



Infectious Diseases of the Ruminant Gastrointestinal System

**DISEASES THAT HAVE THE POTENTIAL TO
CAUSE LOSS OF PRODUCTIVITY**





Wooden Tongue

- **Etiology**

- *Actinobacillus lignieresii* is a gram negative bacteria that is the causative agent of wooden tongue

- **Clinical signs**

- Usually affects the soft tissue of the mouth
- When the tongue is affected, the animal may not be able to prehend food normally, excessive salivation, and sometimes a large tongue that protrudes from the mouth
- Atypical lesions can also be seen and are usually located in the lymph nodes of the head





Wooden Tongue

- **Diagnosis**

- Diagnosis requires biopsy and culture of the lesion
- Pus from the lesion will have sulfur granules and can appear like club colonies

- **Pathogenesis**

- *Actinobacillus lingieresii* is a normal inhabitant of the mouth of ruminants
- When the organism invades the soft tissue through a lesion, a granulomatous abscess
- The classic site of infection is the tongue causing a diffuse nodular swelling
- The disease has world wide distribution

- **Treatment**

- Treatment is usually successful
- Sodium iodide – 70 mg/kg given IV as a 10 – 20 percent solution
 - Given once and then repeated in seven to ten days





Lumpy Jaw

- **Etiology**

- Lumpy jaw is caused by *Actinomyces bovis*, a gram positive bacteria that is a normal inhabitant of the ruminant mouth
- Mostly occurs in cattle, but occasionally be found in sheep and goats

- **Clinical signs**

- Hard, immovable, painless mass on the mandible
- Initially it does not drain but may develop a fistulous tracts later in the course of diseases
- Teeth may become involved and is manifested by difficulty chewing





Lumpy Jaw

- **Differential Diagnosis**

- Differentials include tooth root abscesses, fracture, tumors, and osteomyelitis

- **Pathogenesis**

- *Actinomyces bovis* enters the bone through mucous membrane punctures or through diseased teeth

- **Treatment**

- Treatment usually results in arrest of the lesion but the mass does not significantly regress
- Sodium iodide – 70 mg/kg intravenously in a 10 to 20 percent solution given every seven days
- Isoniazid – 10 mg/kg/day given orally for one month
- Penicillin 10,000 units/kg intramuscularly twice daily for 7 to 14 days



Bluetongue

- **Etiology**

- Bluetongue is an arthropod borne noncontagious disease of ruminants caused by bluetongue virus

- **Clinical signs**

- Clinical disease is limited to sheep, cattle can become infected but do not show clinical signs
- Initial signs include:
 - **High fever, up to 41 degrees Celsius or higher**
 - **Edema of the face, lips, muzzle, and ears**
 - **Excessive salivation**
 - **Hyperemia of the oral mucosa**
 - **Profuse serous nasal discharge that turns mucopurulent after a few days, leaving crusts around the nose and muzzle**
 - **Oral erosions and ulcers are prominent on the dental pad**
 - **Pulmonary edema may develop and present as pneumonia**
 - **Diarrhea may develop later in the course of disease**



Bluetongue

- **Differential Diagnosis**
 - Sheep
 - Foot and mouth disease, PPR, and sheeppox
 - Cattle
 - Bovine viral diarrhea, malignant catarrhal fever, rinderpest





Bluetongue

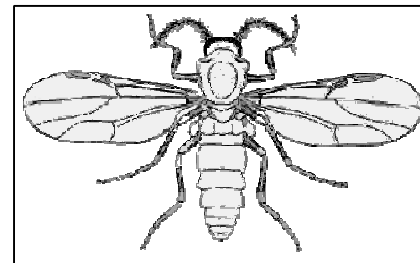
- **Diagnosis**
 - Samples to collect
 - **Blood can be used to detect antibodies against the bluetongue virus or for virus isolation**
 - **Virus isolation requires blood to be taken from tubes containing EDTA, heparin, or sodium citrate**
 - **Serology requires a red top tube which contains no anticoagulant so that the serum can be separated**
 - Serology
 - **Serum samples can be used to identify antibodies in the blood, demonstrating previous exposure**
 - Agar immunodiffusion assays and ELISAs are use to detect antibodies
 - Virus detection
 - **Animal must be viremic to detect virus**
 - **Virus isolation can be done during active infection**
 - **Polymerase chain reaction assays can also be used to detect infection**





Bluetongue

- **Pathogenesis**
 - Bluetongue virus infects vascular endothelium causing vasculitis
 - Vasculitis results in edema and necrosis of the epithelial and mucosal surfaces
- **Epidemiology**
 - Spread by culicoides
 - **Most prevalent in summer and fall when the vector is present**
 - Bluetongue has been detected world wide and been detected in India
- **Treatment**
 - Provide soft feed and ensure adequate water intake
 - Anti-inflammatory medication may provide relief from pain and fever
 - Broad spectrum antibiotics can help prevent secondary infections





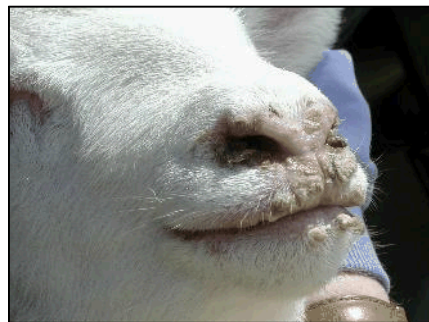
Contagious Ecthyma

- **Etiology**

- Also known as sore mouth, orf, contagious pustular dermatitis, scabby mouth
- Caused by a parapox virus that is contagious to humans – zoonotic pathogen

- **Clinical signs**

- Affects sheep and goats
- Crusty, proliferative lesions of the mucocutaneous junctions of the mouth and nose
- Mostly occurs in young animals but naïve older animals may also be affected
 - In older animals lesions may also be found on the coronary band, on the tongue, interdigitally, on the conjunctiva of the eye, on the external genitalia, or on the udder and teats
 - Ewes and does may develop mastitis as a secondary complication





Contagious Ecthyma

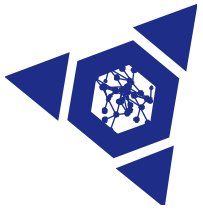
- **Differential diagnosis**
 - Sheep and goat pox – tend to have more systemic signs such as fever, rhinitis and conjunctivitis
 - Bluetongue – seasonal
- **Diagnosis**
 - Diagnosis is based on clinical presentation
- **Epidemiology**
 - World wide distribution, occurs in India
 - Occurs in sheep, goats, and humans
 - Virus is very stable in the environment and can persist for a year



Contagious Ecthyma

- **Pathophysiology**
 - Six stages are characterized
 - **Maculopapular stage:** An elevated red spot,
 - **Target stage:** A halo of dilated vessels and has the appearance of a target
 - **Acute state:** Lesion progresses to a red weeping nodule
 - **Regenerative stage:** Lesion becomes dry with small black spots
 - **Papillomatous stage:** Surface becomes rough with papillomas
 - **Regressive stage:** Lesion decreases in size and papillomas regress
 -





Bovine Viral Diarrhea (BVD)

- **Etiology**
 - Bovine viral diarrhea virus
 - Two biotypes
 - **Cytopathic**
 - Type one and type two
 - **Noncytopathic**
 - Mostly causes disease in cattle, can infect sheep
- **Epidemiology**
 - Has been documented in many countries worldwide
 - Is common in India
 - In the United States the prevalence of antibody positive cattle ranges from 20 to 90 percent
 - **Prevalence depends on density, management practices (vaccination and quarantine of new animals)**
- **Clinical syndromes**
 - Infection with bovine viral diarrhea virus can result in a number of different clinical manifestations ranging from subclinical disease to death
 - Clinical manifestations are dependent on age, immunological status, pregnancy status, and virus type







Bovine Viral Diarrhea: Clinical Syndromes



Subclinical Bovine Viral Diarrhea Virus Infection

- **Most infected animals have subclinical infection that cause:**
 - Mild fever
 - Low white blood cell count
 - Development of antibodies
- **It has been estimated that 70 to 90 percent of bovine viral diarrhea virus occur without clinical diseases**





Acute Bovine Viral Diarrhea Virus Infection

- **Acute infection is defined as clinical disease that occurs in immunologically normal cattle that are not persistently infected**
 - Infection incubation period is about five to seven days
 - Clinical manifestations include:
 - **Fever**
 - **Low white blood cell count**
 - **Depression**
 - **Reduced feed intake**
 - **Oral erosions and ulcerations**
 - **Diarrhea**
 - **Decreased milk production**
- **Differential diagnosis**
 - Neonates:
 - **Rotavirus, coronavirus, cryptosporidium, E. coli, and salmonellosis**
 - Adults:
 - **Salmonellosis, Johne's disease, intestinal parasites, malignant catarrhal fever, vesicular stomatitis, foot and mouth disease, and bluetongue**





Severe Acute Bovine Viral Diarrhea Virus Infection

- **Atypical form was characterized about 20 years ago**
- **Caused severe disease in cattle with a normal immune system**
 - Fever
 - Pneumonia
 - Sudden death
- **Genetic analysis of the isolate revealed differences in the genetic sequence**
 - As a result the virus that caused severe disease was classified as bovine viral diarrhea virus type 2
 - Classic disease is caused by bovine viral diarrhea virus type 1





Bovine Virus Diarrhea Virus Hemorrhagic Syndrome

- **Clinical signs of hemorrhagic syndrome include:**
 - Low platelet and white blood cell count
 - Bloody diarrhea
 - Bleeding from injection sites
 - Fever
 - Hemorrhage from mucosal surfaces
 - Bleeding from the nose
- **Virus induced reduction of platelets is thought to cause the disorder**
- **Differential diagnoses include hemorrhagic septicemia, anthrax, exposure to bracken fern or estrogens**





Bovine Viral Diarrhea Virus

Respiratory Disease

- Bovine viral diarrhea virus has been shown to contribute significantly to bovine respiratory disease complex
- Bovine viral diarrhea has been isolated in association with respiratory disease in the United States
- Infection enables colonization of the lungs by *Mannheimia haemolytica*
- Respiratory signs are typically mild
 - Increased respiratory rate
 - Fever
 - Lack of appetite





Bovine Viral Diarrhea Virus Infection Immunosuppression

- **Bovine viral diarrhea virus reduces the function of the immune system**
- **Reduced immune system function makes infected animals more susceptible to infection by other pathogens**
- **Infection with bovine viral diarrhea virus has been associated with secondary infection with *Salmonella*, *E. coli*, rotavirus or coronavirus**
- **The virus targets macrophages and lymphocytes reducing the animals ability to respond to challenge with other pathogens**



Bovine Viral Diarrhea Virus Reproductive Syndrome

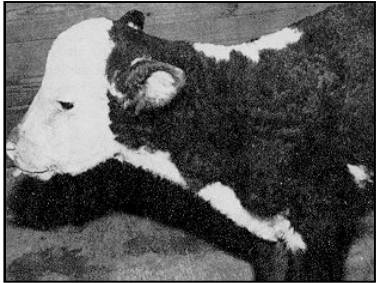
- **Reproductive syndrome - Bulls**
 - Bulls with persistent infection can shed virus in semen
 - **Semen contains infective virus**
 - Bulls that experience acute infection can shed virus in semen for up to 60 days
- **Reproductive syndrome - Cows**
 - Cows may experience problems with infertility, they fail to conceive or experience early embryonic death
 - Abortions and stillbirths are a manifestation of reproductive syndrome
 - Congenital effects occur when pregnant cows are infected and the congenital abnormality is dependent on time of infection during gestation
 - Weak and small calves are borne to cows infected with bovine viral diarrhea virus
 - Persistent infection can result if the pregnant cow is infected before the calves immune system develops – less than 100 days gestation



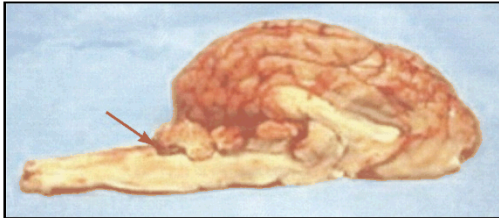


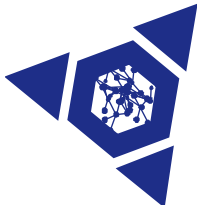
Bovine Viral Diarrhea Virus

Congenital Defects



- **Congenital defects**
 - Infection of fetus between 100 and 150 days gestation
 - Final stages of development of nervous system and immune system disrupted by inflammation caused by the virus
 - **Cerebellar hypoplasia**
 - **Hydrocephalus**
 - **Microphthalmia**
 - **Cataracts**
 - **Retinal atrophy**
 - **Musculoskeletal abnormalities**
 - **Disrupted eye and vision development**
 - **Musculoskeletal deformities**
 - **Thymus not developed (important immune system organ)**
 - **Missing hair or curly hair coat**
 - **Lung/respiratory blood supply disrupted**
 - **Growth retardation**





Bovine Viral Diarrhea Virus Persistent Infection

- **Infection of the fetus with the noncytopathic bovine viral diarrhea virus prior to the development of the immune system results in persistent infection**
 - Because the virus is present before the development of the immune system, the virus is perceived by the immune system as self
 - The immune system is thought to become functional after 100 days of gestation, so infection prior to 100 days results in persistent infection
- **Persistently infected animals can become infected with other forms of bovine viral diarrhea virus and therefore may have antibodies against the virus**
 - Thus cannot be identified by the lack of antibodies to the virus
- **Persistently infected animals may be small at birth, fail to thrive, and seem to be susceptible to infections**
- **Persistently infected animals survive and continuously shed virus**
 - Are often the source of new outbreaks





Bovine Viral Diarrhea Virus

Acute Mucosal Disease

- **Occurs when cattle that are persistently infected with the noncytopathic type become infected with a cytopathic type of bovine viral diarrhea virus**
- **Has different clinical presentations depending on the viral strains involved**
 - Acute mucosal disease
 - Disease occurs 10 to 14 days after exposure and manifests as fever, reduced appetite, high heart rate, decreased milk production, and profuse watery diarrhea
 - Oral papillae may be blunted and the epithelium of the tongue, palate, buccal surfaces, and pharynx may have erosions
 - Erosions may occur on the teats and interdigital region of the feet
 - All erosions may be ulcerative to diphtheric
 - Other signs may include nasal and ocular discharge, corneal opacity, excessive salivation and bloat
 - Over 90 percent of infected animals die after five to seven days
- **Differential diagnosis**
 - Severe bovine viral diarrhea infection, rinderpest, malignant catarrhal fever
 - Differentials that have oral erosions but no diarrhea include foot and mouth disease, vesicular stomatitis, and bovine papular stomatitis





Bovine Viral Diarrhea Virus

Chronic Mucosal Disease

- **Cattle that develop mucosal disease and do not die within seven days become chronically infected**
- **Chronically infected cattle are:**
 - Unthrifty
 - Chronic diarrhea
 - Chronic bloat
 - Reduced appetite
 - Weight loss
 - Interdigital erosions
 - Nasal and ocular discharge
 - Hoof deformities
 - Anemia, low white blood cell count, and low platelet count
 - Rarely survive more than 18 months





Bovine Viral Diarrhea Virus Transmission

- **Source and Transmission**
 - Because they appear clinically normal, persistently infected animals are the primary reservoir for virus
 - Acutely infected animals are a source of virus for transmission
 - **Shedding is less than with persistent infection, cattle acutely infected have a shorter duration of shedding and do not shed as much virus**
 - Inhalation and ingestion of the virus is the most common mode of infection
 - Indirect contact through vectors and contaminated supplies can result in infection
 - Contaminated semen is a source of infection and can occur through artificial insemination
 - Vertical transmission from the dam to the fetus results in persistent infection or congenital anomalies depending on the time of infection
 - Cattle can transmit the virus to sheep, camelids, and wild ruminants
- **The spread of bovine virus diarrhea occurs mostly with the introduction of a persistently infected animal into a herd**



Bovine Viral Diarrhea Virus Diagnosis

- **Diagnosis of bovine viral diarrhea virus can be made by:**
 - Serology
 - Virus isolation
 - Viral antigen detection
 - Viral gene detection
- **Diagnostic testing for bovine viral diarrhea is complex and depends on the disease syndrome that is suspected**
 - **Acute infection**
 - **Serology three to four weeks apart can detect rising titers**
 - Serum should be collected in a tube with no anticoagulant – red top
 - **Virus isolation or polymerase chain reaction assays (test for genetic material) can detect virus in active infections**
 - Nasal swab
 - Whole blood in an heparinized tube (green top)
 - **Tissue from necropsy**
 - Ileum, mesenteric lymph nodes, spleen, thymus, and lung can be collected for fluorescent antibody test, virus isolation, or polymerase chain reaction assay





Bovine Viral Diarrhea Virus Diagnosis

- **Diagnostic testing for bovine viral diarrhea depends on the disease syndrome that is suspected**
 - **Infertility**
 - **Serology can be done to establish previous exposure history**
 - Serum should be collected in a tube with no anticoagulant – red top
 - **Abortion**
 - **Serology – paired sera three to four weeks apart**
 - Serum should be collected in a tube without anticoagulant
 - **Fetal fluid – to detect antibodies**
 - Collect in a clean container without anticoagulants
 - **Fetal tissue – to detect antigen using isolation, polymerase chain reaction assays (PCR), fluorescent antibody**
 - Ileum, mesenteric lymph nodes, spleen, thymus, liver, and lung are the best tissues to collect





Bovine Viral Diarrhea Virus Diagnosis

- **Diagnostic testing for bovine viral diarrhea depends on the disease syndrome that is suspected**
 - **Persistent infection**
 - **Virus isolation, polymerase chain reaction, ELISA to detect virus**
 - Serum or whole blood should be used – heparinized or blood without anticoagulant can be used
 - Skin biopsy (ear notch) can be used to identify antigen
 - **Mucosal disease**
 - **Virus isolation, polymerase chain reaction, immunofluorescence, ELISA to detect virus**
 - Serum or whole blood should be used – heparinized or blood without anticoagulant can be used
 - Tissues from necropsy can also be used
 - **Herd screening**
 - **Milk – polymerase chain reaction assays (PCR) to look for viral shedding**
 - **Serum – antibody detection in herd, use unvaccinated animals**



Bovine Viral Diarrhea Virus SNAP Test

- A stall side test used to detect bovine viral diarrhea virus in persistently infected cattle
- Can be completed in 20 minutes using an ear notch as the sample
- A useful screening tool to evaluate bovine viral diarrhea status in herds





Bovine Viral Diarrhea Virus Control

- **Good husbandry practices aimed at preventing the introduction of bovine viral diarrhea to a farm**
 - In the United States it is not uncommon to have closed herds where replacement animals are raised at the farm and animals are not purchased from other farms
 - If new animals are introduced, testing and isolation should be done to prevent introduction
 - Limiting animal movement, limits exposure to virus
- **Vaccination programs are aimed at increasing herd immunity that will reduce viral replication after infection**
 - Modified live vaccines should not be used in pregnant animals, the vaccine can cause fetal anomalies
 - Killed vaccines can stimulate immunity, however, immunity is aimed at reducing clinical disease and viral shedding
 - Animals should be vaccinated before they are bred to optimize immunity
- **Screening**
 - In herds where bovine viral diarrhea is thought to be problematic, screening of animals should be done





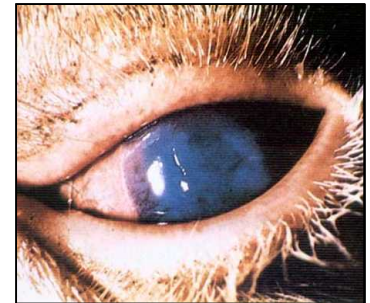
Malignant Catarrhal Fever

- **Etiology**

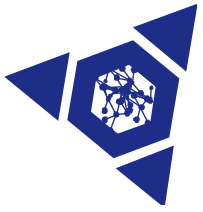
- Acute malignant catarrhal fever is a highly fatal disease of cattle, bison, and other ruminants
- Sheep experience subclinical infection
- It is caused by a herpes virus

- **Clinical signs**

- All ages of cattle are affected
- The virus attacks the vascular endothelium causing a vasculitis resulting the following clinical signs
 - Oral erosions
 - Necrosis of the muzzle
 - Diarrhea
 - Conjunctivitis
 - Mucopurulent nasal discharge
 - Thickened skin
 - Encephalopathy
 - Enlarged lymph nodes



Mortality is high



Malignant Catarrhal Fever

- **Differential diagnosis**

- Bovine viral diarrhoea mucosal disease
- Rinderpest
- Bluetongue
- Vesicular stomatitis
- Foot and mouth disease
- Foot and mouth disease and vesicular stomatitis are not usually associated with diarrhoea and the mortality rate is not high despite high morbidity
- Bluetongue is rare in cattle and not usually associated with diarrhoea





Malignant Catarrhal Fever

- **Diagnosis**
 - Serology to detect antibodies can be done using ELISAs and virus neutralization
 - Antigen detection by PCR is definitive
- **Epidemiology**
 - Malignant catarrhal fever is spread to cattle by wildebeests
 - Sheep associated virus is spread to the cattle population via sheep
 - Malignant catarrhal fever has world wide distribution
 - Has been detected in India
 - Most cases only involve a single animal; however, large outbreaks have been reported
- **Treatment and prevention**
 - Limit exposure to sheep and wildebeest reservoirs
 - There is no vaccine to prevent the disease





Foot and Mouth Disease

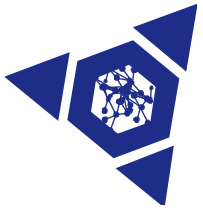
- **Etiology**

- Foot and mouth disease is a highly contagious disease of cloven hooved animals and is characterized by vesicular lesions, erosions, and ulcers in the mouth, interdigital areas, coronary band, muzzle and teats
- Seven immunologically distinct types of FMD exist
 - **A, O, C, SAT 1, 2, 3, and Asia 1**
 - **At least 60 subtypes have been identified**

- **Clinical signs**

- Fever, depression, lack of feed intake, excessive salivation, lip smacking, nasal discharge, and lameness
- Morbidity is high but mortality is low – animals infrequently die from FMD





Foot and Mouth Disease

- **Differential diagnosis**
 - Bovine papular stomatitis, rinderpest, malignant catarrhal fever, severe infectious rhinotracheitis, and some forms of bovine viral diarrhea
- **Diagnosis**
 - Antigen detection
 - **Virus isolation**
 - **ELISA**
 - **PCR**
 - The ideal specimen to collect is epithelial tissue or vesicular fluid from a vesicle that has not been ruptured
 - **The specimen should be transported in medium composed of phosphate buffer and glycerol**
 - If not available, cell culture media and/or phosphate buffered saline can be used
 - pH must be kept between 7.2 and 7.6
 - Oropharyngeal fluid can also be used for virus detection
 - **The fluid can be collected with a probang or via a nasogastric tube**



Foot and Mouth Disease Diagnostic Samples

- **Diagnostic samples**
 - Vesicular fluid
 - Oropharyngeal fluid using a probang or possibly a orogastric tube
 - Epithelial tissue from a vesicle that has not ruptured or a recently ruptured vesicle
 - **Collection of epithelial tissue is painful and will require sedation or local anesthetic**





Foot and Mouth Disease Control

- **Types A, O, and Asia 1 are most prevalent in South Asia**
- **Must vaccinate against individual strains – there is no cross protection**
- **Vaccine manufacturers are located worldwide, must identify manufacturer with the most appropriate strain**
 - Merial
 - MSD Animal Health
 - Brilliant BioPharma
 - India Immunologics
- **In 2011 – type O was the most commonly isolated FMD type**
 - 80 percent of samples sent to the World Reference Laboratory for FMD were type O
 - Has been an increase in Asia 1 especially in South Asia
- **The FAO and OIE have initiated the Progressive Control Program for FMD**
 - The progressive control program has six steps that aid in the control of FMD
 - Stages 0 – 5
- **Afghanistan is currently in Stage 1**

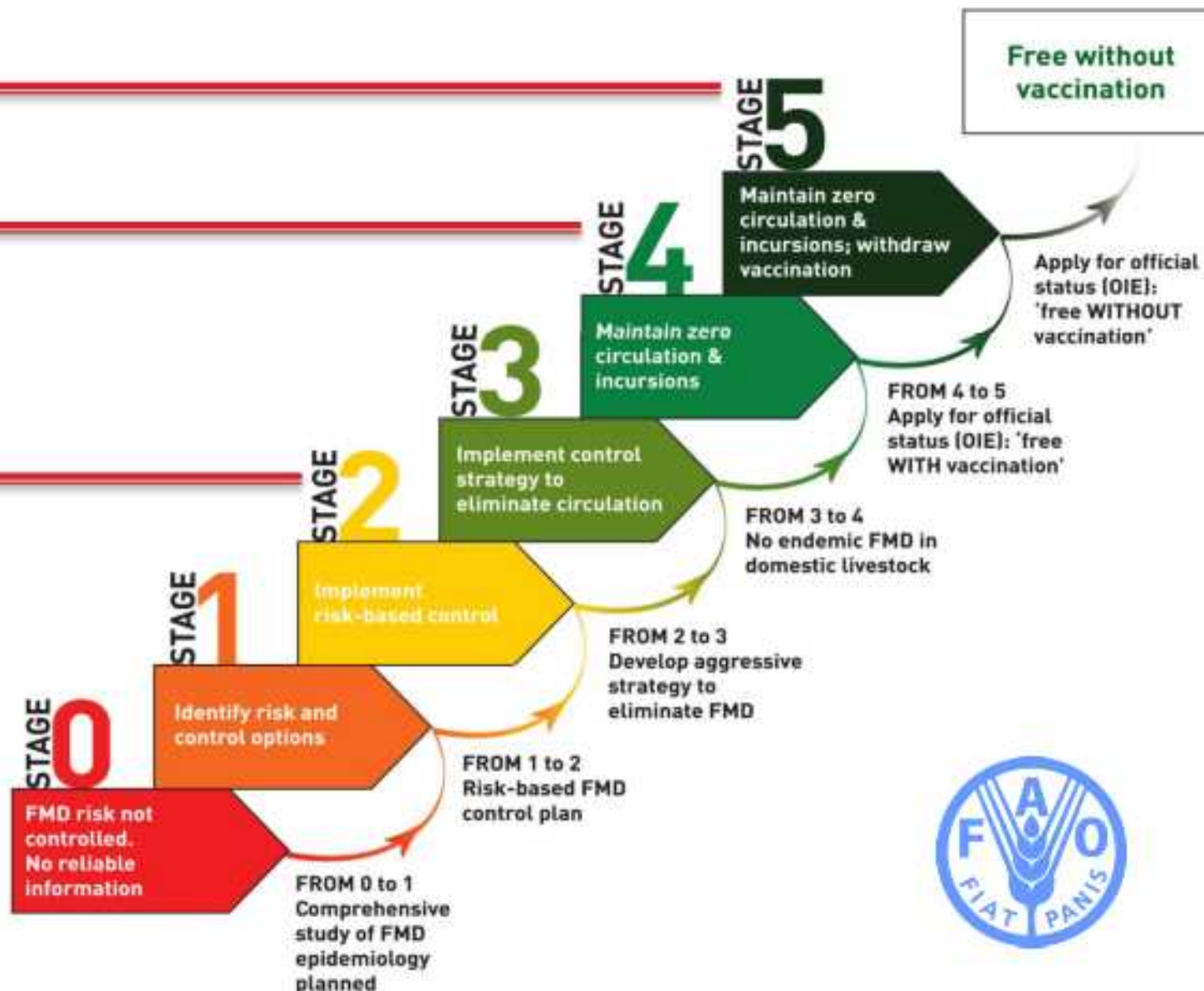


Free without vaccination

Free with vaccination (5)

Infected with endorsed program (3-4)

Infected without endorsed program (0-2)





Foot and Mouth Disease Transmission

- **Highly contagious**
 - Foot and mouth disease virus is thought to be the most contagious virus
 - The virus is easily spread from animal to animal by direct contact, indirect contact, and aerosols
 - **During the outbreak in the United Kingdom, it was determined that the foot and mouth disease virus traveled up to 300 kilometers in an aerosol**
 - Foot and mouth disease virus is very stable in the environment
 - **Stability is enhanced during colder months with higher humidity**
 - **Control is difficult with naïve populations**





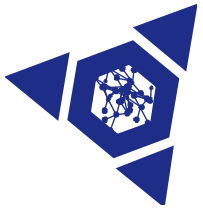
Diseases Caused by *Clostridium perfringens*

- **Clostridium perfringens is a toxin producing, anaerobic, spore forming bacteria**
- **The organism is found in the soil and sometimes a commensal intestinal organism**
- **Clinical isolates are characterized as types A, B, C, D, E based on the type of toxin they produce**
 - Four major toxins are produced
 - **Alpha**
 - **Beta**
 - **Epsilon**
 - **Iota**



Clostridium Perfringens Types

- ***Clostridium perfringens* Type A: produces alpha toxin**
 - Gas gangrene, avian necrotic enteritis, hemorrhagic enteritis in cattle, yellow lamb disease, Type A enterotoxemia of horses, abomasal tympany and ulcers of calves, food poisoning in humans
- ***Clostridium perfringens* Type B: produces alpha, beta and epsilon toxin**
 - Lamb dysentery
- ***Clostridium perfringens* Type C: produces alpha and beta toxin**
 - Necrotic enteritis of calves, lambs, kids, and foals
- ***Clostridium perfringens* Type D: produces alpha and epsilon toxin**
 - Entertoxemia (overeating disease) of sheep, goats, and cattle



Clostridium Perfringens Type A

- **Type A can be found in many soils and is a normal inhabitant of the gut in many species**
- **Yellow Lamb Disease**
 - The toxin causes lysis red blood cells which increases the free bilirubin concentration in the blood causing jaundice
 - Lambs usually have a fever, are weak, and die within 6 to 12 hours
 - May also cause abomasitis, abomasal distention and tympany in neonatal calves
- **Hemorrhagic Bowel Syndrome**
 - Fatal disease of cattle that is characterized by hemorrhage in the intestines
 - Emerging disease of lactating dairy cattle in the United States
 - Thought to be related to feeding practices to promote production
 - Cattle have a high heart rate and low temperature
 - **Feces contain undigested feed and clotted blood**
 - **Distended small intestines may be palpable on rectal examination**
- **There is no vaccine for *Clostridium perfringens* type A**





C. Perfringens Type B Lamb Dysentery

- **Lamb dysentery**
 - Occurs in lambs less than one week of age
 - Lambs become depressed and die suddenly
 - **Usually lambs will have yellowish diarrhea that progresses to brown and bloody**
 - **High morbidity and almost 100 percent mortality**
 - **Death is usually sudden**
 - Ulcers may be found in the small intestines
 - Sanitation and use of vaccine and toxoid products aid prevention and control





Clostridium Perfringens Type C

Necrotic Enteritis

- **Clinical signs**
 - Yellow diarrhea that may have blood or gray streaks of necrotic mucosa
 - Foals with the infection will display abdominal pain and explosive diarrhea
 - Dehydration, depression, and weakness despite therapy
- **Epidemiology**
 - Neonates ingest the organism from the environment from an asymptomatic shedder or contaminated feed
- **Treatment, prevention, and control**
 - Intravenous fluids, broad spectrum antibiotics, intravenous plasma,
 - Toxoids have been given to horses to prevent the disease on contaminated farms





Clostridium Perfringens Type D Enterotoxemia

- **Clinical signs**
 - Most common presentation is the sudden death of a well-fed rapidly growing animal
 - Affected lambs may also show neurological signs including ataxia, convulsions, profound depression, stiff limbs, or opisthotonus
 - Initially the animal is hyperglycemic and will have glucose in the urine at death
- **Differential diagnosis**
 - Diseases that present with neurologic signs and sudden death
 - **Anthrax, botulism, black disease, leptospirosis, listeriosis, enterotoxigenic E. coli infection, severe septicemia, polioencephalomalacia, grain overload, hemorrhagic septicemia, and tetanus**

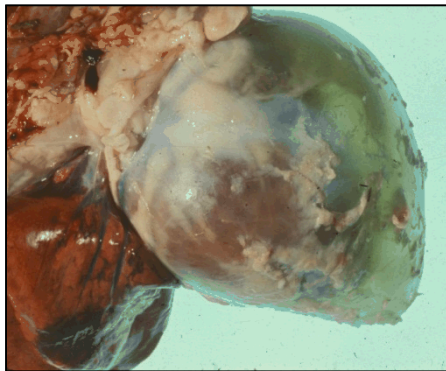




Clostridium Perfringens Type D Enterotoxemia

- **Necropsy**

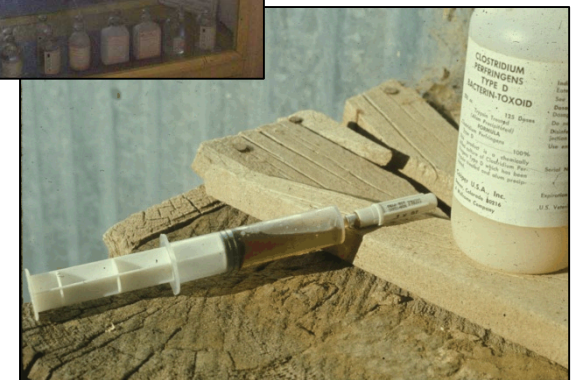
- The epicardium, serosa, thymus, and the diaphragm may have small areas of hemorrhage
- The pericardial sac often contains excess fluid
- Glucose in the urine is the hallmark of disease
 - **Can use urine dipsticks to detect glucose**





Clostridium Perfringens Type D Enterotoxemia

- **Pathophysiology**
 - High carbohydrate diet allows the bacteria to proliferate rapidly and produce toxin
- **Treatment**
 - If identified early, antitoxin and oral sulfa antibiotics can be curative
 - Diets should be adjusted to reduce the substrate, lambs on rich pasture should be moved to less lush pasture
 - Vaccination with type D bacterin-toxoid is effective at preventing the disease
 - **Two doses given 14 to 56 days apart, before heavy grain feeding or exposure to lush pastures**





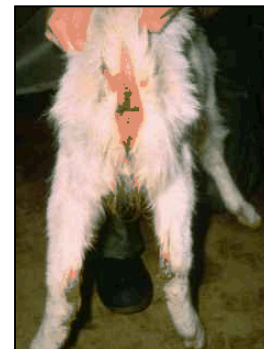
Pestes des Petits Ruminants

- **Etiology**

- Affects primarily sheep and goats and is closely related to rinderpest virus

- **Clinical signs**

- In some cases affected animals may develop high fevers, severe depression, and die suddenly
- Most clinical cases appear with:
 - **Sudden high fever, reluctance to eat and drink, marked depression**
 - **Soon after the onset of clinical signs, serous nasal and ocular discharge develops**
 - **After a few days the discharge turns mucopurulent**
 - **Small, gray, necrotic foci covering shallow erosions appear in mouth**
 - Erosions on lips, gums, dental pads, palate, cheeks, and gums
 - **Profuse diarrhea develops and animals become dehydrated, emaciated, and die**





Pestes des Petits Ruminants

- **Differential diagnosis**
 - Pneumonic Pasteurellosis and contagious caprine pleuropneumonia
 - **Will not see diarrhea with these diseases, they are mostly respiratory**
 - Foot and mouth disease
 - **No diarrhea or respiratory problems**
 - Bluetongue
 - **May not see clinical signs in indigenous breeds, not widespread**
 - Orf
 - **Orf will not produce systemic signs**
- **Epidemiology**
 - The disease is widespread in Asia and the Middle East
 - PPR is spread by close contact and aerosols plays an important part in the transmission
 - The virus is shed in urine, feces, milk, and nasal and ocular discharges



Pestes des Petits Ruminants Diagnosis

- **Diagnosis**
 - Virus detection
 - **Virus isolation**
 - Virus can be isolated from blood in acutely ill animals, blood should be collected and placed in a heparinized tube (green top) or a tube containing EDTA
 - **ELISA, PCR, and virus isolation**
 - **Tears and gum debris can be used to look for virus using**
 - Tears can be collected by swabbing the conjunctival sac with a cotton swab and placed in a container with 2 milliliters of phosphate buffered saline
 - Gum debris can be collected by rubbing the fingers across the gum and inside the upper and lower lips, the material should be scraped into a container containing 2 milliliters of phosphate buffered saline
 - **Tissues from post mortem examination including lymph nodes around the lungs and gastrointestinal tract, and portions of spleen and lungs**
 - Two sets of tissues should be collected, one stored on ice, the other in formalin
 - **Serology**
 - **Antibody measurement requires clotted blood or serum**



Pestes des Petits Ruminants Treatment

- **Treatment**
 - Supportive care to replace fluid losses and broad spectrum antibiotics to protect against secondary bacterial infections
 - **Animals that recover have immunity lasting four years or longer**
 - Ring vaccination and quarantine can help slow the spread of the virus during an outbreak
 - Vaccination can protect animals against infection
 - **Immunity induced by the vaccine can last up to three years**



Johne's Disease

- **Etiology**

- Johne's disease is a chronic disease caused by *Mycobacterium avium subspecies paratuberculosis* that primarily affects ruminants
- The bacterium is ingested in the perinatal period

- **Clinical signs**

- Majority of infected animals appear clinically normal, but over a prolonged period of time, two to ten years, infected animals start to show clinical signs
 - **Weight loss despite normal appetite, intermittent diarrhea, decreased milk production, and finally progression of diarrhea to constant watery diarrhea**
 - **Protein may get low enough to cause edema**





Johne's disease

- **Pathophysiology**

- *Mycobacterium avium subspecies paratuberculosis* enters the intestinal tract via ingestion and is taken up by the cells of Peyer's patches
- The organism proliferates in macrophages and then spreads to the lymph nodes
- An inflammatory response is initiated
- The intestines become thickened and lose their absorptive capability





Johne's Disease

- **Transmission**

- Fecal-oral transmission
 - **Neonates ingest the organisms by sucking manure contaminated teats or eating contaminate feed material**
- In utero
 - **If the dam is showing clinical signs during pregnancy, 25% of fetuses will be infected in utero**
- Infected colostrum and milk can be a source of infection

- **Diagnosis**

- Diagnosis can be made based on clinical signs, cattle with a good appetite and profuse diarrhea are likely to be infected
- Isolation of the organism from feces is the gold standard
 - **The organism is difficult to isolate**
 - **Negative culture doesn't rule out infection**
 - **Expensive**
 - **Pooling fecal samples from herd may decrease costs**
- Polymerase chain reaction assays can be used to detect the organism and is probably the most sensitive method of detection





Johne's Disease Treatment

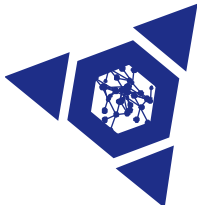
- **Treatment**
 - No good therapy exists for the treatment of Johne's disease
 - A killed vaccine is available in several states but does not eliminate the infection
- **Epidemiology**
 - Johne's is widespread in the United States and results in tremendous losses in production
 - Johne's is also problematic in India







Hepatobiliary Diseases



Black Disease

- **Etiology**

- Infectious necrotic hepatitis is a disease of grazing animals but mostly affects sheep
- The disease is caused by *Clostridium novyi type B*

- **Pathophysiology**

- *Clostridium novyi type B* is widely distributed in soils
- The spores are ingested by grazing animals
- Disease only occurs when there is sufficient liver damage to allow the organism to colonize the liver
 - **Liver damage is almost always caused by the migration of liver flukes**
- As the organism proliferates, it produces toxins that cause liver necrosis

- **Clinical signs**

- Sudden death is the most common clinical finding
- Animals will be depressed, isolated, high fever, and respiratory distress
- Animals are usually sickened during the warmer months when fluke transmission is active





Black Disease

- **Differential Diagnosis**

- Other causes of sudden death
 - Anthrax, botulism, black disease, enterotoxigenic E. coli infection, severe septicemia, grain overload, hemorrhagic septicemia, bacillary hemoglobinuria, enterotoxemia

- **Diagnosis**

- Diagnosis is made by necropsy and typical findings include
 - Engorgement and hemorrhage of subcutaneous tissues resulting in a black discoloration
 - The abdominal, thoracic, and pericardial fluids are tinged
 - Hemorrhages are found throughout
 - Migrating fluke channels will be present in the liver
- Smear from liver should reveal gram positive rods typical of Clostridium





Black Disease

- **Treatment**
 - Treatment of infected animals is usually unsuccessful
- **Prevention**
 - Commercial bacterin/toxoids against *Clostridium novyi* are available
 - **Protection is short lived and provides immunity for approximately six months**
 - Control liver fluke infestation
 - **Limit animals access to areas where flukes reside such as streams, ponds, canals, and marshes**
 - **Treat animals with products that eliminate flukes**
 - Ivermectin and clorsulon combination
 - Albendazole



Bacillary Hemoglobinuria

Redwater

- **Etiology**

- Cause of sudden death in cattle caused by *Clostridium haemolyticum* also known as *Clostridium novyi type D*

- **Pathophysiology**

- *Clostridium haemolyticum* is found in alkaline soils
- The spores are ingested by grazing animals
- Disease only occurs when there is sufficient liver damage to allow the organism to colonize the liver
 - **Liver damage is almost always caused by the migration of liver flukes**
- As the organism proliferates, it produces toxins that cause hemolysis

- **Clinical signs**

- Sudden death is the most common clinical finding
- When the disease is recognized animals are reluctant to move, have a high fever, rapid shallow breathing, and infrequently, dark red urine is present



Bacillary Hemoglobinuria

- **Differential diagnosis**
 - Other causes of sudden death
 - Anthrax, botulism, black disease, enterotoxigenic E. coli infection, severe septicemia, grain overload, hemorrhagic septicemia, Black disease, enterotoxemia
- **Diagnosis**
 - Necropsy findings are the primary method of diagnosis
 - Diagnosis is made by necropsy and typical findings include
 - Subcutaneous and petechial hemorrhages
 - The abdominal, thoracic, and pericardial fluids are tinged
 - Dark red urine may be present in the bladder
 - Coagulative necrosis of the liver
 - Migrating fluke channels will be present in the liver





Bacillary Hemoglobinuria

- **Treatment**
 - Treatment of infected animals is usually unsuccessful
- **Prevention**
 - Commercial bacterin/toxoids against *Clostridium haemolyticum* are available
 - **Protection is short lived and provides immunity for approximately six months**
 - Control liver fluke infestation
 - **Limit animals access to areas where flukes reside such as streams, ponds, canals, and marshes**
 - **Treat animals with products that eliminate flukes**
 - Ivermectin and clorsulon combination
 - Albendazole