union) launched the first-ever roadmap on tackling zoonotic TB in October 2017.

- The roadmap is centered on a One Health approach, recognizing the interdependence of human and animal health sectors, to tackle the major health and economic impact of this disease.
- It articulates clear immediate actions that all stakeholders can take to address this issue across different sectors and disciplines, and defines milestones for the short- and medium-term.
- The roadmap calls for concerted action from government agencies, donors, academia, non-governmental organizations and private stakeholders across political, financial and technical levels.
- Ten priorities for action are defined, which will also bring substantial benefits for the control of other zoonotic and foodborne diseases. These include:
  - Develop strategies to improve food safety.
  - Develop capacity of the animal health sector to reduce the prevalence of TB in livestock.
  - Identify key populations and risk pathways for transmission of zoonotic TB.
  - Increase awareness of zoonotic TB, engage key public and private stakeholders and establish effective intersectoral collaboration.
  - Develop and implement policies and guidelines for the prevention, surveillance, diagnosis, and treatment of zoonotic TB, in line with intergovernmental standards where relevant.
  - Identify opportunities for community-tailored interventions that jointly address human and animal health.
  - Develop an investment case to advocate for political commitment and funding to address zoonotic TB across sectors at the global, regional and national levels.

Session 2. COVID-19

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control and prevention of COVID-19

Time ***30 minutes***

Session 2.1 Cause and Symptoms of COVID-19

- COVID-19 is a respiratory condition caused by a coronavirus.
- Some people are infected but don't notice any symptoms.
- Most people will have mild symptoms and get better on their own. But about 1 in 6 will have severe problems, such as trouble breathing.
- The odds of more serious symptoms are higher if you're older or have another health condition like diabetes or heart disease.
- Here's what to look for if you think you might have COVID-19.

- Fever
- Fatigue
- A dry cough
- Loss of appetite
- Body aches
- Shortness of breath
- Mucus or phlegm
- Symptoms usually begin 2 to 14 days after you come into contact with the virus.

- Other symptoms may include: Sore throat, Headache, Chills, sometimes with shaking, Loss of smell or taste, Congestion or runny nose, Nausea or vomiting and Diarrhea

Emergency Symptoms:

Call a doctor or hospital right away if you have one or more of these COVID-19 symptoms:

- Trouble breathing
- Constant pain or pressure in your chest
- Bluish lips or face
- Sudden confusion
- You need medical care as soon as possible.
- Call your doctor's office or hospital before you go in.

Session 2.2 Prevention of COVID-19

- If COVID-19 is spreading in your community, stay safe by taking some simple precautions, such as:
  - Physical distancing
  - Wearing a mask
  - Keeping rooms well ventilated
  - Avoiding crowds
  - Cleaning your hands
  - And coughing into a bent elbow or tissue
  - Check local advice where you live and work.
  - **Do it all!**

Session 3. Animal Diseases Diagnosis, Prevention and Control

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission mechanism and control and prevention of important diseases

Time: ***4 hours***

Session 3.1 The Signs of Health and Disease in Livestock

Objective:	At the end of the training the trainees *understand the signs of ill-health, cause, transmission, diagnose, treatment and prevention methods of locally important diseases.*

Time: ***30 minutes***

Early detection of ill-health condition and understanding the causes and spread of disease is the foundation for disease treatment, prevention and control. It is important to relate the veterinary understanding of the causes of the disease to the local understanding of the causes of the disease, and to discuss the differences.

It is also important to describe microscopic pathogens in simple terms; the term 'germ' (or the local translation of such terms such as very small for virus; small for bacteria; medium for protozoa; and large for parasites) may be sufficient. Trainees should also understand the other causes of disease in livestock such as malnutrition, foreign body injected with feed, physical injury and poor management. Problems such as poor breeding and abortion should be mentioned if livestock owners perceive these as problems in their area.

During this part of the training it is necessary to describe locally important specific diseases in detail, and adequate training aid or live animals should be used. There is also a need to reinforce existing knowledge on signs of good health and of illness, and to enable the trainees to differentiate between the two states in different species of livestock. The trainees should be able to list the features of a healthy animal and those of an unhealthy animal.

Practical examination of sick and healthy animals by all trainees is a crucial component of this training. Where possible, the training can be re-enforced by post-mortem examination of healthy and diseased animals, with emphasis on the location, size and appearance of healthy and diseased organs and systems.

Session 3.2 Causes, Transmission and Prevention of Disease

Objective:	At the end of the training the trainees should be able to describe common causes of ill health in livestock, modes of disease transmission and simple disease prevention measures.

Time: ***30 minutes***

Trainees should understand the causes of disease in livestock such as malnutrition, physical injury, infection with disease agents and poor management. Various modes of disease transmission can be presented, including direct contact between sick and health animals, contact with contaminated items and disease spread via biting flies or snails.

Understanding of the causes and spread of disease can lead to training on the principles of disease prevention and control. It is important to relate the medical understanding of the causes of the disease to the local understanding of the causes of the disease, and to discuss the differences.

Session 3.3 Disease Diagnosis

Objective:	At the end of the training the trainees should be able to identify sick animals.

Time: ***30 minutes***

The training approach is to assess and reinforce the trainees' existing knowledge of diseases in different livestock species. This knowledge includes clinical signs, mode of transmission and other factors.

As the relative importance of different diseases varies from place to place, and since different CIGs may keep different livestock species, this part of the training should vary from place to place. The aim is to ensure that all the local problems that can be practically addressed by CIGs are emphasized and taught. CIGs should be able to assess cases and report to a veterinarian or AHA.

Note that diseases can be categorized according to either the method of prevention/treatment the main presenting clinical signs observed in the areas concerned, or the method of prevention/treatment.

Session 3.4 Anthrax Disease

Objective:	At the end of the training the trainees should be able to described cause, transmission, control and prevention of anthrax disease in livestock.

Time: ***30 minutes***

Cause and Transmission of Anthrax:

Anthrax is an infectious disease caused by spore-forming bacteria (Bacillus anthracis). It occurs naturally in all animals. Hoofed animals, such as cattle, goats, and sheep, are the main animals affected by this disease. Usually affects individual animals but it could be seen in-group of animals exposed to spores while grazing from contaminated site. They usually get the disease by swallowing anthrax spores while grazing on pasture contaminated with anthrax spores. Inhaling (breathing in) the spores, which are odorless, colorless, and tasteless, may also cause infection in animals and people.

Signs of Anthrax Disease in Animals:

Signs of the illness usually appear 3 to 7 days after the spores are swallowed or inhaled. Once signs begin in animals, they usually die within two days. Infected animals may stagger, have difficulty breathing, tremble, and finally collapse and die within a few hours. Sometimes animals may have a fever and a period of excitement followed by staggering, depression, unconsciousness, difficulty breathing, seizures, and death and bloating advances quickly. Dark blood may ooze from the mouth, nose, and anus. No rigor mortis (Legs remain flexible).

Diagnosis and Treatment of Anthrax Disease in Animals:

A diagnosis is made by finding the anthrax bacteria or antibodies to anthrax in the blood of infected animals through laboratory tests.

It is usually hopeless to treat animals that are sick. Sometimes, if the disease is detected soon after infection, antibiotics, along with nursing care, may help. Oxy tetracycline injection is very useful but in a general term treatment is believed to be less successful

Control and prevention of Anthrax:

- Do not open cases died of anthrax at all.
- Dispose any discharges and the carcass properly: burn, bury deep and cover it tightly to avoid wild carnivores, vultures, dogs, etc, exposure.

- Vaccinate animals every 12 months.
- Anthrax vaccine may cause fever that pregnant animals may abort especially at the first three-four months.
- Avoid contaminated fields for grazing.

Session 3.5 Black leg Disease

Objective:	At the end of the training the trainees should be able to described cause, transmission, control and prevention of black leg disease.

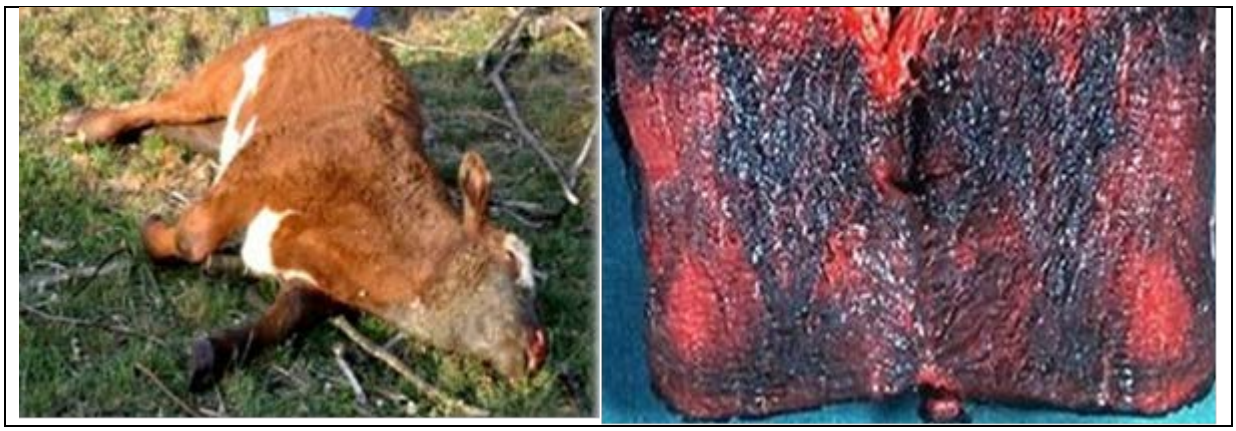
Time: ***30 minutes***

Cause, Symptoms, Transmission, Treatment and Prevention of Black leg disease:

Blackleg is a highly fatal disease of young cattle caused by the spore forming, rod shaped, gas producing bacteria Clostridium chauvoei. The spores of the organism can live in the soil for many years. The bacteria enters the calf by ingestion and then gains entrance to the body through small punctures in the mucous membrane of the digestive tract. Cattle that are on a high plane of nutrition, rapidly gaining weight and between 6 months and 2 years of age are most susceptible to the disease. The disease is not transmitted directly from sick animals to healthy animals by mere contact.

The first sign observed is usually lameness, loss of appetite, rapid breathing and the animal is usually depressed and has a high fever. Characteristic swellings develop in the hip, shoulder, chest, back, neck or elsewhere. First the swelling is small, hot and painful. As the disease progresses, the swelling enlarges and becomes spongy and gaseous. If you press the swelling, gas can be felt under the skin. The animal usually dies in 12 to 48 hours. In most cases the animal is found dead without being previously observed sick. The speed with which blackleg kills usually makes individual treatment useless.

Blackleg is almost entirely preventable by vaccination. The most commonly used clostridial vaccination in Ethiopia is black leg vaccine which protects against for 6-months. Typically, treatment is ineffective against blackleg, and the mortality rate of the disease is relatively high. In some cases, if the disease is detected early enough, penicillin can be effective in saving an animal's life. A cow that survives blackleg, however, usually suffers from a permanent deformity or lameness.



Session 3.6 **Contagious Bovine Pleuropneumonia (CBPP)**

Objective:	At the end of the training the trainees should be able to described cause, transmission, control and prevention of CBPP disease.

Time: ***30 minutes***

Etiology and Transmission of CBPP:

The disease is caused by a bacterium called *Mycoplasma mycoides* var. *mycoides* which is difficult to see even with a light microscope but growth of the organism can be seen when infectious material is cultured in the laboratory. CBPP is an infectious disease of lungs in cattle. CBPP is spread by inhalation of infected droplets expelled by animals with the disease.

CBPP is invariably introduced into a herd by contact with an infected animal; transmission occurs from direct, close, repeated contacts between diseased and healthy animals in shared night accommodation or at water holes, dip tanks, markets, common grazing and gathering places for mass vaccination campaigns. Because the organism can only survive a few hours outside the host, direct contact is needed for infection to be passed on. This is why infection is spread faster in places where cattle are gathered together, e.g. in housing, in transit, etc.

The causative agent is present in liquid droplets in the breath and in urine and, although the CBPP organisms are killed rapidly in hot, dry environments, air-borne transmission appears possible over distances up to 200 meters. Transmission is favored by close crowding of cattle and outbreaks are more common and extensive when cattle are housed or have been transported by truck and trekked on foot in groups.

Chronically infected and symptomless animals play an important role in the persistence and spread of the disease. In this context pastoral herds are especially significant since they may contain many chronically infected animals. Herdsmen fleeing from a focus of disease with apparently healthy animals have been known to spread disease widely.

CBPP is one of the most serious diseases of cattle in the country which can cause great economical loses if it is not controlled. Suspected cases should be reported to the veterinary authorities to prevent it from spreading. Vaccination is an effective option for controlling the disease. However, it needs to be combined with other measures to keep the disease under a total control.

Clinical Signs of CBPP:

The affected animals usually have a fever, breathe rapidly and are generally depressed. When the lungs become more infected, these animals develop a cough and their breathing gets increasingly difficult and painful. The sick animals are usually reluctant to move. In order to relieve the pain of breathing, these animals stand with their head extended and elbows turned out. Recovered animals remain carrier of infection.

Typically, when first introduced into a herd CBPP is severe and mortality relatively high. A small proportion of cattle may die rapidly without showing any signs other than fever. It may be possible to link the onset of disease to previous contact with other cattle three to six weeks earlier but this is not always the case as the incubation period may appear to be as long as six months.

Clinical signs may become apparent only several months after the contact. Thus, the disease can become established in a herd before it is noticed and tracing back to the origin can be difficult. This is particularly so where routine vaccination has been practiced with long intervals between campaigns and where antibiotics have been used to treat clinical cases. Both reduce the incidence of clinical disease, making its recognition more difficult, but do not prevent infection occurring in the herd. After some time, the disease in the herd becomes chronic, the clinical signs become less severe and the mortality rate falls. However, losses do continue.

Not all the animals are affected in the same way and often the disease has a chronic course from its onset.

The **hyper-acute form**, involving up to 10 per cent of infected animals, may be observed at the onset of an outbreak, death is sudden and is often not accompanied by any other signs. Clinical diagnosis is difficult.

The acute form is observed in approximately 20 per cent of the diseased animals. The course is 5 to 7 days. The earliest signs are a sudden onset of fever to 40° C or more and, in milking cows, a drop in milk yield. Sick cattle tend to isolate themselves from the herd and stop eating.

A typical respiratory disease develops; breathing is labored and obviously painful. Abdominal breathing with a respiratory rate of 50 to 55 breaths/ min may be seen and cattle may 'grunt' when breathing out. Some animals develop a shallow, dry and painful cough particularly noticeable on exercise. Application of pressure between the ribs is painful and resented by affected cattle which sometimes react violently. On percussion, the ventral part of the chest sounds dull owing to the presence of fluid in the chest cavity.

Acutely affected cattle stand with head and neck extended and forelegs spread apart, dilated nostrils and with mouth open panting for air. There may be nasal discharge, sometimes streaked with blood, and frothy saliva accumulates around the mouth. Some animals develop swellings of the throat and dewlap. Pregnant cows and heifers may abort and diarrhea has been recorded. If the animal survives, the disease turns chronic; clinical recovery is only apparent.

The **sub-acute form** occurs most frequently in about 40–50 per cent of the animals affected. The symptoms resemble those of the acute form but are less severe; fever is intermittent. This form usually becomes chronic.

The **chronic form** is a natural evolution of both acute and sub-acute ones but in some animals it can develop directly. The clinical signs regress but cattle can still have intermittent fever together with loss of both appetite and weight. **Calves**, in the first six months of life, more often show lameness from swollen, hot, painful limb joints.

The mortality rate is variable, rarely exceeding 50 per cent, and depends on a range of factors such as age, breed, nutrition, presence of other infections or infestations and the type of management. Many affected cattle can appear to recover fully, yet the lesions in the lungs take a long time to heal fully and inside them the causative agent can survive for as long as two years. Up to 25 per cent of affected cattle can become chronic carriers of infection. They are often referred to as 'lungers' and are believed to play a role in initiating new outbreaks when they are moved into susceptible herds.

In summary, look for one or more animals with fast, difficult or noisy breathing, discharges from the nose or mouth, coughing, especially after exercise and any chronic

(persistent) mild cough in cattle otherwise appearing normal or losing weight should be a reason to suspect CBPP.

Treatment of CBPP:
In non-endemic areas, treatment is rarely used as it may convert clinical cases to clinically normal carriers. The emphasis is usually on prevention and eradication of the disease. However, in endemic areas, treatment may be the only option. Sulfadimidine and tylosin are the drugs in use. Tylosin has to be injected intramuscularly every twelve hours for three days.

Prevention and control of CBPP:
Ideally, CBPP control is achieved by eliminating the whole cattle herd population wherever the disease is detected i.e. stamping-out. However, this is not realistic and quarantine coupled with vaccination is the most frequently used CBPP control measure. Yet, to be effective vaccination must target 100 per cent of cattle within an epidemiologically and geographically definable area.

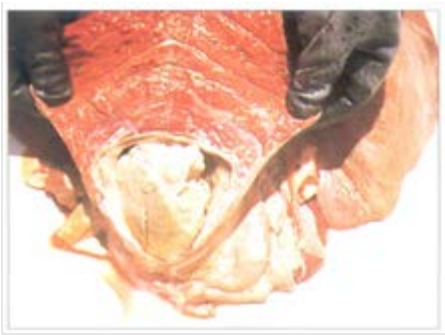
Vaccination must be repeated, initially at short intervals and thereafter annually over several years, i.e. not less than 3 to 5 years. Such vaccination must be maintained until evidence of CBPP eradication is demonstrated by structured surveillance. Live attenuated vaccines are widely used in Africa and only those with PANVAC certification should be used. Vaccine quality control is, thus, an essential element of CBPP control programs.

In endemic areas, vaccination may be an effective option to control the disease. Several vaccines have been developed, and the effective ones are those based on live mycoplasma organisms. However, it is difficult to use in practice because mycoplasma strains that are mild enough to be used safely are usually poor to stimulate immunity, whereas those that are good immunogens may cause severe reactions.

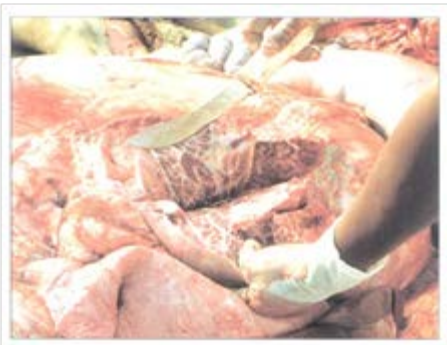
None of the existing vaccines can confer live-long immunity, hence, they have to be repeated each year. Successful control of the disease should combine vaccination with other measures. In ideal situations, all the clinical cases and carriers should be slaughtered. However, this is too expensive and not feasible to implement in reality. Steps must be taken to minimize the spread of the disease. When moving cattle into a new area, tests should be carried out to detect any infected or carrier animals which should be removed.

Post-mortem Examination, look for:

- yellow fluid in the chest cavity
- lungs covered with yellowish material
- lungs adhering to the chest wall, which do not collapse and are solid or marbled
- sequestra in the lungs of chronic cases



Sequestrum in the lung



Marbled lung

Session 3.7 Contagious Caprine Pleuropneumonia (CCPP)

Objective:	At the end of the training the trainees should be able to described cause, transmission and control and prevention of CCPP disease.

Time: ***30 minutes***

Etiology: CCPP is caused by Mycoplasma capricolum subsp. capripneumoniae (formerly Mycoplasma biotype F-38), a member of the family Mycoplasmataceae. OIE limits the cause of CCPP to only M. capricolum subsp. capripneumoniae.

CCPP is one of the most severe diseases of goats. This disease, which affects the respiratory tract, is extremely contagious and frequently fatal; in some naive flocks, the morbidity and mortality rates may reach 100%. CCPP causes major economic losses in Ethiopia and other countries where it is endemic. Definitive diagnosis can be difficult, as the causative agent is one of the most fastidious mycoplasmas and can be missed during routine bacteriological analysis.

Transmission of CCPP:

CCPP is a disease affecting goats caused by Mycoplasma capricolum subsp. capripneumoniae (Mccp). Contagious caprine pleuropneumonia is highly contagious. The incubation period is commonly 6 to 10 days, but is reported to range from 2 days to 4 weeks. This disease is transmitted during close contact, by the inhalation of respiratory droplets. Chronic carriers may exist, but this remains unproven. Some outbreaks have occurred in endemic areas when apparently healthy goats were introduced into flocks. Mycoplasma spp. are generally short-lived, fragile organisms in the environment.

Clinical Signs of CCPP:

It is manifested by anorexia, fever and respiratory signs such as dyspnea, cough and nasal discharges. CCPP is strictly a respiratory disease. Per-acute, acute and chronic

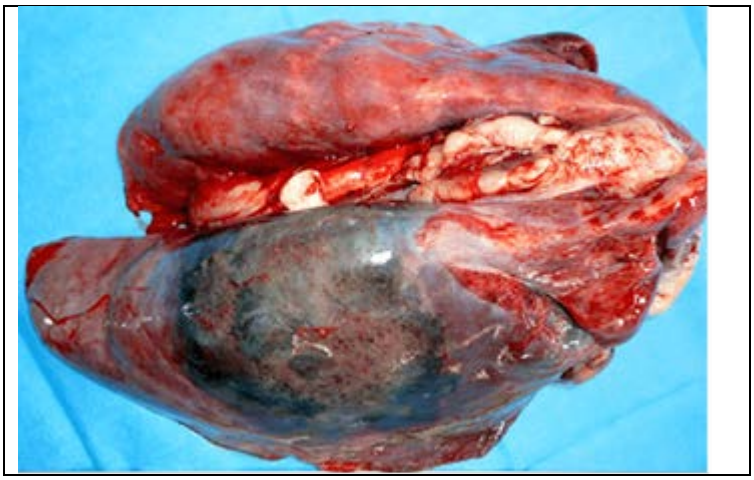
forms may be seen in endemic areas. Per-acutely affected goats can die within 1 to 3 days with minimal clinical signs. In acute disease, the initial signs are a very high fever (41-43°C), lethargy and anorexia, followed by coughing and labored respiration. The cough is frequent, violent and productive.

In the final stages of disease, the goat may not be able to move, and stands with its front legs wide apart, and its neck stiff and extended. Saliva can drip continuously from the mouth, and the animal may grunt or bleat in pain. Frothy nasal discharge and stringy saliva may be seen terminally. Pregnant goats can abort. Acutely affected goats generally die within 7 to 10 days. Sub-acute or chronic cases tend to be milder, with coughing mainly following activity. Chronic CCPP is characterized by a chronic cough, nasal discharge and debilitation.

Post Mortem Lesions of CCPP:

The lesions of CCPP are limited to the respiratory system. Acute disease is characterized by unilateral or bilateral pneumonia and serofibrinous pleuritis with straw-colored fluid in the thorax. On cut surface, the lung is granular with copious straw-colored exudate. Pea-sized, yellow nodules may be found in the lungs; these nodules are surrounded by areas of congestion.

Varying degrees of lung consolidation or necrosis can be seen, and the regional (bronchial) lymph nodes are enlarged. Some long-term survivors have chronic pleuropneumonia or chronic pleuritis, with encapsulation of acute lesions and numerous adhesions to the chest wall. The interlobular septa are not usually thickened in domesticated goats.



Treatment of CCPP:
Some antibiotics, such as long acting 20% Oxy Tetracycline can be effective if given early. Complete elimination of mycoplasmas is reported to be rare, and treated animals may be potential carriers. The degree of risk from treated animals spreading M. capripneumoniae is still uncertain.

Control and Prevention of CCPP:
A quick response is vital for containing outbreaks in regions free of CCPP. Veterinarians who encounter or suspect this disease should follow the national guidelines for disease reporting. CCPP is most likely to spread through infected animals, due to the poor survival of mycoplasmal organisms in the environment. It is uncertain whether long-term subclinical carriers exist. Outbreaks can be eradicated with quarantines, movement controls, slaughter of infected and exposed animals, and cleaning and disinfection of the premises.

In endemic areas, care should be taken when introducing new animals into the flock. Flock on-site quarantine may be helpful in controlling the spread of disease. Vaccines help prevent disease in some countries. In addition, antibiotic treatment and reductions in animal density (to decrease contact between animals) were commonly employed.

Session 3.8 Peste-des-Petits Ruminant (PPR) Disease

Objective:	At the end of the training the trainees should be able to described cause, transmission and control and prevention of PPR disease.

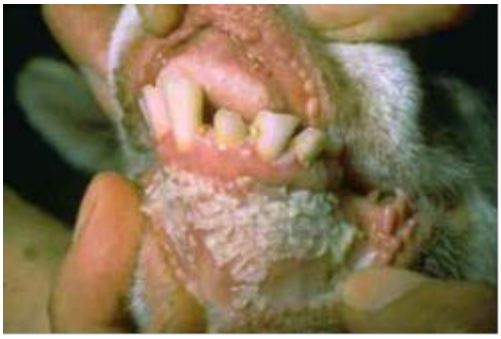
Time: ***30 minutes***

Cause and Transmission of PPR Disease:

- The Peste-des-Petits Ruminants disease is caused by Moribillivirus of Paramyxoviridae family.
- PPR disease is an acute highly contagious viral disease of small ruminants characterized by fever, loss of appetite, stomatitis, gastroenteritis and pneumonitis.
- Natural transmission occurs primarily through direct contact with infected sheep and goat.
- Transmission may take place through contaminated feed, water, beddings and other appliances.
- Secretions and excretions are rich source of virus and spread of the disease take place through their contamination. Faeces are the main spreading agent and through it the disease may occur in epidemic proportion.
- The disease may spread in a flock through introduction of newly purchased sick animal from market.
- There is no carrier state in animals; the spread of the disease is possible through animals with subclinical infection.
- Ingestion of infected material is the main way of transmission but it may also take place through inhalation and contact with ocular secretions.

Symptoms of PPR Disease:

- High rise of temperature (39 to 40 ℃).
- The animal will show dull coat, dry muzzle and inappetance.
- There will be profuse serous nasal discharge accompanied by sneezing and coughing.
- The discharge may be crust like, hard and matt the nasal and ocular surroundings.
- Oral necrotic lesions noticed in lips, buccal mucosae, gums, dental palate & tongue, with malodour.
- Congestion of conjunctival mucous membranes and matting of eye lids.
- **Signs of pneumonia** and animal may die due to respiratory distress.
- Diarrheic faces may contain mucus and blood.
- Pregnant goat may abort.
- Most of the animals recover and death may occur in few of them.



Control and prevention management of PPR:

- Regular and proper vaccination of animals.
- Strict sanitation and hygienic measures are to be adopted in a flock.
- Restriction should be made for introduction of new animals in a flock especially in areas where the disease is prevalent.
- Sick animals bought from market should not be introduced without observation for a definite period.
- Quarantine measures should be strictly attended in imported sheep and goat before introduction.

Session 4: Poultry Diseases

Objective:	At the end of the training the trainees should be able to describe clinical signs, transmission, control and prevention of important poultry diseases.

Time: 2 hours

Session 4.1: Newcastle Disease

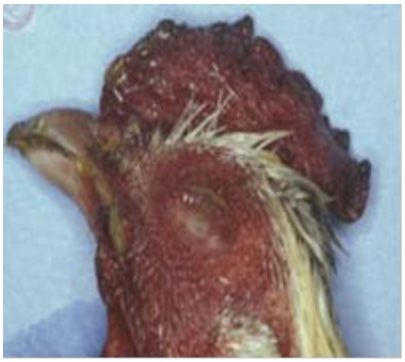
Cause and Clinical Signs of Newcastle Disease:
Newcastle disease (ND) is caused by a paramyxovirus which mainly affects poultry. Young Chickens are the most susceptible host. The incubation period varies with the strain of virus, and is generally 4 to 5 days (range 2 to 15 days). Onset is rapid, and signs appear throughout the flock within 2–12 days (average 5) after aerosol exposure.

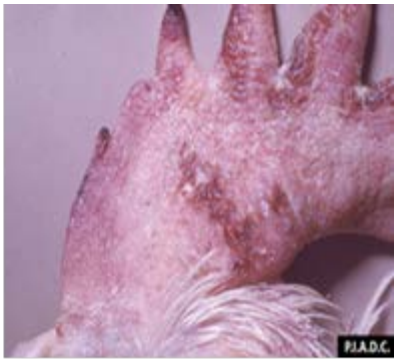
Observed signs depend on whether the infecting virus has a predilection for respiratory, digestive, or nervous systems. Respiratory signs of gasping, coughing, sneezing, and rales. Nervous signs of tremors, paralyzed wings and legs, twisted necks, circling, clonic spasms, and complete paralysis may accompany, but usually follow, the respiratory signs in neurotropic velogenic disease. Nervous signs with diarrhea are typical in pigeons, and nervous signs are frequently seen in cormorants and exotic bird species.

Clinical manifestations vary from high morbidity and mortality to asymptomatic infections. Severity of infection depends on virus virulence and age, immune status, and susceptibility of the host species.

The chicken fluffs its feathers and appears to ‘have its coat dragging on the ground’:

- Lethargy and inappetance.
- Respiratory signs such as mild rales and snick can be detected by careful observation.
- Severe respiratory distress and gasping.
- Swelling of the head and neck.
- The comb is markedly edematous and contains multiple foci of hemorrhage.
- Greenish diarrhea.
- Marked decrease in egg production. Sometimes deformed eggs may be produced.
- Nervous signs of tremor, torticollis, convulsions and paralysis of wings and legs will not be seen until the disease is advanced.





Post-mortem findings are characteristic but not definitive. ND can be suspected if the following lesions are encountered, particularly in combination (and when the flock history is also consistent with an ND outbreak):

- congestion and mucous exudate in the trachea;
- congestion of the lungs (heavier than normal; lungs sink in water/formalin);
- haemorrhages of the mucosa of the pro-ventriculus;
- haemorrhagic and necrotic ulceration of lymphoid patches of the intestine, caecal tonsils and bursa of Fabricius;
- Congested ovarian follicles in chickens in lay.

Transmission, Prevention and Control of Newcastle Disease

Infected birds shed virus in exhaled air, respiratory discharges, and feces. Virus is shed during incubation, during the clinical stage, and for a varying but limited period during convalescence. Virus may also be present in eggs laid during clinical disease and in all parts of the carcass during acute vNDV infections. Chickens are readily infected by aerosols and by ingesting contaminated water or food. Infected chickens and other domestic and wild birds may be sources of NDV.

Movement of infected birds and transfer of virus, especially in infective feces, by the movement of people and contaminated equipment or litter are the main methods of virus spread between poultry flocks.

Vaccination is the only effective way of controlling ND. However, vaccines currently in use are mainly of benefit to commercial poultry producers whose chickens are kept in large, single-age, confined flocks. Manufacturers produce heat-labile ND vaccines in multi-dose vials, often containing 1,000 or 2,500 doses, which must be kept cold (within 19 a ‘cold chain’) from manufacture until administration to the chickens. In contrast, village chickens are raised in small, multi-age, free-range flocks and large multi-dose vials of vaccine are inappropriate.

Session 4.2: Salmonellosis (Pullorum, Bacillary white diarrhea)

Etiology:

Diarrhea in poultry is one of the commonly noticed sickness or disease in any poultry farm globally. They are also common to backyard birds rearing in rural areas. This problem is very common & pose a challenge to the farmer or poultry keeper. Most of the mortality among backyard poultry takes place due to this problem. In biological term it is nothing but Pullorum Disease or Bacillary White Diarrhea, It is also called Salmonellosis Pullorum disease (PD), also referred to as bacillary white diarrhea, is an acute septicemic disease affecting primarily chickens and turkeys. It is caused by the Salmonella enterica subsp. enterica Gallinarum-Pullorum bacterium. It is one of several different diseases that are collectively referred to as Salmonellosis. PD rarely occurs in commercial poultry anymore, however it is quite common in backyard flocks.

Disease caused by one of the two poultry-adapted strains of Salmonella bacteria, Salmonella Pullorum, this usually only causes mortality in birds up to 3 weeks of age. Occasionally it can cause losses in adult birds, usually brown-shell egg layers. It affects chickens most commonly.

Morbidity is 10-80%; mortality is increased in stressed or immune-compromised flocks and may be up to 100%. The route of infection is oral or via the navel/yolk. Transmission may be transovarian or horizontal mainly in young birds and may sometimes be associated with cannibalism. The bacterium is fairly resistant to normal climate, surviving months but is susceptible to normal disinfectants.

Clinical signs:

- In appetence
- Depression
- Ruffled feathers
- Closed eyes
- Loud chirping
- White diarrhea
- Vent pasting
- Gasping
- Lameness
- Per-acute infection with sudden death
- Acute infection in first few days:
    - Weakness

- Somnolence
- Anorexia
- Poor growth
- Pasting of vent with chalky white excreta
- Death

- In older birds:
  - Lethargy
  - Huddling under brooders
  - Wing droop
  - Dyspnoea.
  - Growth retardation and poor feathering of survivors.

Post-mortem findings:

- Gross lesions may be seen in chronic disease, but are usually absent in per-acute disease.
- When present the following may be seen:
  - Enlargement and congestion of liver, spleen and kidneys,
  - Yolk sac retention, with yolk appearing creamy or caseous,
  - Lung and heart may have white nodules, pericardium may be thickened, with yellow or fibrinous exudate,
  - Gastro-intestinal tract – may have white nodules on the gizzard, caeca, large intestinal wall.
  - Caseous cores may be seen in the caeca.
  - Joints may be swollen with yellow viscous fluid.
  - Grey nodules in lungs, liver, gizzard wall and heart.
  - Intestinal or caecal inflammation.
  - Splenomegaly.
  - Urate crystals in ureters.

Diagnosis:

- Isolation and identification.
- In clinical cases direct plating on Brilliant Green, McConkey and non-selective agar is advisable.
- Enrichment procedures usually rely on selenite broth followed by plating on selective media.

Differential diagnosis:

- Fowl typhoid,

- Fowl cholera,
- Erysipelas

Transmission: From infected birds, their faeces and their eggs. Ingestion of contaminated food, water or bedding, and contact transmission; also mechanical spread by humans, wild birds, mammals, flies, and on trucks, feed sacks. May occur in newly-hatched birds due to trans-ovarial transmission.

Risk of introduction: Pullorum could be introduced by importation of live infected chicken, hatching eggs. The bacteria can also be found in poultry meat but contamination of poultry flocks through this route is at low risk.

Control / vaccines: Live and inactivated vaccines are available for fowl typhoid in some countries. If introduced control should focus on eradication of the disease through isolation and destruction of contaminated flocks, proper disposal of carcasses and disinfection of fomites.

Treatment:

- Sulphadiazine, Sulphamerazine, sulphapyrazine, Sulphamethazine are the most effective in chicken (not in turkey poults).
- Furazolidone is effective.
- Also chloramphenicol, colistin and apromycin are effective. No vaccination practiced and all positive birds may be disposed off by slaughter.

Prevention:

- Eradication from breeder flocks.
- As with other salmonellae, recovered birds are resistant to the effects of infection but may remain carriers.
- Vaccines are not normally used as they interfere with serological testing and elimination of carriers.
- Prevention focuses on a clean and comfortable environment:
- Check feed for mold spores
- Provide fresh, clean water
- Keep the pen dry
- Check for vent prolapse – blowouts can result in diarrhea
- Add two tablespoons of vinegar to a gallon of water for drinking
- Try a probiotic such as yogurt
- Properly dispose of any dead animal carcasses, do not allow birds contact with dead or decaying animals.

There are a number of simple steps you can take:

- Feed your birds a commercial, good quality pellet.
- Ensure feed is fresh, dry and in date and suitable for species and age of bird.
- Supply fresh drinking water in clean drinkers.
- Try to get rid of puddles in the range as these often contain lots of harmful bacteria and for some reason birds like to drink from them rather than their drinkers
- Regularly clean and disinfect your poultry coop using a detergent followed by an approved disinfectant.
- Always worm your birds with –Albendazole/fenbendazole etc at least every three to four months.
- Give —Probiotics powder— every four to six weeks to top up the good bacteria in your birds gut.