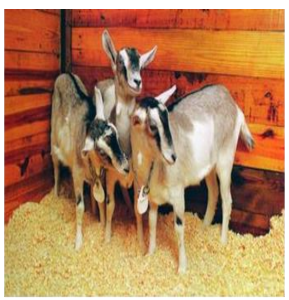
ADDIS ABABA UNIVERSITY

Collage of Veterinary Medicine & Agriculture

Program: VLT, Year III

Fundamentals of Farm Animal Disease



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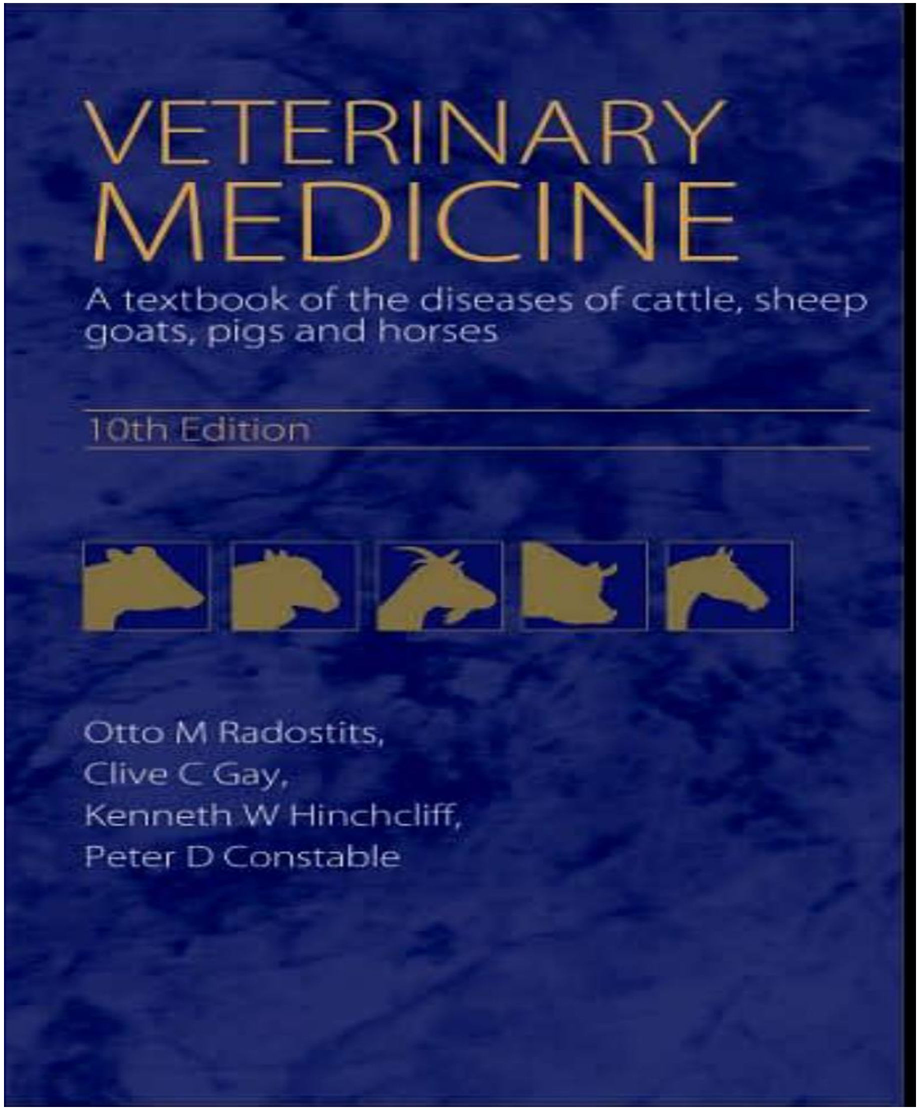
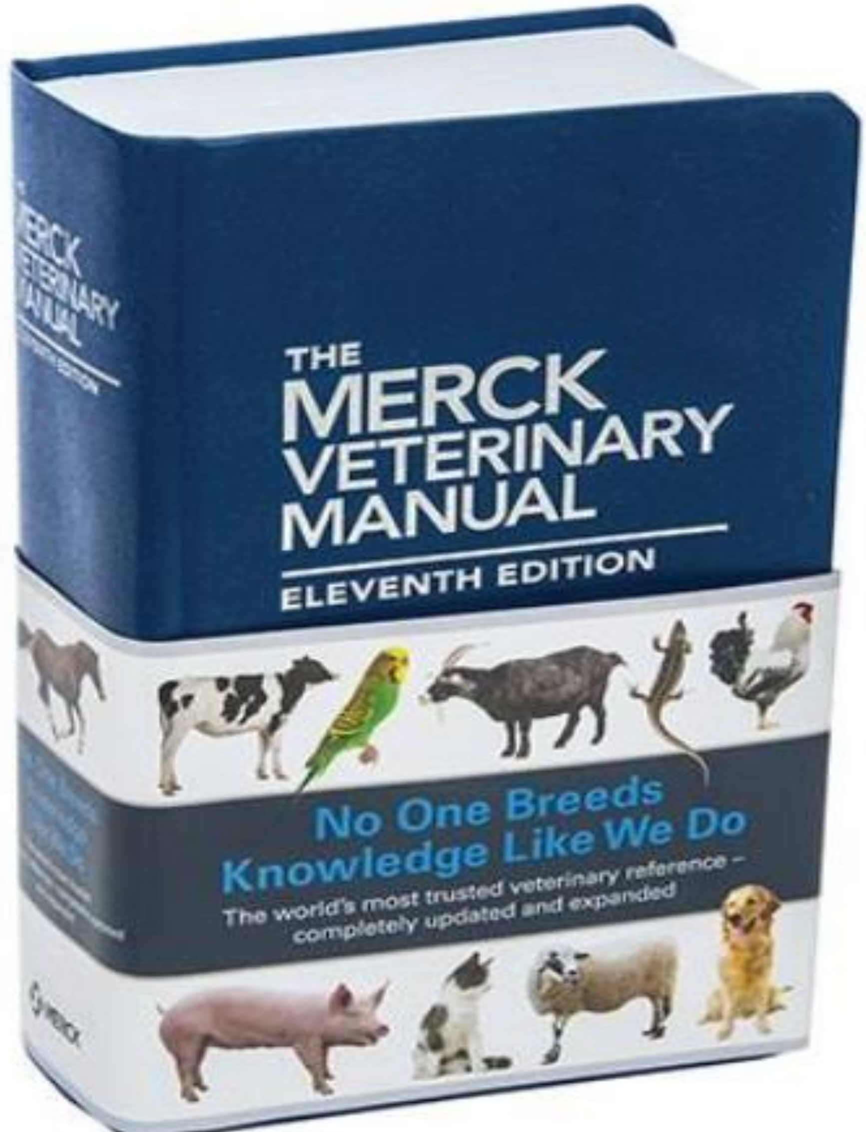
Recommended Text Books





Chapter One

Introduction

Definitions

What are Farm Animals?

- **Farm animals** are animals **raised** or **kept primarily** for **consumption**, to **generate income** and/or to **help with some farm operations**

- **Another name** for them is **Livestock**

  - The **term livestock** is normally defined as **animals raised to produce milk**, **meat**, **work**, **egg** and **wool**

- The difference between farm animals and wild animals is that **farm animals live among men** while **wild animals live in the wild**, forest, jungle etc

- The common **farm animals** are **goats**, **sheep**, **cattle** (dairy cows), **camels**, **buffalos**, **horses**, **donkeys**, **mules**, **pigs**, **poultry** and **bees**

What is Health?

- **Health** is **a state of complete physical**, **mental**, and **social well-being** (i.e. it **is not merely** the **absence** of **disease** or **illness**)

Whereas disease is?

- **Disease** is **any deviation** from **normal physical** and **physiological** conditions
- It is **a change** of **structure** or **function** of **a host** caused by an infectious agent or a non infectious agent
- **Any condition** that causes the **systems of an animal** not to function properly
- Broad definition – **not being at ease** (uncomfortable)

1.1. Importance of livestock

- There are **many reasons** attributed to **raising farm animals**, **these include:**

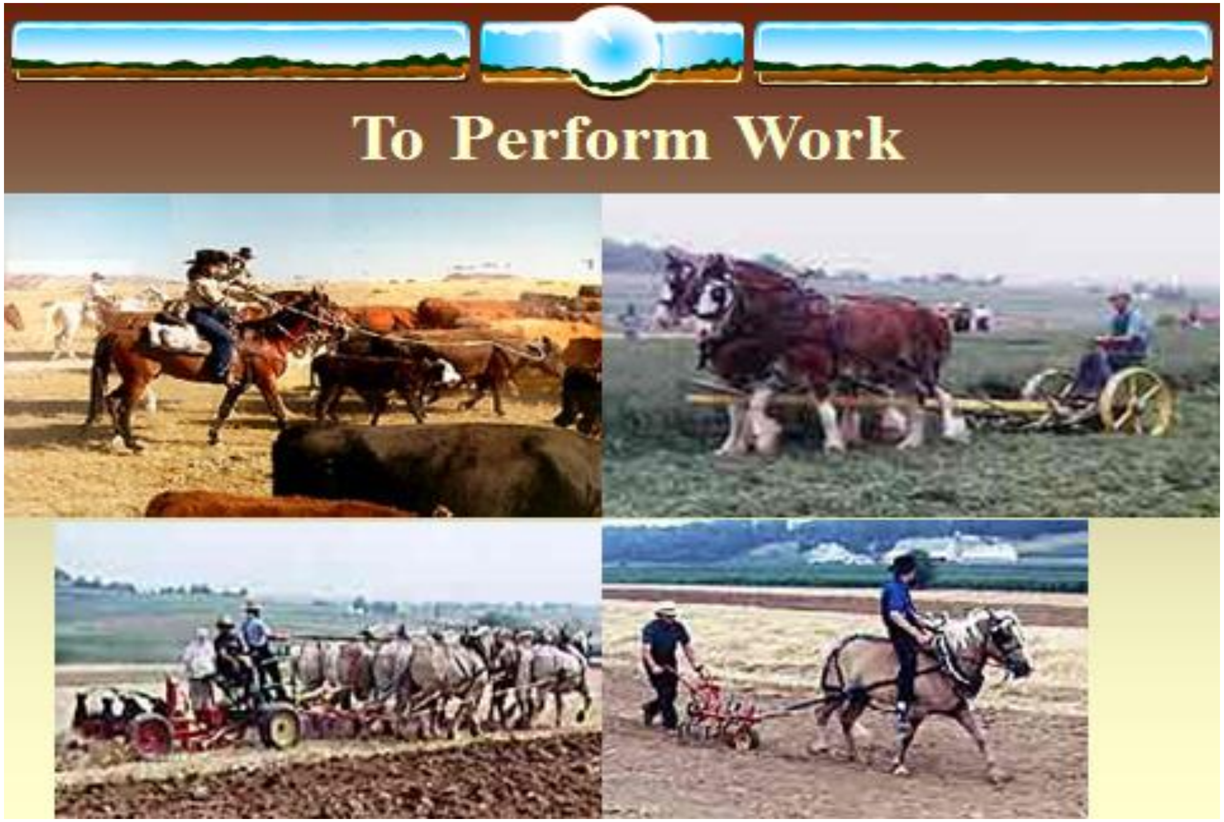
  - As **food elements** **such as** meat, milk, egg & honey productions:

Animal products are critical to the nutrition, food security, livelihoods and resilience of hundreds of millions of people throughout the world

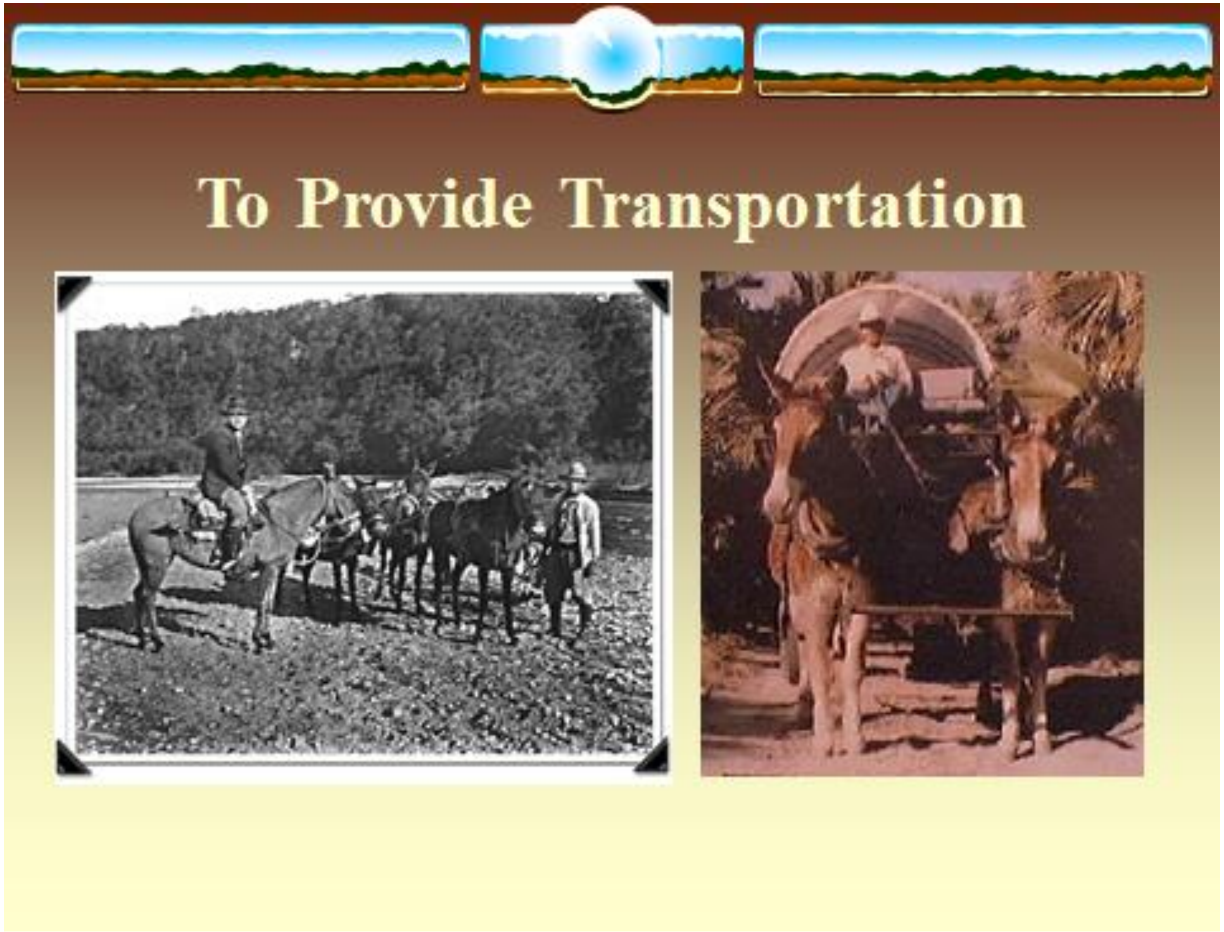
- **To generate cash (income generation)** for owners following **sell of animal** & animal products

- To serve as bank (Livestock are capital assets)

- To **generate draught power** (to **Perform Work**):



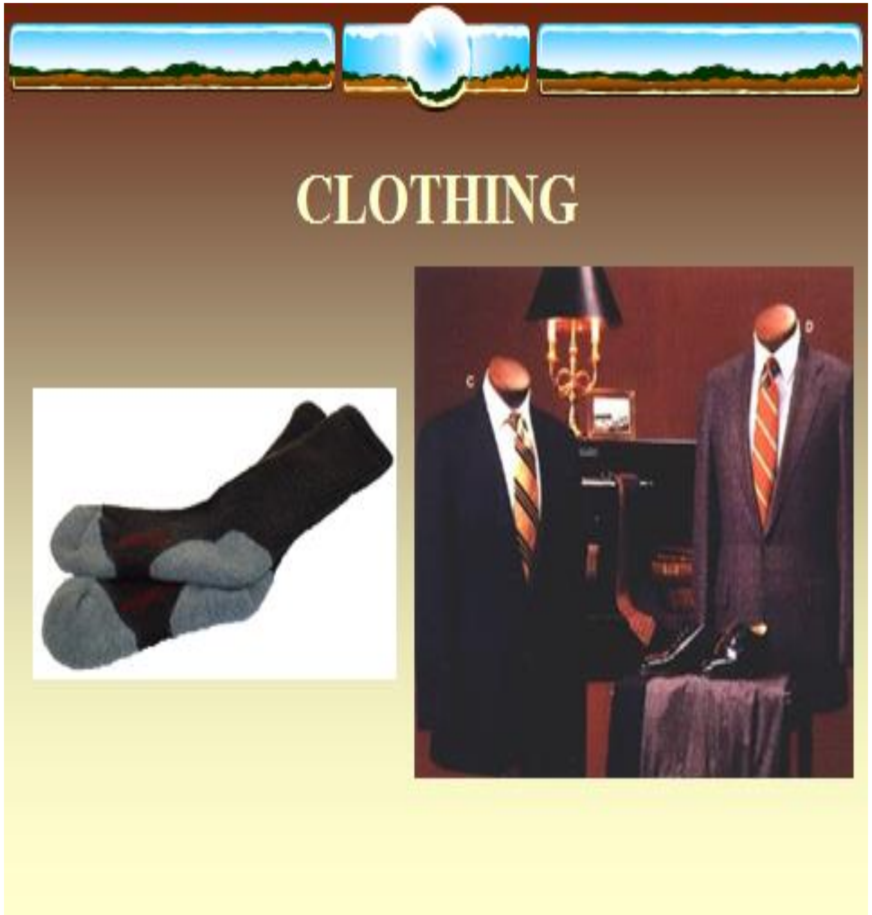
- To Provide **Transportation:**

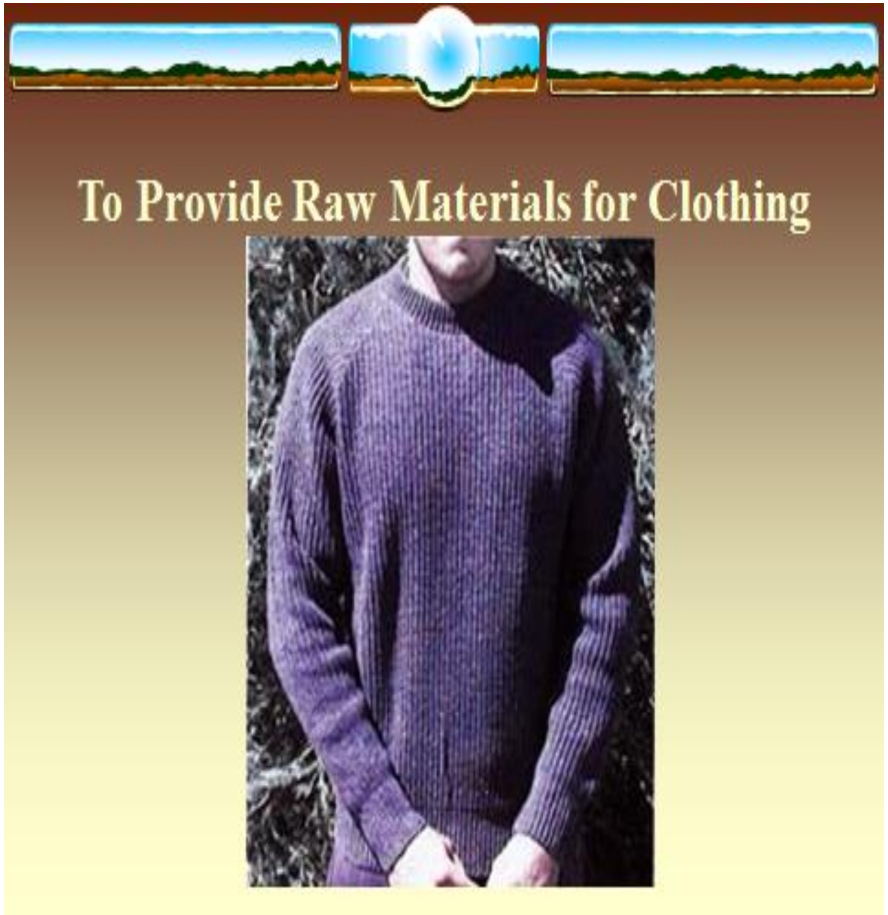


- To get **dung** to use as **source of fertilizer** & **fuel** for fire
  - ✓ The animal-wastes do not go to waste - they make excellent natural fertilizers

- To Provide **Raw Materials** for **Clothing:**

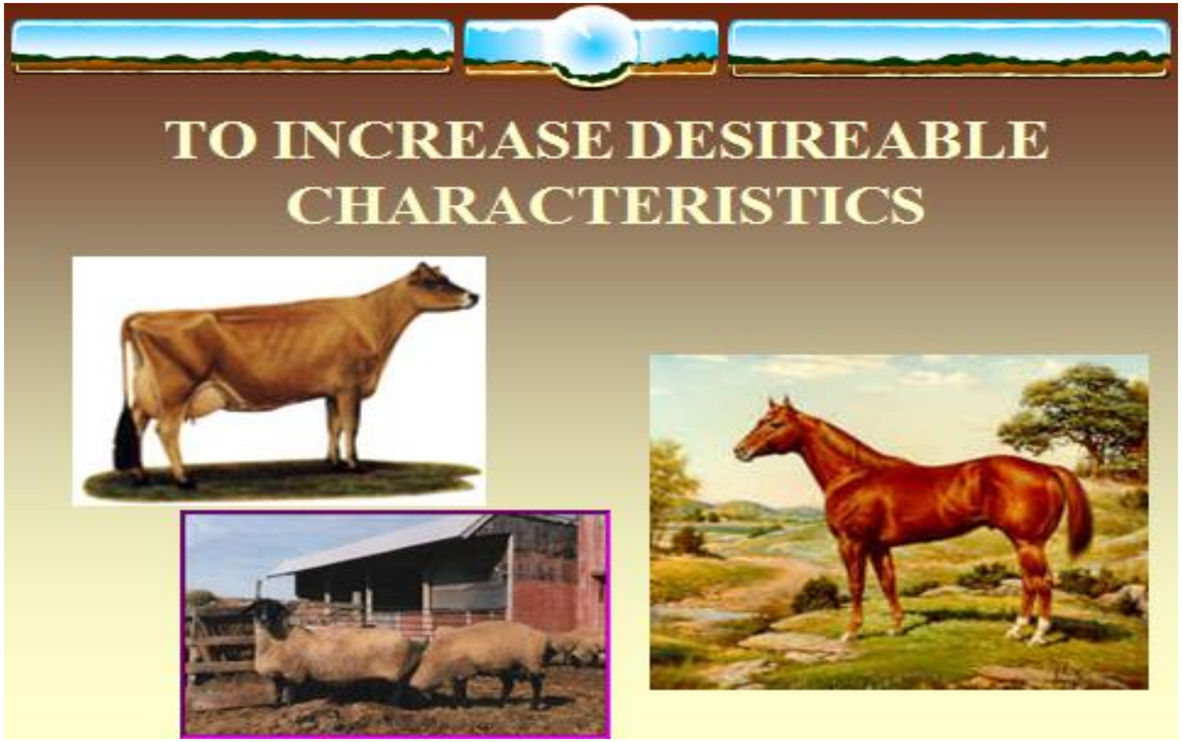
  - ✓ The **skins** or **hides** and even hair of **these animals** have been used to make **blankets**, **clothing**, **shoes** and the like





- To Provide **Entertainment** & **Recreation:**

- To **increase** **desirable characteristics:**



- The usefulness of **livestock organs** in **medicines** like insulin has been understood only recently

- **To top of these**, livestock contribute **16% of GDP** and 14% of the Ethiopia foreign exchange earnings through export

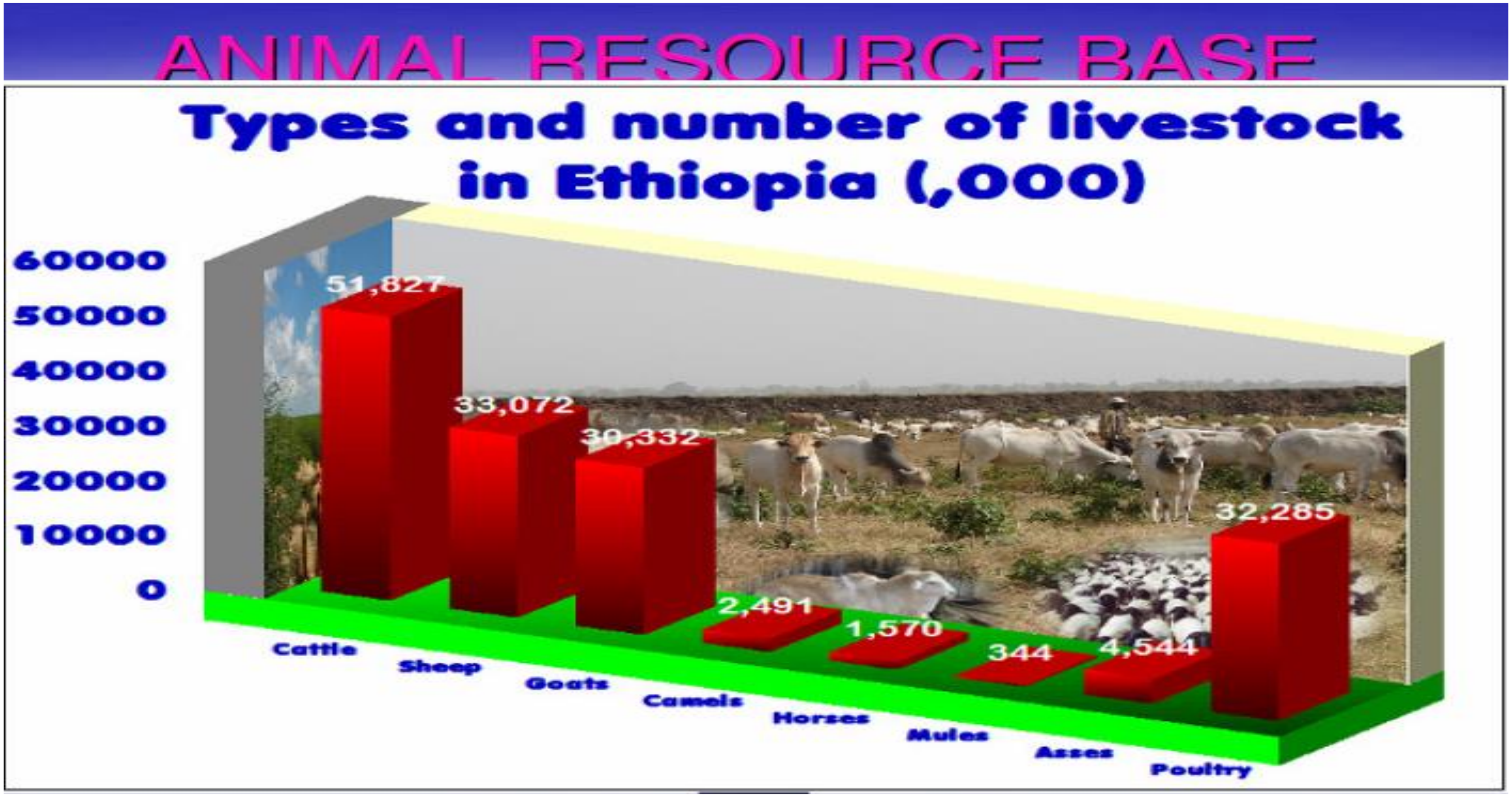
Livestock accounts for 40% of worldwide income from agriculture

Importance of livestock in developing countries

- Livestock as an important **food source**
  -Trends in livestock as a food security commodity in developing countries
  - Livestock help to alleviate seasonal food variability.
- Livestock as a **source of income**
- Livestock as a **generator of employment**
- Livestock as a **source of energy**
  - Draught animal power
  - Dung for fuel
  - Biogas production.
- Livestock as a source of **compost/fertilizer and soil conditioner**
- Livestock as a **weed control**
- Livestock for **investment and savings** .

1.2. Constraints of livestock productivity

- **Ethiopia's livestock population** is **the largest in Africa** (the rough estimates of **national livestock populations** for **2008/09** was **59 million** cattle, **35 million** sheep, **31** million goats and **38 million** poultry)



- **Despite these huge livestock resources**, its contribution for **growth of household** and **national economy is limited**
- Thus, the **livestock sub sector contributes only** about **16-20% of the total Gross Domestic Product** (GDP)

ECONOMIC CONTRIBUTION OF THE SECTOR AND PER CAPITA CONSUMPTION

Item No	Parameter	Unit	Level
1	Agric. GDP	Percentage	30-40
2	Contribu. to GDP	Percentage	16-20
3	Contribu. to foreign Exchange	Percentage	14-16
4	Beef per cap	Kg/per person /year	4.6
5	Mutton Per cap	Kg/per person /year	2.8
6	Milk per ca[	Kg/per person /year	16

- **Different factors** or **constraints limit the full exploitation** of the livestock sector productivity

- **Major constraints affecting** the **performance** (productivity) of the **livestock sector** include:

  - **Inadequate feed** and **water** both in **quantity** and **quality**
  - **Widespread livestock diseases** and poor health (**poor veterinary service**)
  - **Poor genetic potential for production traits** (**low genetic potential** with respect to productivity) ⇨ **local** vs **exotic**
  - **Inadequate management** (housing)
  - **Inadequate or inappropriate livestock policies** with respect to credit, extension, marketing and infrastructure

MAJOR GAPS

1. Diseases
2. Poor nutrition
3. Un improved genetic base
4. Poor product handling and processing
5. Socio economics and market information
6. Technology transfer
7. Lack of understanding its impact even by donors, RED-FS and CAADP.

1.3. General impacts of livestock diseases in Ethiopia

- **Loss of productivity** through **reduced feed intake** & reduced efficiency of feed utilization

- **Slow growth rates** and **poor condition** at slaughter:

- **Loss of livestock through mortality:**

Annual direct losses due to mortality for cattle: 8-10 percent of the national herd

Annual direct losses from mortality
Sheep: 14-16 %
Goat: 11-18 %

- **Treatment** and **professional service cost**

- **Reduce/lack of draught power**, thereby influence crop production

- **Public health hazards** (**zoonotic diseases**)

1.4. General concepts of animal diseases

Classification of animal diseases:

- **Disease may be classified in various ways:-**

(1). According to mode of Origin/Genesis

(a).Hereditary Disease: In the broad sense, any disease **transmitted** from **parents (sire or dam) to offspring,**

- **In the more restricted sense**, a disease is only **regarded** as hereditary if it is **directly transmitted to the ovum or sperm** (genetic disease)

E.g.:- **Hairlessness** (**hypotrichosis**), **Parrot mouth** (**brachygnathia inferior**), **Mulefoot** (**syndactyly**)etc

Figure 6. Parrot mouth (brachygnathia inferior).

Figure 3.Mulefoot (syndactyly).

The two toes are **fused together** to produce only one toe

(b). Congenital Disease: A congenital disease must be distinguished from a hereditary one,

- Diseases which are acquired during intra-uterine life of an individual and are appreciable at birth (i.e. **It is really a disease** which is **acquired in utero** and it is **appreciable at birth**)

(c). Acquired disease: An acquired disease is **neither** hereditary **nor** congenital and most frequently develops after birth

(2). According to system involved

(a). **Local Disease:** Diseases **confined** to a particular spot or **organ/part**

E.g. Glossitis, localized abscess

(b). **Generalized disease:** Diseases, which **affect most** or **all parts of a body** (**systematic disturbance**)

E.g. **Septicemia:** invasion of bloodstream by virulent microorganisms. Acute illness caused by infectious agents or their products circulating in the bloodstream

Toxaemia: Blood poisoning caused by bacterial toxic substances in the blood, Toxin circulating in the bloodstream

(3). According to Cause of the disease:

(a). Infectious diseases: Caused by **living microorganisms** (called **pathogens/infectious agents**) that invade the animal's body

Classification of infectious agents:

- Bacteria – survive on appropriate media, stain gram-positive or -negative
- Viruses – obbligate intracellular parasites which only replicate intracellularly (DNA, RNA)
- Fungi – non-motile filamentous, branching strands of connected cells
- Metazoa – multicellular animals (e.g.parasites) with complicated life cycles often involving several hosts
- Protozoa – single cell organisms with a well-defined nucleus

(b).Noninfectious Diseases: Not caused by living microorganisms

- **It is caused by Injuries, Poisons/chemicals, Poor nutrition, Birth defects** and etc

(4). According to Clinical Manifestation:

(a). **Acute disease:** Such type of disease is **characterized by a sudden onset** & comparatively short courses with **sever manifestation** and that runs its course quickly

- Generally illness may prevail for **3-14 days** (**usually sooner**), and recovery in those who survive is usually complete

- Has Identifiable beginning and **end**

E.g. FMD

(b). **Per-acute Disease:** Duration of disease is **shorter than acute disease** illness and lasts for **few hours to 48 hours**

E.g. **Anthrax**

(c). Sub-acute disease: Diseases whose onset & severity is lesser than acute and usually it has a course of 2-4 weeks

E.g. Sub-acute mastitis

(d). Chronic disease: Diseases which has got a protracted course of illness (**symptoms**), which **lasts 1 or two months** or **even more** and **in some cases for the remainder** of the animal's life

- **Recovery is slow** and sometimes incomplete
- **Symptoms develop over** time
- **Continue for rest of life** of animal

E.g. Tuberculosis, **Paratubercullosis**

(5). According to the Intensity & Spread of Disease

(a). Endemic disease: It refers to **the constant presence** of a disease or infectious agent within a given geographic area or population group

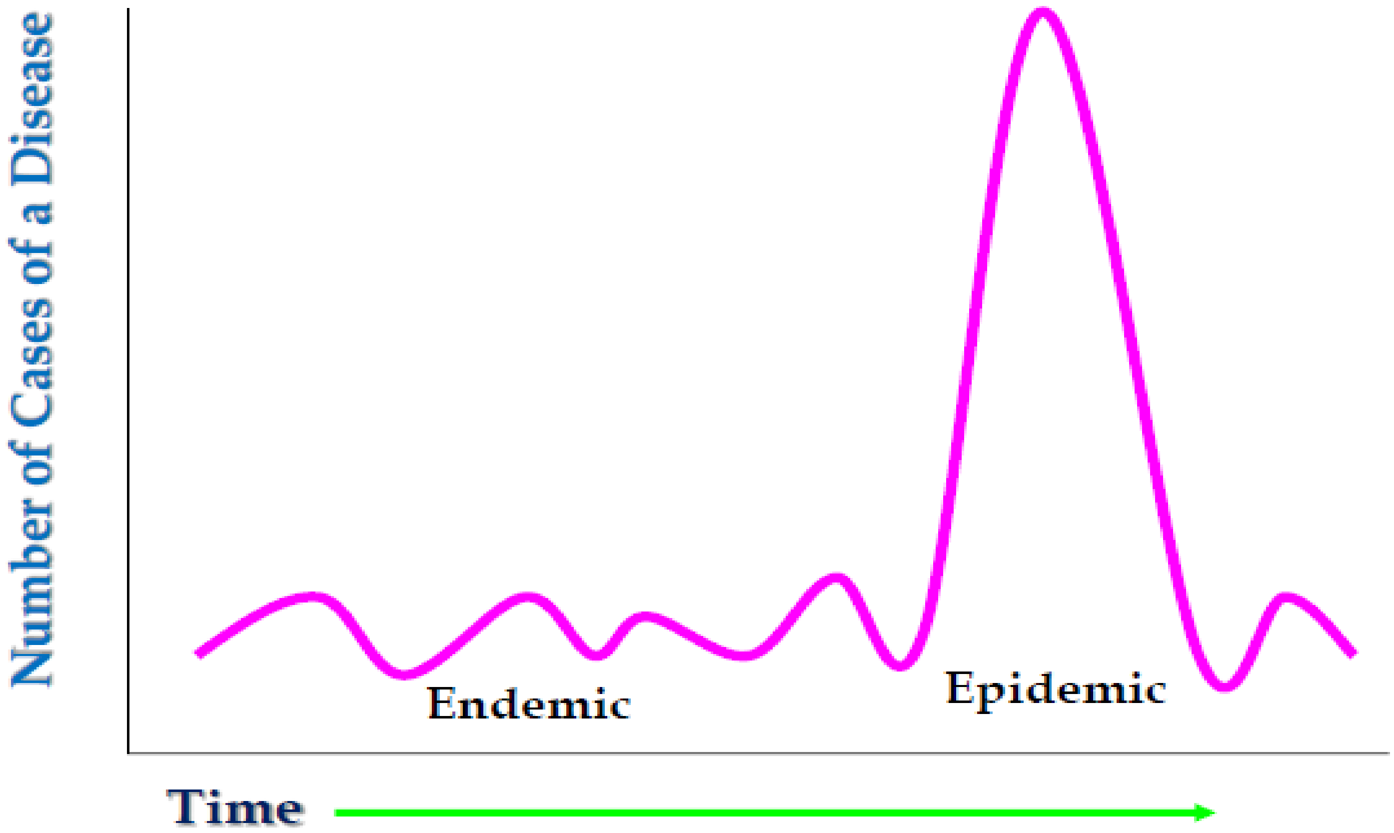
- It is the usual or expected frequency of disease within a population (i.e. usually present; steady prevalence)
- (En = in; demos = people)

E.g. Blackleg

(b). Epidemic: Diseases which affect large population of animals in large area & the disease spreads quickly (rapid spread) & is capable of covering a wide area

- The unusual occurrence of disease (in excess of expected occurrence)
- (**Epi**= upon; **demos**= people)

Endemic vs Epidemic



(c). Sporadic disease: those **diseases occur occasionally** in animal population

- **Isolated incident** in a single animal
- The word sporadic means "**scattered about**" i.e. the cases occur **irregularly**
- **However, a sporadic disease could be the starting point** of **an epidemic** when the conditions are favorable for its spread

(d). Pandemic: When the **epidemic reaches** usually large size in some country **or spreads over many countries** or even **continents**,

- **When epidemics occur at several continents** – global epidemic

E.g. Influenza

- **Occurring across countries** and **in multiple** populations

1.5. Routes of Disease Transmission

- Disease causing agents (or **pathogens**) can be **spread** from **animal-to-animal** or **animal-to-human** through **a variety** of **transmission routes**

- There are **five main routes** of disease transmission: (**aerosol**, **Oral**, **direct contact**, **fomite** and **vector)**

(1). Aerosol (Inhalation) Transmission:

- **Droplets containing pathogens** travel through the air and are **inhaled by another animal** or human (i.e. Pathogenic agents contained in aerosol droplets are **passed** from one animal to another, or between animals and humans)

- **Most pathogenic agents do not survive** for **extended periods** of time within the **aerosol droplets** **and close proximity of infected** and susceptible animals is **required** for transmission

Aerosol Transmission

- Disease agents contained in droplets
  - Pass through air
- Most agents not stable in droplets
- Close proximity required
  - Enclosed barns
  - Coughing, sneezing
  - Contaminated soil
    - Birthing tissues
    - Feces, urine

(2). Oral (Ingestion) Transmission

- Oral transmission of disease causing organisms **involves ingestion through the consumption of contaminated feed or water**, or **by licking/chewing on contaminated environmental** objects

- **Feed and water contaminated** with **feces**, **urine** or **saliva** are **frequently** the cause of **oral transmission of disease agents**

- **Fecal-oral transmission of diseases** is a **common means** of infection in animals (and people)

- **Shared feed and water sources** can contribute to the **spread of the disease**

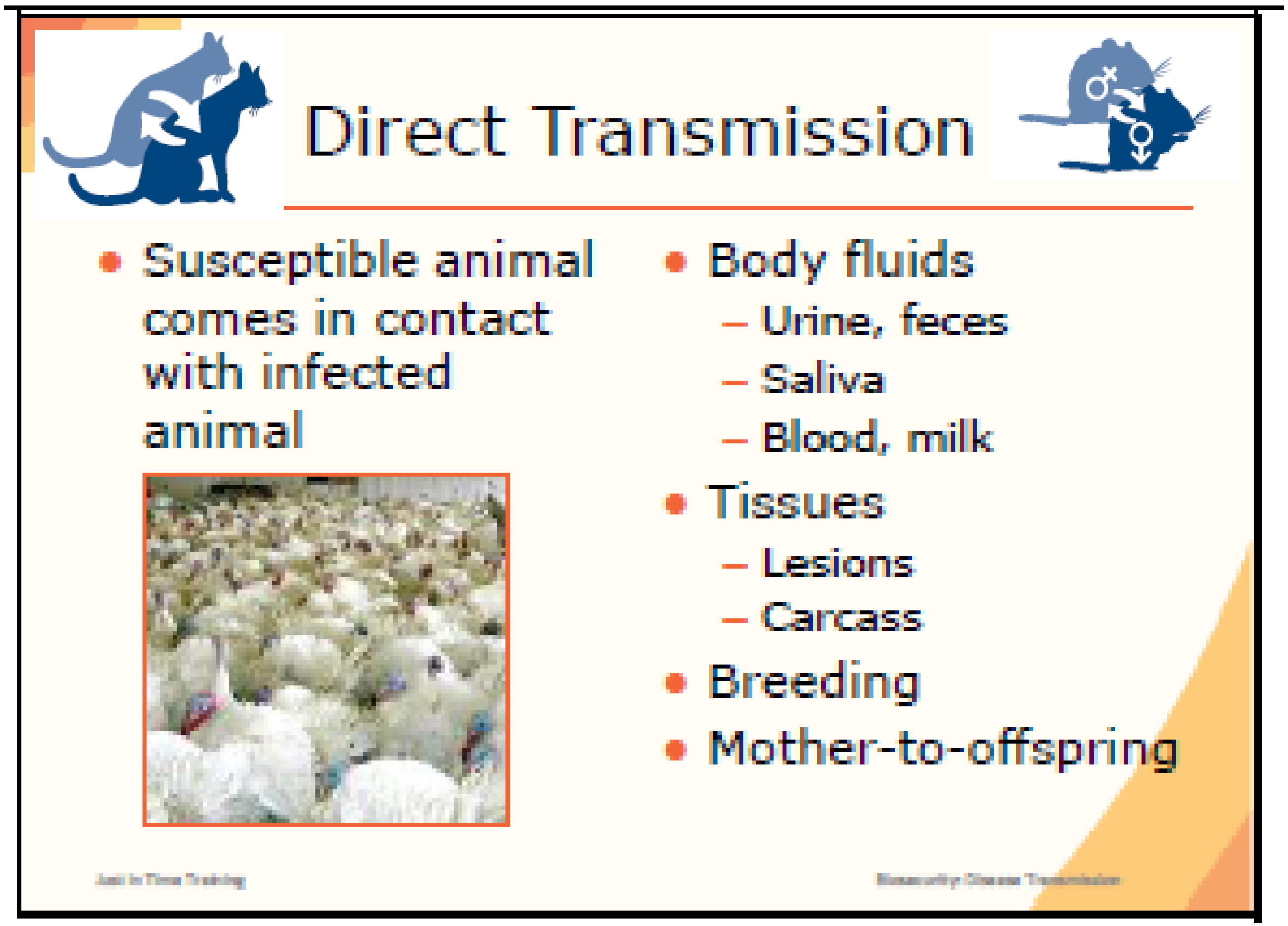
- **Contaminated environmental objects** could include equipment, **feed bunks**, **water troughs**, **fencing**, **salt** and **mineral blocks**, and other items an animal may lick or chew


Oral Transmission
Ingestion of contaminated feed or water
– Feces, urine
– Saliva
– Milk
Licking/chewing contaminated environment
Shared feed or water sources

(3). Direct Transmission (Direct contact)

- Direct contact is **one of the main methods of disease spread** between animals
- It occurs when a susceptible animal comes in **direct contact** with **an infected animal**, **its body fluids** or **tissues** (i.e. **Spread of pathogens through contact with open wounds**, **mucous membranes** (such as the eyes, nose, or mouth), or **abraded skin** (open wounds) **contacting an infected animal** or its tissues or fluids (e.g., blood, saliva, urine)
- So, **possible exposures can occur** from **nose-to-nose contact**, **biting or rubbing against** each other
- It may also be spread through contact with infected animal **lesions or tissues**

- **A subtype of direct contact** **involves** **Reproductive Diseases that** spread through **venereal contact** (from **animal-to animal through coitus**) and **in-utero** (**from dam to offspring** during **gestation**)



(4). Fomite Transmission

- **Indirect transmission** may occur **by fomites** (i.e. A contaminated **inanimate object transmits a disease agent** from one susceptible animal to another)

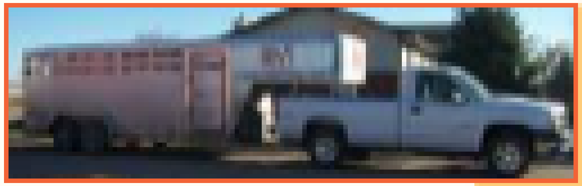
  - **Fomites** are **inanimate objects**, such as **equipment**, clothing, footwear or **vehicles**, that can transfer microorganisms from an infected animal to another animal or person)

  - **Examples of fomites** that may be present during a response include **needles**, balling guns (used to dispense medication to cattle), **feed or water buckets** & bedding

- Even items such as clothing or vehicles may become contaminated and **serve to spread pathogens**

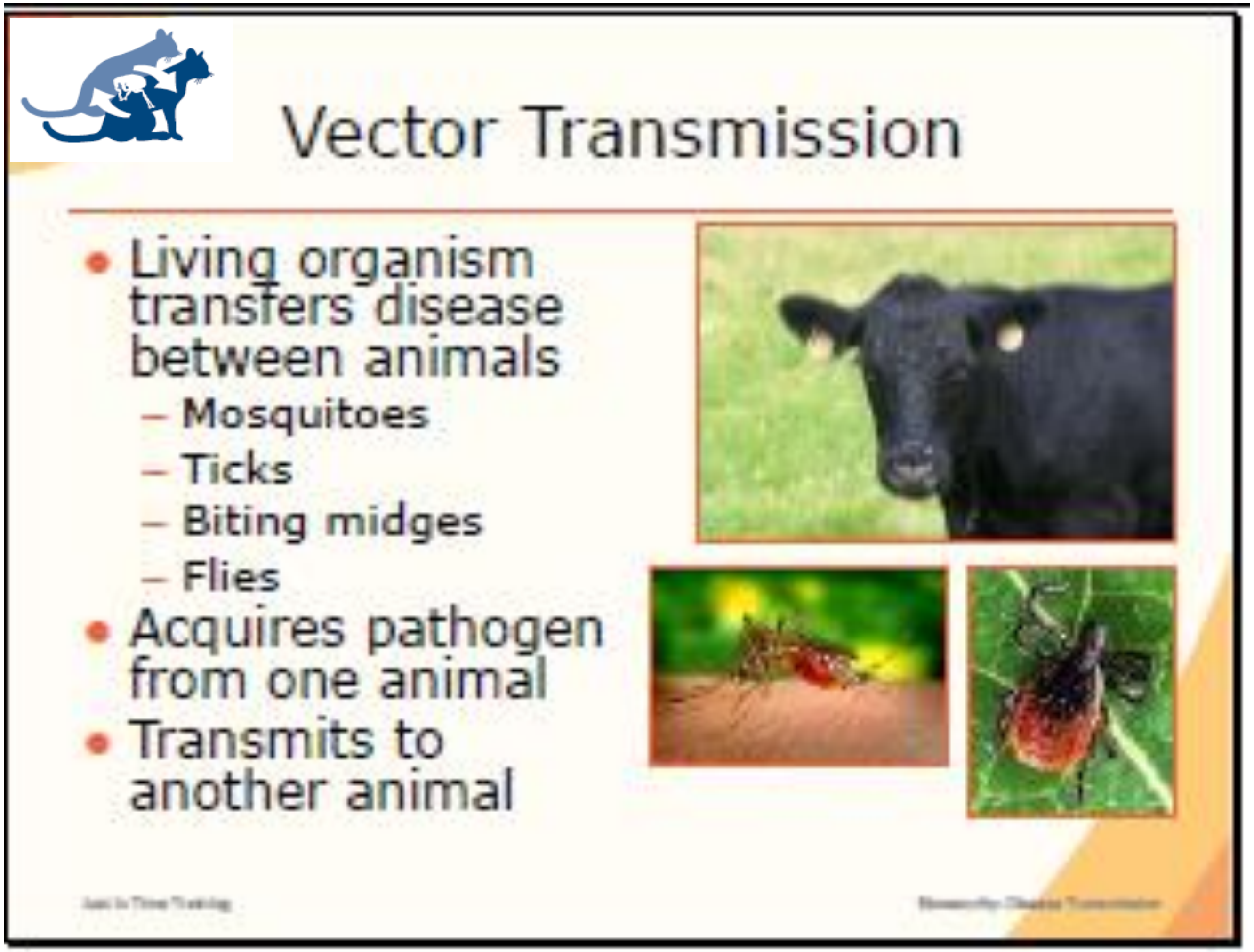
Fomite Transmission

- Contaminated inanimate object
- Carries pathogens to other animals
  - Needles, balling guns
  - Buckets
  - Bedding, shovels
  - Vehicles, trailers
  - Humans, clothing



(5). Vector Transmission

- **An insect acquires a pathogen** from one animal and **transmits it** to another **either mechanically** or **biologically** (**i.e.** Transfer of a **pathogen from an infected animal** to another animal or a human **by an insect** (e.g., flea, tick, mosquito))
  - **Mechanical transmission:** disease agent **does not replicate** or **develop in**/on the vector; it is simply transported by the vector from one animal to another (e.g., flies)
  - **Biological transmission:** vector takes up the agent, usually through a blood meal from an infected animal, **replicates and/or develops it**, and then **regurgitates the pathogen onto or injects** it into a susceptible animal
  - Fleas, ticks, and mosquitoes are common biological vectors of disease
- Sometimes **rodents** or **birds** can serve as disease vectors

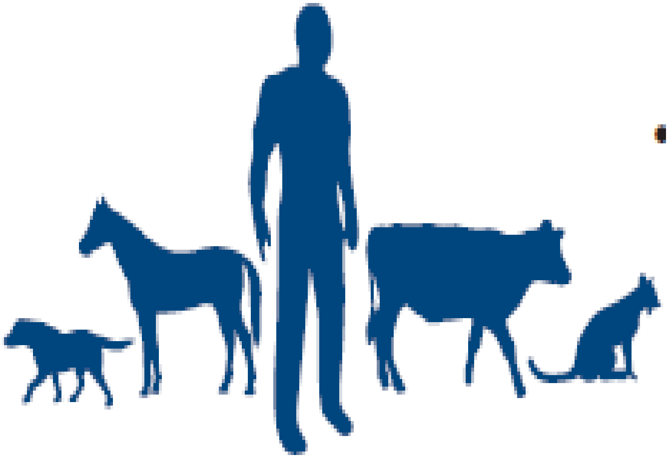

Vector Transmission
Living organism transfers disease between animals
– Mosquitoes
– Ticks
– Biting midges
– Flies
Acquires pathogen from one animal
Transmits to another animal

Zoonotic (Zoonoses):

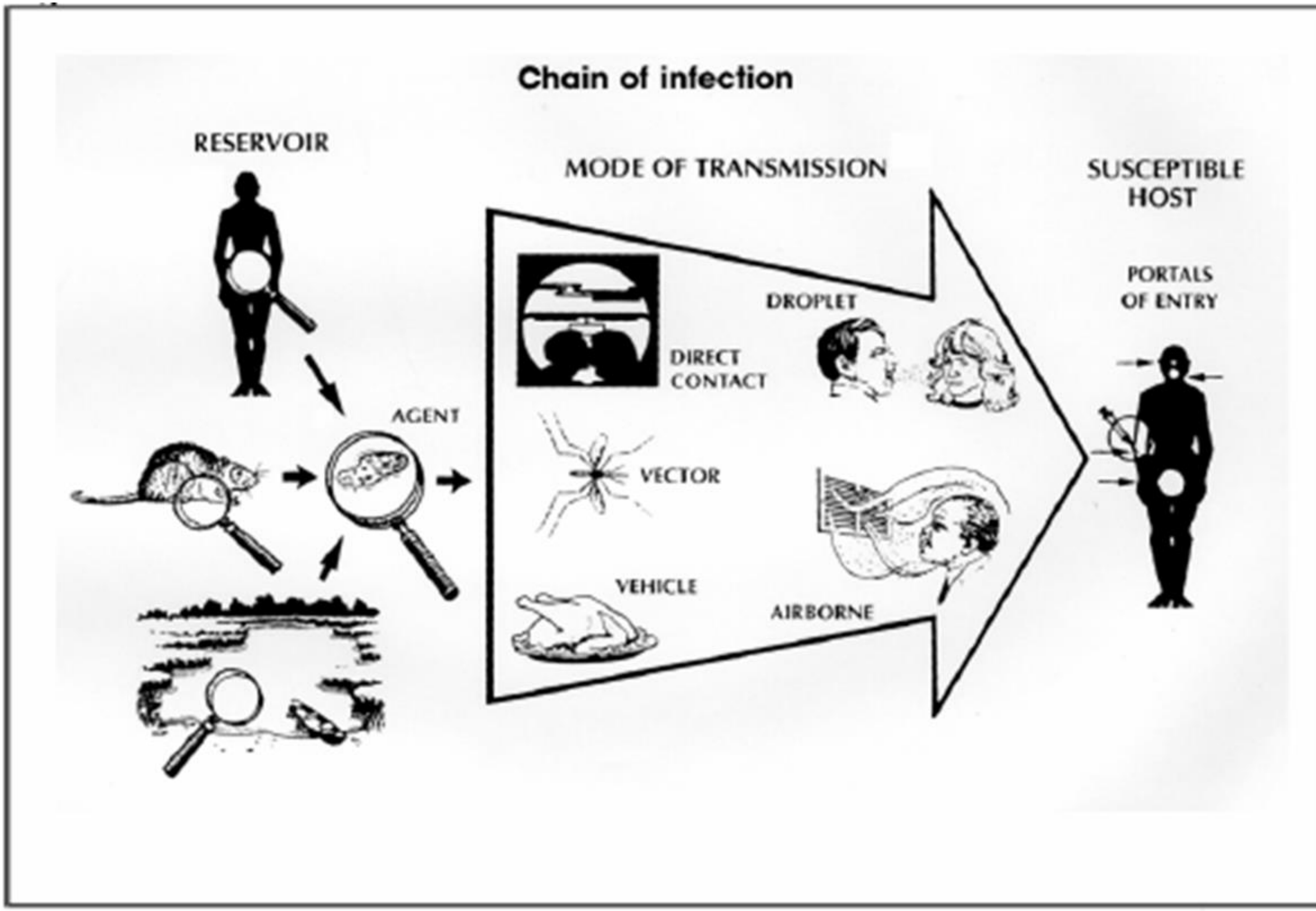
- Diseases transmitted **between animals** and **humans** (**i.e**. Diseases **spread between animals** and **humans**)

- **Human exposure occurs through** one of the **previously listed five main routes of transmission** (aerosol, direct contact, fomite, oral, and vector-borne)

- **It is a separate route** of transmission due to its importance



Disease Transmission (Summary):

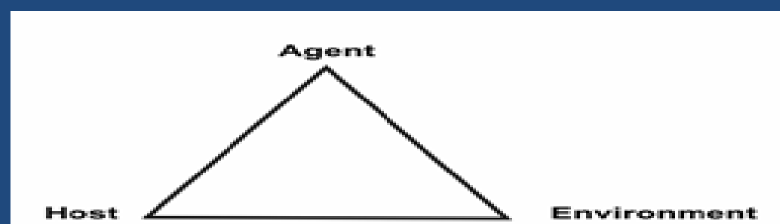


1.6. Some Terminologies

Disease: Impairment of normal functioning, manifested by signs and symptoms

Infection: Is the **invasion** of living organism (**host**) by **another organism** (**disease-causing agent**), their multiplication, and the reaction of host tissues to the infectious agents and the toxins they produce

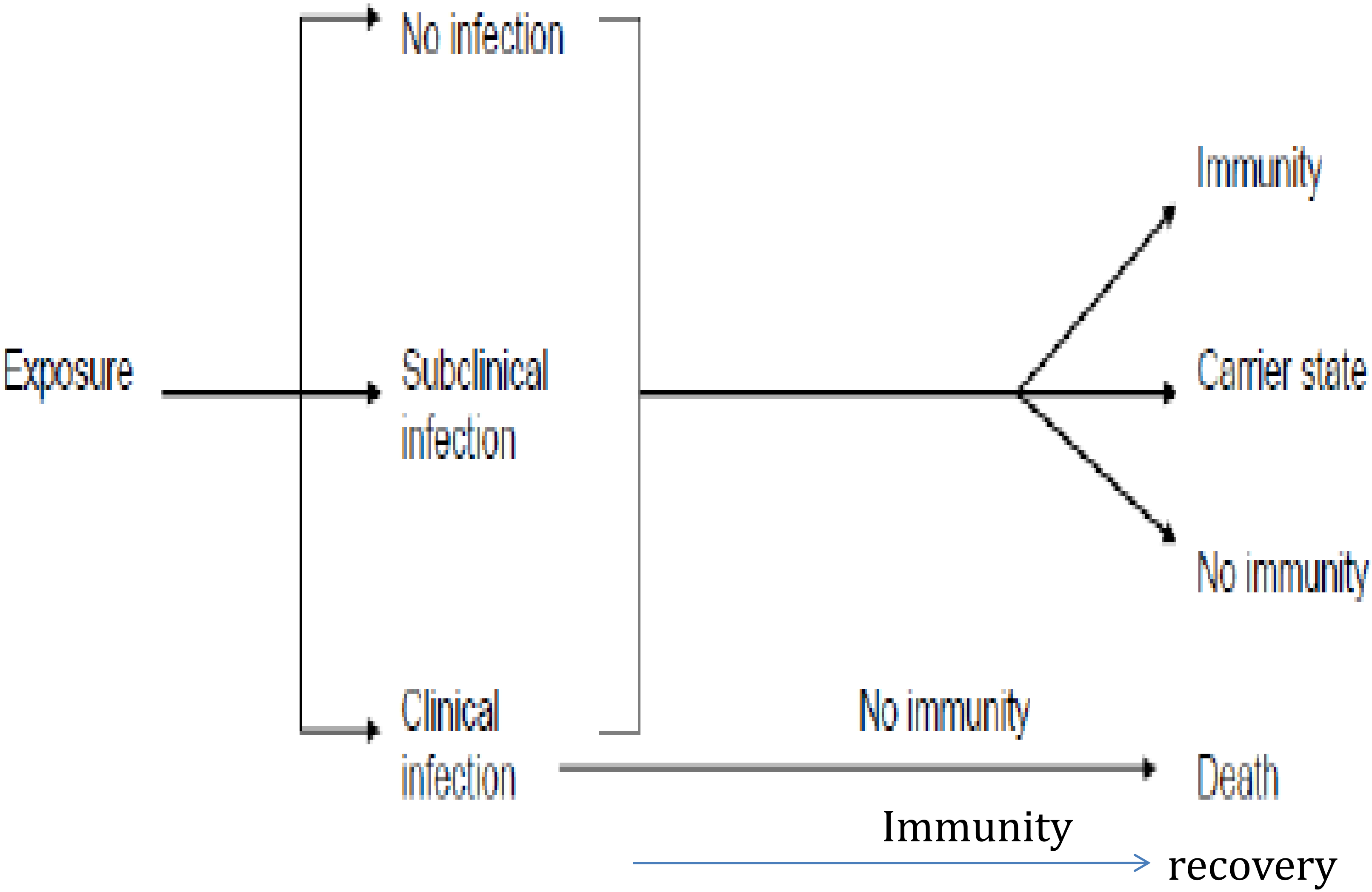
Disease Causation



HOST Characteristics	Types of AGENTS	ENVIRONMENTAL Factors
Age	Biologic – bacteria, virus, molds	Housing – crowding, noise
Gender	Chemical – poison, alcohol	Air – temperature, humidity
Occupation	Physical - trauma, fire	Water , food
Race		
Marital status		
Genetic profile		

Interaction between the agent and the susceptible host **in an environment** that supports transmission of the agent → DISEASE

The different outcomes of an exposure to an infectious agent



Infectivity: The ability of disease-causing agent to infect a host

Pathogenicity: The ability of disease-causing agent to cause disease in the host

Virulence: The ability of disease-causing agent to cause severe disease in the host

Immunogenicity: The ability of disease-causing agent to induce an immune response in the host

Inapparent infection: No clinical symptoms generated

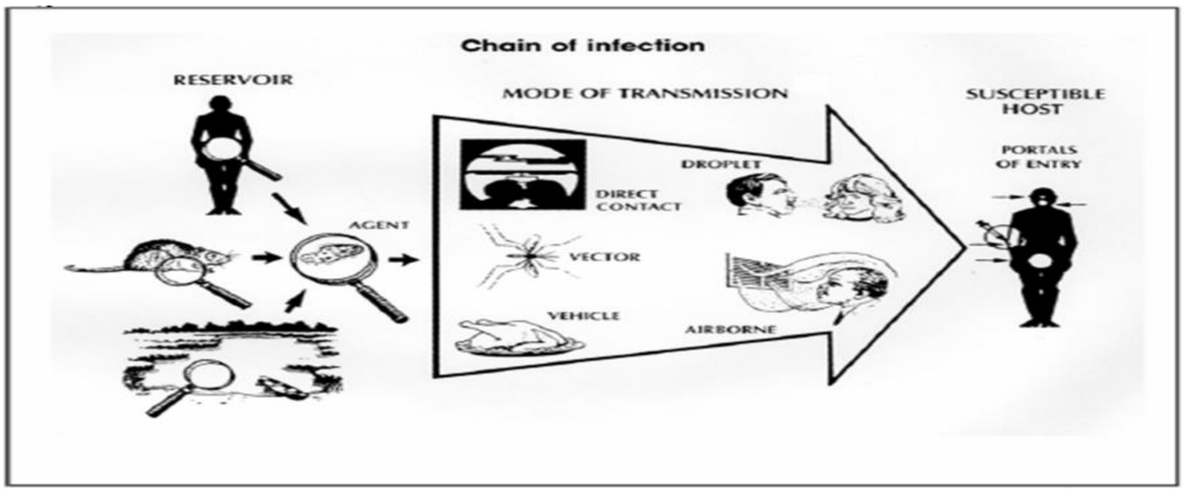
Carrier state: Usually no clinical symptoms but host can transmit infection for long periods

Carrier: Individual harbors the agent but does not have symptoms (can infect others)

Reservoir: Habitat (**man**, **animal**, etc) in which the agent normally lives, grows, and multiplies

A reservoir is a population that the disease propagates in

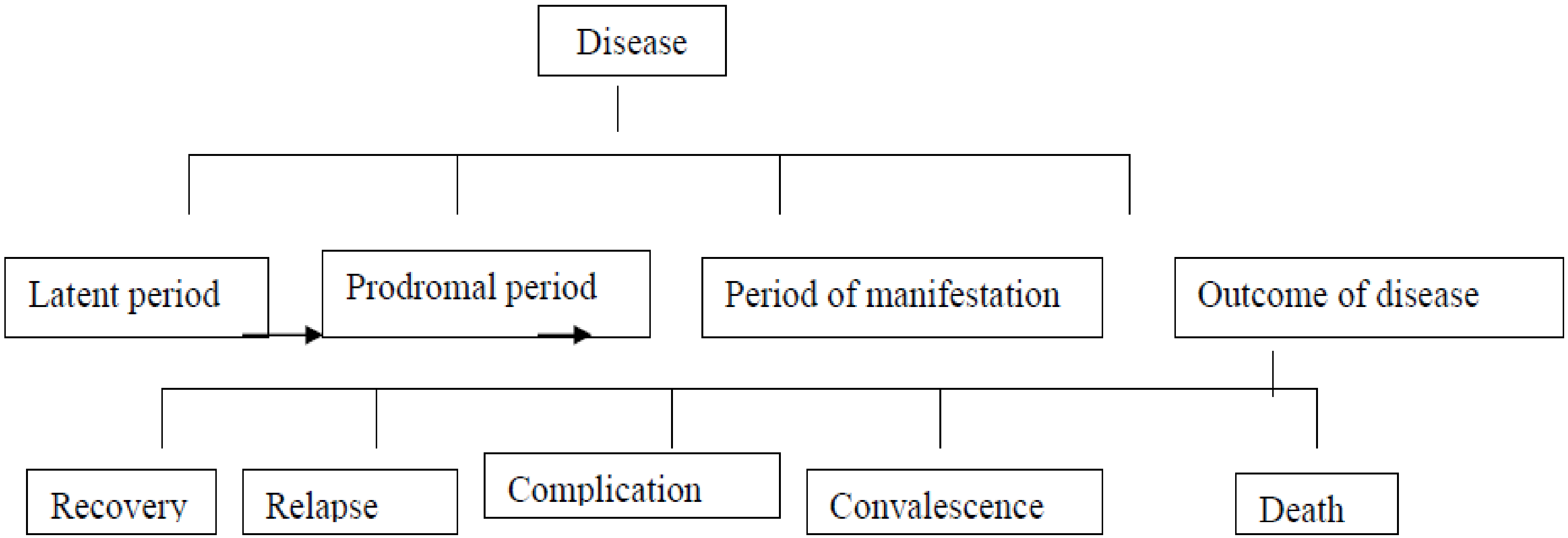
A carrier is an individual who is contagious but otherwise not symptomatic or obviously so



Vehicle: inanimate object (food, water, etc) that can carry an agent from one organism to another

Vector: an organism (as an insect) that transmits an agent from one organism or source to another

- **Period and course of disease (i.e. The four major stages of a disease):**



- **Incubation period:** The period (time) elapsed between exposure to a pathogen, chemical, or radiation, and onset (manifestation) of clinical symptoms of disease
    - **i.e.** It is the time from the **first contact** (infectious agent enters the host's body) to the time when the host shows first signs and symptoms of the disease
    - It ranges from **several minutes** to **several months**

- **Prodromal period:** is one of the four major stages of a disease
    - The first stage is referred to as **incubation period** followed by a prodromal period
- **Prodromal period:** lasts from discovery of the first sign (incubation period) of the disease to its complete manifestations (period of manifestation)
    - **At prodromal period**, the symptoms may not be very specific or sever
    - E.g. Manifestations such as **depression**, in appetence, rise in temperature etc (general clinical signs)

- **Period of manifestation: i.e. The peak (clinical) stage:** It follows prodromal period, **at this stage** the disease **reaches its highest point** of development
  - **It is a period of marked development** of **Clinical manifestations**
  - The **manifestation helps the clinician** to **diagnose** the **disease** (specific clinical signs)

Outcome of the disease (the 4th stage of the disease):

(1):Recovery: restoration of body function, the pathogen has been mostly eliminated, **which can be either:**

- **Complete recovery**
- **Incomplete**

(2): Convalescence: Is **the gradual recovery of health** and **strength** after illness or injury

- It refers to the **later stage of an infectious disease** or illness **when the patient recovers** and **returns to pervious health**, **but** may continue to be a source of infection **to others even if feeling better** (this is called **Convalescent Carrier**)

Convalescent Carrier: An individual who is fully cured of a particular disease but is still capable of transmitting the disease to others

Whereas Incubatory Carriers: An individual who is capable of transmitting a disease causing agent to others during the incubation period of the disease

(3). Relapse: means **recurrence** of the disease **after clinical recovery** during period of convalescence

- It may be due to the **break in the immune competency of the host** as a result of exposure, exertion & errors in the diet

(4). Complications: in some cases, primary disease causes a weakness of the body, which then becomes susceptible to other diseases called secondary **complication**

(5). Death: if the animal is **unable to adjust the altered conditions of existence**, **its adaptation mechanism become exhausted** and unable to continue life

Types:

- **Local death:** death of part of the body only (necrosis)
- **Somatic death:** death of entire body

Thank You!!

Chapter Two

Principles of Disease Treatment, Control, Prevention & Eradication

2.1. Principles of Treatment /Therapeutic Measure

- Treatment depends on **general care** & **nursing** which are **very important** for **rapid recovery** of **diseased animal**

Treatment may be classified as: (I) General treatment & **(II)** Specific treatment

(I). General treatment: Include measures to **be taken to combat certain complications** during the course of disease & to resuscitate (save) the **vitality** of the animal

- **General treatment includes**:

(a). Fluid and **electrolyte therapy-** for dehydration (**i.e. to treat dehydration**)

E.g. **Dextrose** with NaCl

(b). Mechanical treatment

E.g. Exercise, massage, etc

(c). Physical treatment

E.g. Use of **heat**, **electricity**, **x-rays** etc

(d). Dietetic treatment: is therapeutic nutrition

(II). Specific treatment: is administration of **specific curative drugs** against certain diseases

- **Only possible** when the **specific etiology of disease is diagnosed**

E.g. Ca-therapy for **milk fever**

-Specific anthelmintic drugs for helminthiasis
-Antibacterial for bacterial infection

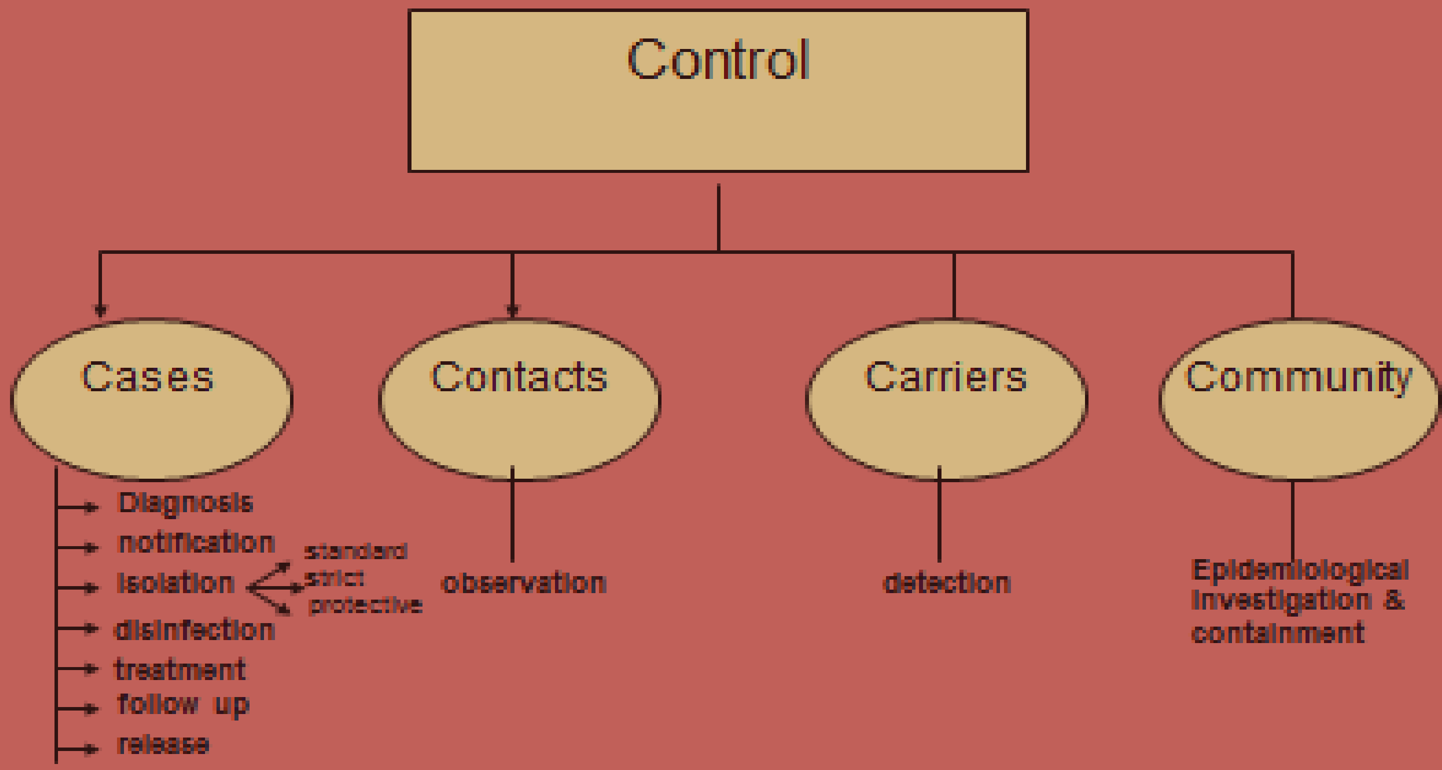
2.2. Principles of Disease Control

- The **term disease control** describes ongoing operations **(i.e. control requires ongoing intervention)** aimed at reducing:-
  - The **incidence** of disease
  - The **prevalence** of disease
  - The **effect of infection i.e. morbidity** & **mortality** of an infectious disease to **a locally acceptable level**
- Control is local and so needs to be looked at from the local perspective,
  - i.e. The level of control that is optimal for one country (region) may not be optimal from the perspective of the world as a whole,
- **Thus, a need exists** to distinguish between, say, a locally optimal level of control and one that is globally optimal

- **Effective control of infectious diseases** is dependent on **good knowledge of etiology** & **epidemiological characteristic** (distribution in age, mortality & morbidity rate, seasonality of occurrence etc)

- **The ultimate achievement of control** is **eradication** **but** not every disease that can be controlled **can be eradicated**

Control of infectious diseases (the 4 "C"s



Methods of Disease Control (i.e. Disease control measures)

(a). Quarantine:

- Quarantine is segregation of apparently healthy animals (brought into the herd for 1st time) which have been exposed to risk of infection from healthy & unexposed animals
    - i.e. When someone has been exposed to a contagious disease and it is not yet known if they have caught it, they may be quarantined or separated from others who have not been exposed to the disease (it is used to separate & restrict the movement of well individual who may have been exposed to a communicable disease to see if they become ill)
- **Quarantine period depends** on incubation period of the disease (usually 30-60 days), it could be up to 6months sometimes
- Objective of quarantine is to prevent the introduction of new diseases into the farm together with the new entrant animals

(b). Isolation:

- **Isolation is used to separate ill (sick animals)** individuals who have a communicable disease from those who are healthy
    - i.e. When someone is known to be ill with a contagious disease, they are placed in isolation and receive special care, with precautions taken to protect uninfected population from exposure to the disease
- **Isolation restricts the movement of sick animals** to help stop the spread of certain disease

(C). Treatment of sick animals: To prevent suffering, death & economic losses

(d). Vaccination:

Vaccination is the administration of a vaccine to help the immune system develop protection from a disease

- i.e. Artificially building up animal body immunity against specific infectious diseases by injecting biological agents called vaccine (this means in stimulating the body's adaptive immunity, the vaccine help to prevent sickness from an infectious disease)

- The vaccine stimulates the immune system so that it can recognize the disease and protect the animals from the future infection (i.e. the animal become immune to the infection)

- **Vaccines contain a microorganism** in **a weakened** (Attenuated/live vaccine) or inactivated (killed vaccine), or proteins or toxins from the organism

(e). Depopulation (if economy allows): Total elimination of animals from the herd **whenever the disease is chronic**, not treatable & fast spreading

(f). Elimination of carriers: In some diseases the carrier state may remain for years & the animal become a potential danger to susceptible animals

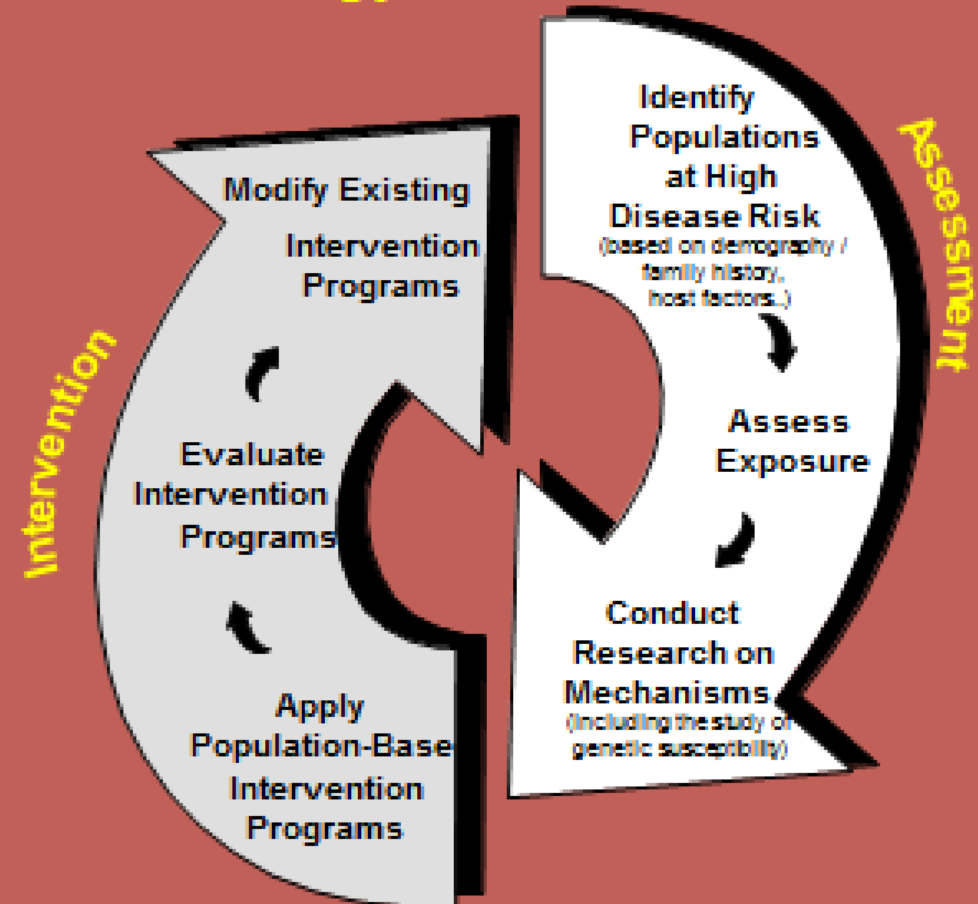
E. g. Tuberculosis, brucellosis

(g). Sanitation: It refers to the maintenance of hygienic conditions, through services such as garbage collection, waste disposal, disinfection & sterilization

2.3. Principles of Disease Prevention

- It refers to actions aimed at avoiding the manifestation of a disease
    - i.e. Disease prevention is a procedure through which individuals, particularly those with risk factor for a disease are treated in order to prevent/protect a disease from occurring
- **The goals of medicine** are to promote health, to preserve health, to restore health when it is impaired, and to minimize suffering and distress
    - **These goals are** embodied in the word "**prevention**"
- **Successful prevention depends on:**
    - A knowledge of causation
    - Dynamics of transmission
    - Identification of risk factor & risk groups
    - Availability of vaccines/prophylactic antibiotics or early detection & treatment measures

Strategy for Prevention



Epidemiology Division

2.4. Principles of Disease Eradication

- The term **denotes the certified total absence a disease/cases**, the absence of a reservoir for the organism in nature, and absolute containment of any infectious source

- **Eradication is literally means** to "**tear out by roots**"

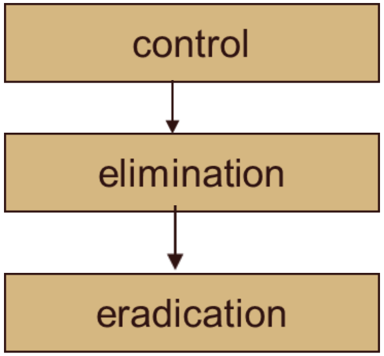
- **Eradication** is an absolute process, an "all or none" phenomena, restricted to termination of an infection from the whole world, and this implies that disease will no longer occur in a population

- Eradication differs from control in that it is global

2.5. Principles of Disease Elimination

What is the difference between Disease elimination & Eradication??

- **Between** control & eradication, an intermediate goal has been described, called regional elimination



- The term "**elimination**" is used to describe interruption of transmission of disease

- This term to denote the cessation of transmission of an organism throughout a country or region (i.e. regional elimination is now seen as an important precursor of eradication)

- Like control, elimination is location-specific and would require ongoing interventions to be sustained in order to **prevent reemergence of the disease** from microbe importations


Thank you
|26/12/2013|
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Chapter Three

Infectious Disease

Introduction to Infectious Disease

- **According to the cause**, **disease is broadly** divided in to **infectious** and **non-infectious** diseases

- The **infectious diseases** are of **major importance** in farm (**agricultural**) animals

- **Infectious diseases** are caused by **microorganisms** (**bacteria**, **virus**, **fungus**, **protozoal parasites**) and **account for a major economic losses** in the livestock sector

- The **infectious diseases** are capable of affecting many animals **in a short period of time** and the case fatality rate in some diseases can be **very high** and **the economic losses** may be very large

- **Certain infectious diseases**, especially the viral diseases, are **endemic in some countries** and **pose a threat** to other countries considered to be free of them

- The **clinical** and **laboratory diagnosis** of the **infectious diseases** can be difficult

- **However, with the appropriate laboratory support** and **suitable samples**, most of them can be **diagnosed definitively**

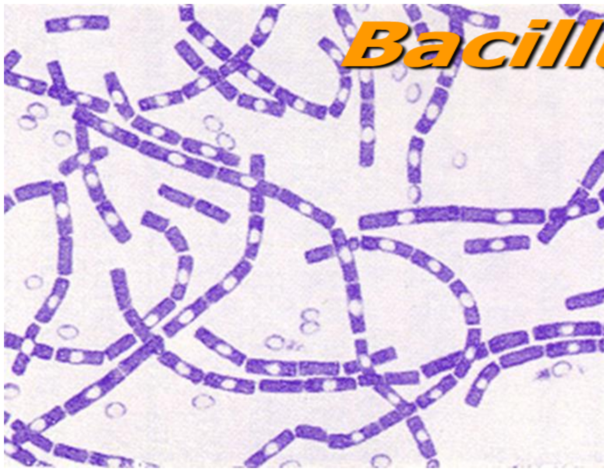
- For each disease **certain samples must be submitted** to the **laboratory** for **isolation or demonstration of the specific pathogen**

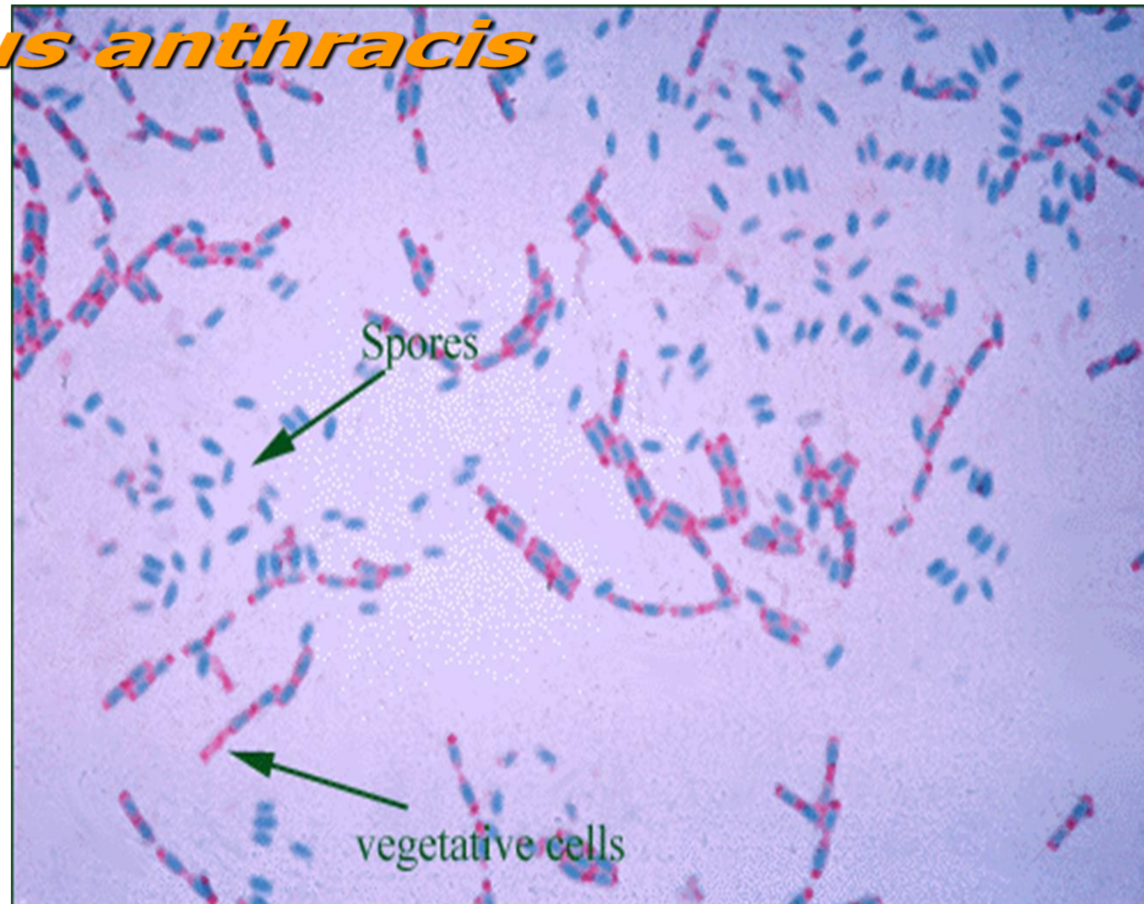
- **Clinical** and **epidemiological findings** will usually result in **a tentative diagnosis** and a rule-out list of possible **diagnoses**

3.1. Bacterial diseases

3.1.1. Anthrax

Bacillus anthracis





3.1.1. Anthrax

- **Anthrax** is **a per-acute** or **an acute** infectious disease (septicemic disease) disease of almost all warm-blooded animals including humans (i.e. **it is a zoonotic disease**)

  - i.e. Cattle, sheep, goats (ruminants are at greatest risk), horse, donkey, pigs, etc can be affected by Anthrax, however, carnivores are more resistant and birds are rarely affected

- **Anthrax is caused** by the **spore-forming bacteria** *Bacillus anthracis*

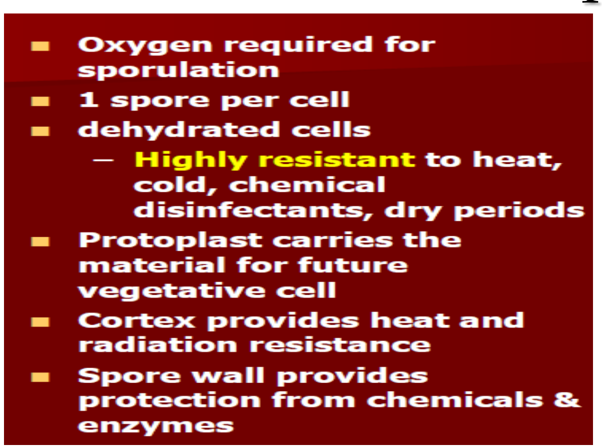
In animals (particularly cattle and sheep), **Anthrax** is characterized, in most instances, by **sudden death** and oozing of unclotted tarry blood from the external natural orifices

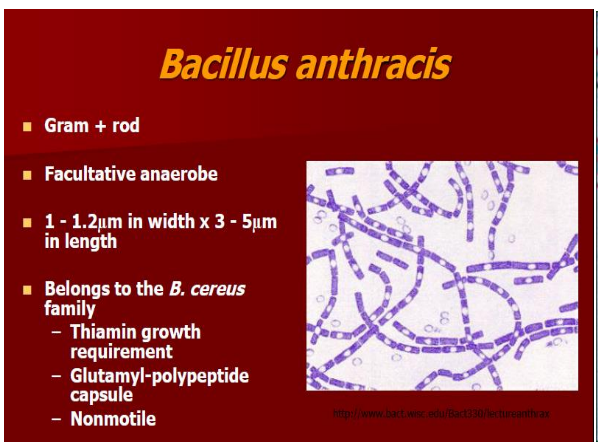
3.1.1.1. Etiology

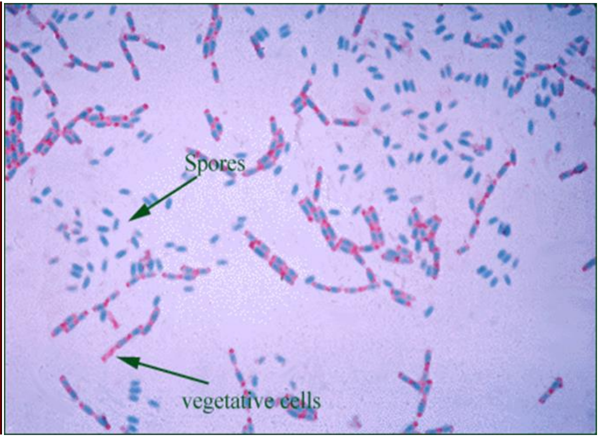
- *Bacillus anthracis* is the specific cause of the disease and pathogenic strains have plasmid-encoded virulent factors: a **poly-D-glutamic acid capsule**, which aids in resistance to phagocytosis

- The organism forms spores that persist in the environment for decades (i.e. can persist in the soil in warm moist climate for long periods)

- i.e. the spores are resistant to most external influences, normal environmental temperatures and standard disinfectants)







3.1.1.2. Epidemiology

- Anthrax **occurs all over the world** (i.e. it has spread to have a **worldwide distribution**), although the area prevalence varies with the **soil**, the **climate (warm)** and the efforts put into **suppressing** its occurrence

- Outbreaks originating from a soil-borne infection always occur after a major climate change, for example heavy rain after a prolonged drought, or dry summer months after prolonged rain, and always in warm weather when the environmental temperature is over 15°C

    - i.e. Anthrax outbreak occurs irregularly and is commonly associated with neutral or alkaline soils where the spores revert to the vegetative form and multiply to infectious levels if environmental conditions of **soil, moisture, temperature,** and **nutrition are optimal**

- Many sudden deaths occur without observed illness, in areas that have recently had appropriate climatic conditions and in which the disease has occurred as long ago as 30 years previously

- Spread of the organism within an area may be accomplished by streams, **insects**, dogs, feral pigs, and other carnivores, and by fecal contamination from infected animals

- Avian scavengers such as gulls, and vultures can carry spores over considerable distances

- Infected wildlife are also a source for domestic animals on common grazing lands

3.1.1.3. Transmission of the infection

- **Infection gains entrance to the body** by **ingestion**, **inhalation**, or **through the skin**

Common routes of anthrax infection are:

  - **Cutaneous**: Enters animal body through a cut **on skin**
  - Gastrointestinal: By eating undercooked meat from an infected animal
  - **Inhalation:** While breath in anthrax spores

- it is generally considered that most animals are infected by the ingestion of contaminated food or water

- The increased incidence of the disease on sparse pasture is probably due both to the ingestion of contaminated soil and to injury to the oral mucosa facilitating invasion by the organism

- **Inhalation infection** is thought to be of minor importance in animals, although the possibility of infection through contaminated dust must always be considered
- 'Woolsorter's disease' in **humans** is **due to the inhalation of anthrax spores** by workers in the wool and hair industries, but even in these industries cutaneous anthrax is much more common

Cutaneous anthrax has occurred in veterinarians following postmortem examination of anthrax carcasses

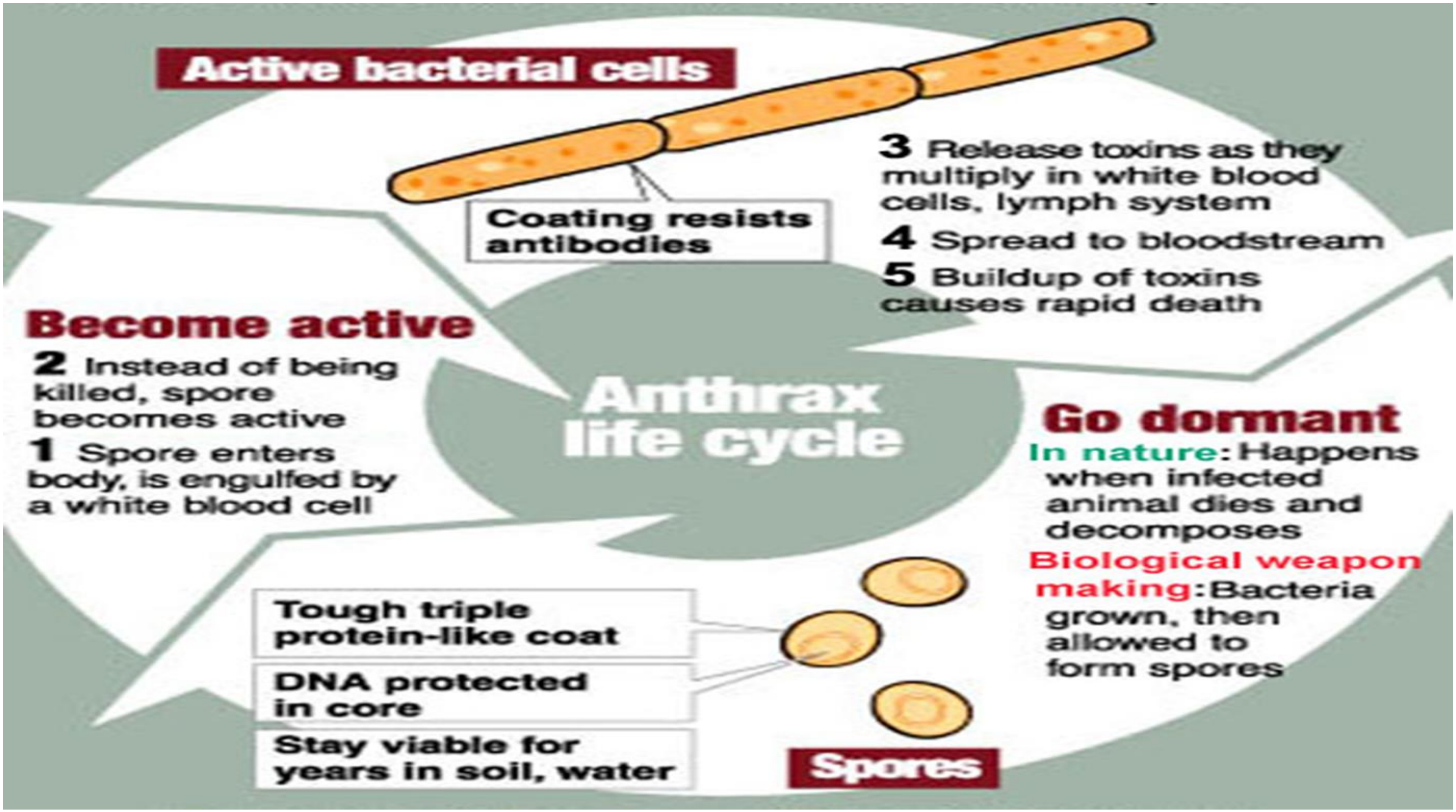
3.1.1.4. Pathogenesis

- **Upon ingestion of the spores**, infection may occur through the intact mucous membrane, through defects in the epithelium or through scratches from tough, fibrous food materials

- **The organisms are resistant to phagocytosis**, in part due to the presence of the poly-D-glutamic acid capsule, and proliferate in regional draining lymph nodes, subsequently passing via the lymphatic vessels into the bloodstream; septicemia, with massive invasion of all body tissues, follows

- *Bacillus anthracis* produces a lethal toxin that causes edema and tissue damage, death resulting from shock and acute renal failure and terminal anoxia

- In pigs, localization occurs in the lymph nodes of the throat after invasion through the upper part of the digestive tract



3.1.1.5. Clinical Signs

Signs differ by species

- The disease occurs in all vertebrates but is most common in cattle and sheep and less frequent in goats and horses
- **Humans occupy** an intermediate position between this group and the relatively resistant pigs, dogs, and cats
- In farm animals, the disease is almost invariably fatal, except in pigs, and even in this species the case fatality rate is high

Three forms of illness

(1). Peracute

- Ruminants (cattle, sheep, goats, antelope)

Peracute infection:
- Rapid onset
- Sudden death
- Bloody discharge from body orifices
- Incomplete rigor mortis
- Rapidly bloat

(2). Acute

- Ruminants and equine

Ruminants (Acute infection):

–Fever, anorexia
–Decreased rumination
–Muscle tremors
–Dyspnea
–Abortions
–Disorientation
–Bleeding from orifices
–Hemorrhages on internal organs

(3). Subacute-chronic

- Swine, dogs, cats

- Ruminants-goat (Chronic infection)

–Pharyngeal and lingual edema
–Ventral edema
–Death from asphyxiation
• Treatment successful if started early

Equine

- Anthrax in the horse is always acute but varies in its manifestations with the mode of infection
- When infection is by ingestion:
  - There is septicemia with enteritis, sever colic, high fever, death within 48-96 hours
- When infection is by insect transmission:
  - Hot, painful, edematous, subcutaneous swellings appear around the throat, lower neck, floor of the thorax and abdomen, external genitalia, death
- There is high fever and severe depression and there may be dyspnea due to swelling of the throat or colic due to intestinal irritation

- The course is usually 48-96 hours

Swine

- In pigs anthrax may be acute or subacute
- There is fever, with dullness and anorexia, and a characteristic inflammatory edema of the throat and face (localized swelling of throat)

- The swellings are hot but not painful and may cause obstruction to swallowing and respiration
- Bloodstained froth may be present at the mouth when pharyngeal involvement occurs

Dog and Cat: Relatively resistant- infection is by ingestion of contaminated raw meat

- Fever, anorexia, weakness
- Necrosis and edema of upper GI tract
- Lymphadenopathy and edema of head and neck
- Death due to asphyxiation, toxemia, septicemia

3.1.1.6. Diagnosis

- Anthrax should be suspected if an animal dies suddenly, characteristic oozing of unclotted tarry blood from the external natural orifices, rapid distension of the body (rapid blot), and etc

- Laboratory examination of stained blood smear from the ear vein usually confirms the presence of anthrax provided the smears are taken immediately after death
  - Samples of peripheral blood needed
  - Cover collection site with disinfectant soaked bandage to prevent leakage
  - Visualization of the capsulated bacilli, usually in large numbers, in a blood smear stained with polychrome methylene blue (Mc'Fadyean reaction) is fully diagnostic

3.1.1.7.Differential diagnosis

- There are many causes of sudden death in farm animals and differentiation is often difficult

- Diseases where there can be multiple deaths suggestive of anthrax include:

  - Lightning strike
  - Peracute blackleg
  - Malignant edema
  - Bacillary hemoglobinuria
  - Botulism
  - Poisoning (Plants, heavy metal, snake bite)
  - Peracute babesiosis

3.1.1.8. Treatment

- Severely ill animals are unlikely to recover but in the early stages, particularly when fever is detected before other signs are evident, recovery can be anticipated if the correct treatment is provided
  - Penicillin (20 000 IU/kg BW twice daily)
  - **Streptomycin** (8-10g/d in two doses intramuscularly for cattle) is much more effective
  - Oxytetracycline (5 mg/kg BW per day) parenterally has also proved superior to penicillin in the treatment of clinical cases after vaccination in cattle and sheep

3.1.1.9. Prevention and Control

- Report to authorities
- Do not open carcass (Necropsy not advised!)
- Infected carcasses should not be opened but immediately burned in situ or buried, together with bedding and soil contaminated by discharges
- If this can not be done immediately, a liberal application of 5% formaldehyde on the carcass and its immediate surroundings
- Burial should be at least 2 m deep with an ample supply of quicklime added

- **The Stern avirulent spore vaccine** (the live Sterne-strain spore vaccine) has overcome the risk of causing anthrax by vaccination and produces a strong immunity that lasts for at least 26 months in sheep and 1 year in cattle

- Vaccination should be done 2-4 weeks prior to the season when outbreaks may be expected

Quiz (5%)

Q1. Which **is the most common route of anthrax infection** in Animals? (1pt)

(A). Inhalation **(B).** Contact **(C).** Ingestion

(D). All of the above **(E).** B & C

Q2. Which characteristic dos Not contribute to Bacillus anthracis being an **ideal bioweapon** (1pt)

(A). It can be dispersed in a form that is easily inhalable

(B). It can cause an illness with high fatality rates

(C). Once made into a powder, its spores are difficult to kill

(D). It is an aerobic, gram-positive bacteria

(E). All of the above

(F). None of the above

Q3. Among the three forms of anthrax, cutaneous anthrax is the most severe form of anthrax which produces very painful skin lesions (1pt)

(A). True **(B).** False

Q4. How is Anthrax diagnosed in Animals? (2pts)

Quiz – Answer (5%)

Q1. Which **is the most common route of anthrax infection** in Animals? (1pt)

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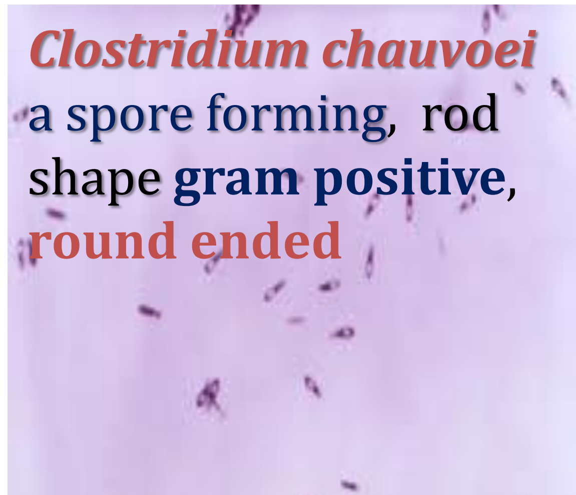
(A). True **(B). False**

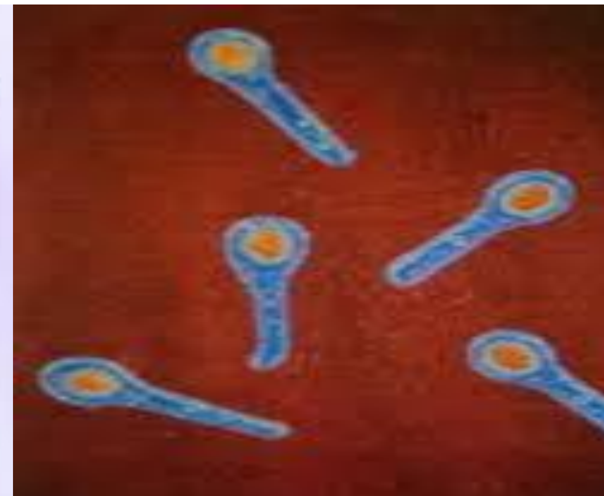
Q4. How is Anthrax diagnosed in Animals? (2pts)

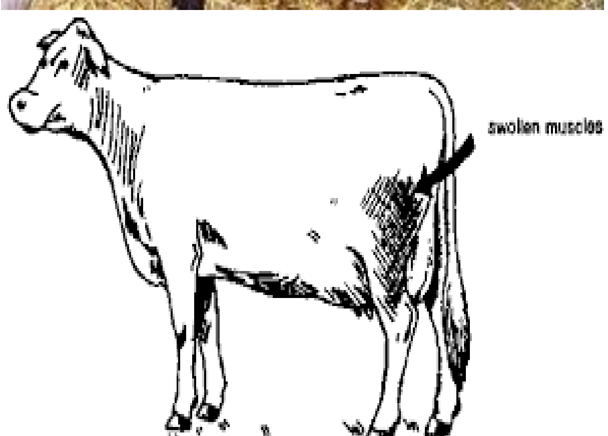
3.1.2. Blackleg

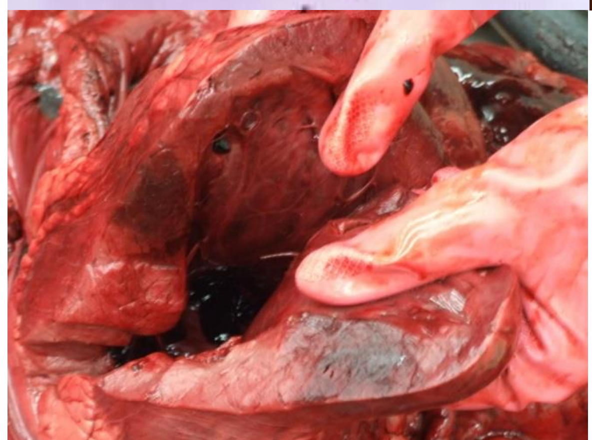


Clostridium chauvoei

a spore forming, rod shape **gram positive**, **round ended**







3.1.2. Blackleg

- **Others name for Black leg:** Black quarter, Quarter-ill, Quarter evil, Symptomatic anthrax, **Emphysematous gangrene**
- Blackleg is **an acute febrile**, highly infectious disease of **cattle** and **sheep** caused by *Clostridium chauvoei*, **characterized by** inflammation of muscle accompanied by emphysematous swelling (**painful, gaseous swellings**) **usually in the heavy muscles** (usually of the **upper parts** of the **legs**), **severe toxemia** and high mortality

- **In cattle**, blackleg infection is **endogenous** (i.e. *Clostridium chauvoei* is found **naturally in the intestinal tract** of animals)
- Lesions develop without any **history of wounds**, commonly, the animals that contract blackleg are of the **beef breeds**, in excellent health, and gaining weight

- **Cattle** are **between** the **ages of six months** and **two years**

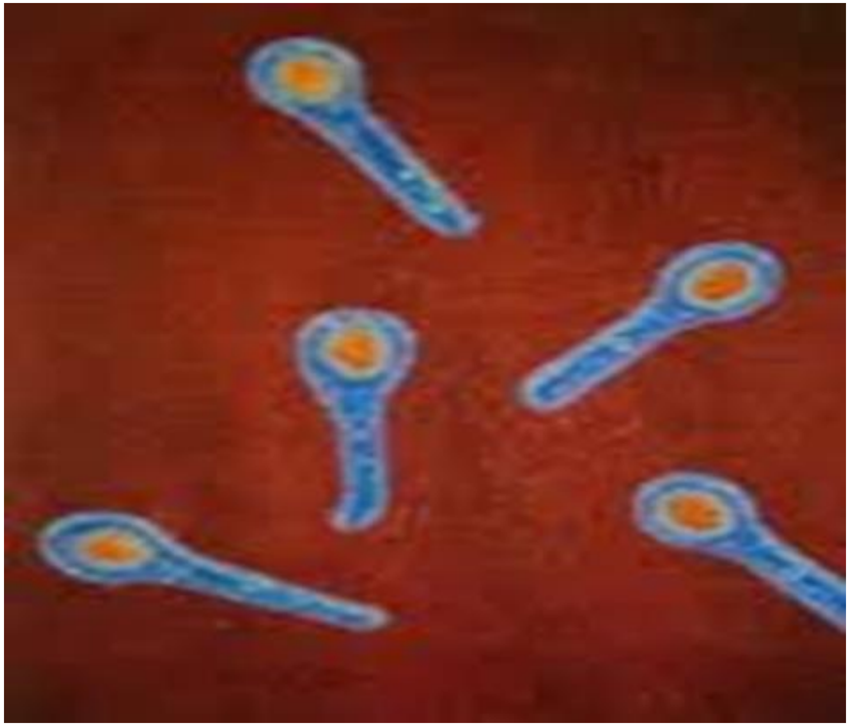
- **Calves as young as 6 weeks** and **cattle as old as 10–12 years may be affected**

- **In sheep**, the **disease is almost always the result of a wound infection** and often **follows some form of injury** such as **shearing cuts, docking**, **castration**

3.1.2.1. Etiology

- ***Clostridium chauvoei*** is **gram positive**, **spore forming**, **road shaped with round ended anaerobic organisms**

- The **spores are highly resistant to environmental changes** and **standard disinfectants** and so it can persist in the soil for many years and are purported to be a **source of infection**



3.1.2.2. Epidemiology

Epidemiology:

- Black leg is a worldwide distributed disease of ruminants but mainly affect cattle.
- The causative organism occurs naturally in the intestinal tract of animals and probably can remain viable in the soil for many years.
- Most cases occur in cattle from 6 months to 2 years old but calves as young as 6 weeks and cattle as old as 10-12 year may be affected.
- The disease usually occurs in summer and autumn.
- Contaminated postures appears to be sources of organism and it is assumed that the port of entry is through the mucosa of the alimentary tract
- The organisms are ingested, pass through the wall the wall of the gastro-intestinal tract and after gaining access to the bloodstream are deposited in muscle and other tissues.
- The bacteria can be found in spleen, liver and alimentary tract of normal animals they act as sources of infection.
- Muscle trauma associated with transporting herding and handing has been incriminated as creating suitable conditions in the muscle to allow bacterial multiplication and myonecrosis

3.1.2.3. Pathogenesis

- **The spores probably are ingested**, pass through the **wall of the gastro – intestinal tract**, and after **gaining access to the bloodstream**, are deposited in muscle (to **skeletal muscles**) and **other tissues** (spleen, liver, and alimentary tract) and may **remain dormant indefinitely**

- **Under muscular fatigue or trauma** to the muscle producing anaerobic conditions allow **spores to activate** (proliferate and produce toxins)

- The **toxins cause capillary damage**, hemorrhagic edema and **necrosis of myofibers** with **formation of gas**

- The organism is able to ferment sugar then form gangrene and toxemia

pathogenesis

Ingestion of spores

↓

intestine

↓

circulation

↓

Localization in skeletal muscle

↓

germination → multiplication → exotoxines

↑ (from exotoxines)

Muscle necrosis , pigment and toxemia

3.1.2.4. Clinical Signs

Clinical findings:

Per acute case:

- Sudden death without showing any symptoms

Acute case:

- Muscle trauma
- Ruminal stasis
- High fever (41-42^{0} C)
- Tachycardia (>100/min)
- Marked lameness with pronounced crepitating muscle and swelling femoral, lumbar gluteal or sacral regions or shoulder and neck .
- Swelling is hot and painful to touch firstly, then become cold and painless, edema and emphysema can felt.
- Depression
- Anorexia

clinical signs :

1-the animal may develop a fever.

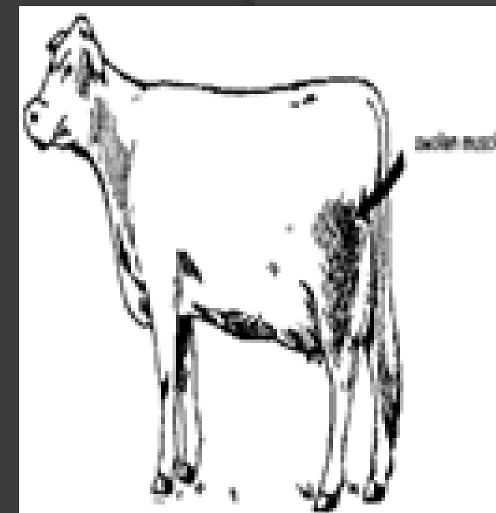
2-The limb usually swells significantly

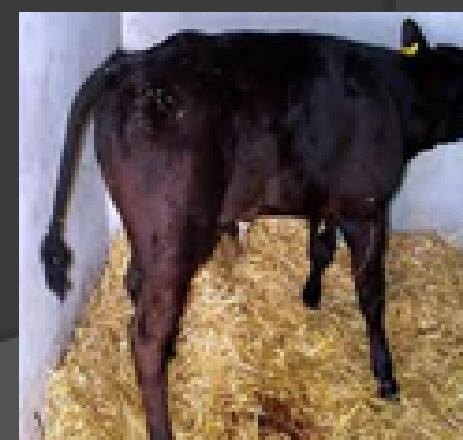
3- the animal can develop lameness on the affected leg.

4- Crepitation (the sensation of air under the skin) can be noticed in the hip, shoulder, chest, back, neck.

5- as the area seems to crackle under pressure

-The animal can live about 12-48 hours.





3.1.2.5. Diagnosis

- The **occurrence of a rapidly fatal febrile disease** in **well-nourished young cattle**, particularly of the beef breeds, with **crepitation sound** (swelling) of **the heavy muscles** **suggests Blackleg**

Muscle is dark red to black and spongy

Fig.: Affected muscle with black leg

Diagnosis:

1. **History:**
   - History of vaccination
   - Course of disease is per acute or acute.
   - Outbreak in 6 months to 2 years of age in cattle.
   - A rapidly fatal, febrile disease in well nourished young cattle with crepitant swelling of the heavy muscles suggests blackleg

2. **Clinical findings:**
   - Ruminal stasis
   - Crepitation of muscle
   - Swelling of muscle

3. Laboratory diagnosis

- Gram's staining of muscle reveals straight,round-ended gram positive rod, or pleomorphic
- Experimental inoculation:o.1-0.2ml of emulsion of lesion inoculation IM to guinea pig-Die within 1 to 2 days. Local lesion in guineapig similar to that in cattle.

4. Necropsy findings:

- Putrefaction can be found in sub cutis
- Muscle is dark red to black and spongy.

3.1.2.6. Differential Diagnosis

- **Anthrax**-No gas formation in muscle, splenic lesion and failure to blood clot.
- **Bacillary hemoglobinuria-**Liver infarct and haemoglobinuria findings identify the disease.
- **Malignant edema-**Little or no gas in the muscle tissue
- **Lactation tetany and acute lead poisonin**g-May cause sudden death but typical lesions of Blackleg are not present.
- **Emphysema** due to injury
- **Pasteurellosis**

3.1.2.7. Treatment

- Treatment of affected animals with **penicillin** and **surgical debridement of the lesion**

- **Recovery rates are low** because of the **extensive nature of the lesions**

- Large doses (40 000 IU/kg BW) should be administered, commencing with crystalline **penicillin intravenously** and followed by longer-acting preparations

- Blackleg **antiserum** is unlikely to be of much value in treatment **unless very large doses are given**

Treatment:

1. Large doses of penicillin @22,000 units/kg body weight should be administered with dose in the affected muscle directly and half dose intramuscularly twice for 5 to 7 days.
2. Alternatively oxytetracycline may be injected intramuscularly (10mg/kg bwt)
3. Antiserum-@100 to 200ml Intravenously
4. Drainage and flushing tissues to allow oxygen to affected tissues
5. Supportive treatment-Parenteral fluid or analgesics may be prescribed.

3.1.2.8. Prevention and Control

- **Control is by vaccination**:
  - On farms where the disease is enzootic, annual vaccination of all cattle is essential
  - In an outbreak all cattle in the remainder of the herd should be vaccinated immediately and injected with penicillin

- **Hygiene** i.e., cleaning and treatment of all traumatic wounds with antiseptics (alcohol, iodine etc.) until the wounds are healed in case of sheep is important

- Carcass of animals dying of blackleg must be destroyed by burning or deep burial to limit soil contamination.

Prevention and control:

- Formalin killed alum precipited vaccine @ 2ml/animal s/c for every year before the onset of rain
- Constant surveillance disease and early treatment of disease
- In an outbreak, all unaffected cattle should be vaccinated immediately.
- Proper disposal off carcasses, by burning or deep burial and reducing further occurrence of disease
- In case of high incidence of disease, vaccination of calves at 3 weeks of age and booster dose at 4 weeks apart.

3.1.3. Tetanus (Lock jaw)

- **Tetanus** is an infectious, highly fatal, **disease affecting all animals** and **man** (horse and man are the most susceptible hosts) caused by *Clostridium tetani* and characterized by **stiffness of muscles** and **closure of jaw**

- An infectious disease caused by **contamination of wounds** from the bacteria *Clostridium tetani*, or the spores they produce that live in the soil, and animal feces

- Tetanus is acquired through contact with the environment; it is **not transmitted from person to person**

- The usual locations for the bacteria to enter the body: **Puncture wounds** (such as those caused by **rusty nails**, splinters, or **insect bites**)

Host (spices affected):

- **Poultry:** Non affected because have antitoxin in the brain
- **Cattle:** Less susceptible because the grass contain spores so due to sublethal dose , resistance occur
- **Horse:** More susceptible because the HCL in the stomach juice destroy the spores contained in grass so the animal not exposed to sublethal dose

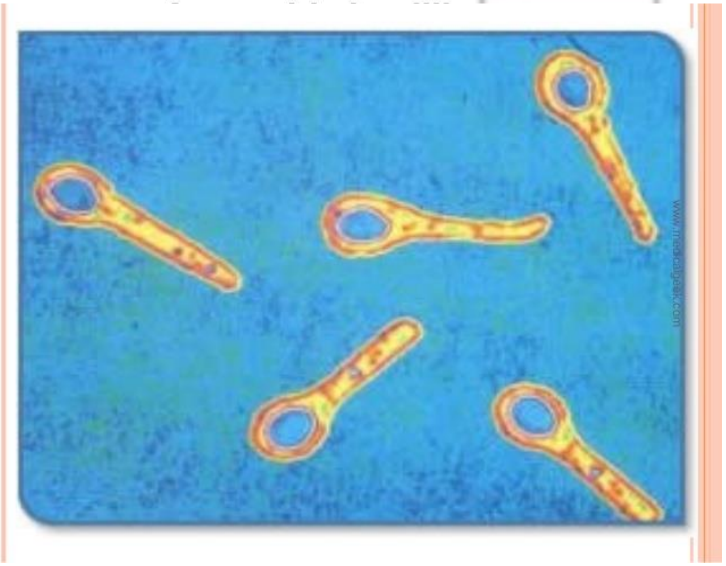
CAUSATIVE AGENT

- Caused by CLOSTRIDIUM TETANI
- Anaerobic
- Motile
- Gram positive bacilli
- Oval, colourless, terminal spores – tennis racket or drumstick shape.
- It is found worldwide in soil, in inanimate environment, in animal faeces & occasionally human faeces.

Caused by *Clostridium Tetani*

Gram positive

Spore-forming



3.1.3. 2. Transmission:

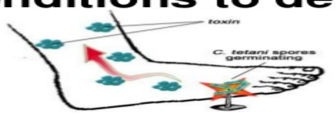
Methods of transmission

Deep wound

- Tetanus may follow burns, deep puncture wounds, ear or dental infections, animal bites, abortion.
- Only the growing bacteria can produce the toxin.
- **It is the only vaccine-preventable disease that is *infectious but not contagious* from person to person.**

C.tetani - *Entry of spores*

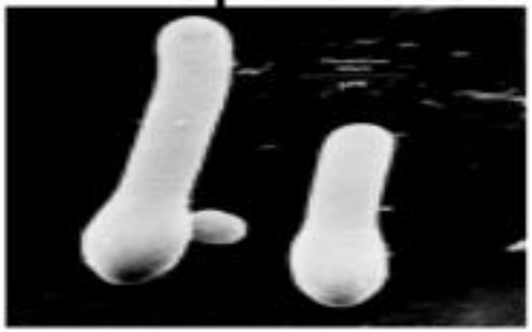
- **Entry of *C. tetani* into the body usually involves implantation of spores into a wound**
- **After gaining entry, *C. tetani* spores can persist in the body for months, waiting for the proper low oxygen growth conditions to develop**



Route of Entry

- Apparently trivial injuries
- Animal bites/human bites
- Open fractures
- Burns
- Gangrene
- In neonates usually via infected umbilical stumps
- Abscess
- Parenteral drug abuse

Sporulated



Vegetative

- Spores that gain entry can persist in normal tissue for months to years under anaerobic conditions.

- When the oxygen levels in the surrounding tissue is sufficiently low, the implanted *C. tetani* spore then germinates into a new, active vegetative cell that grows and multiplies and most importantly produces tetanus toxin - tetanospasmin and tetanolysin.

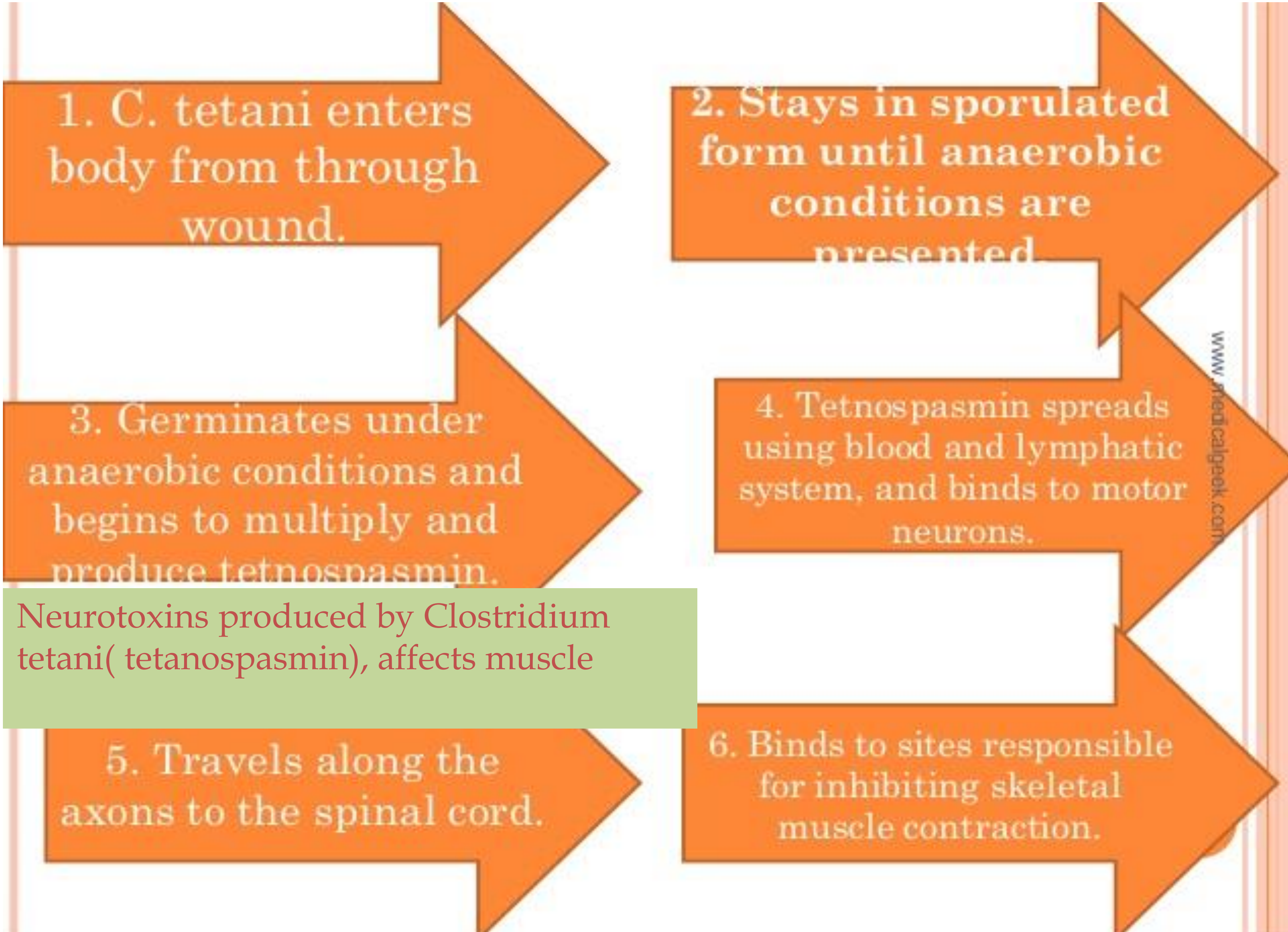
- **Tetanolysin** is not believed to be of any significance in the clinical course of tetanus.

- **Tetanospasmin** is a neurotoxin and causes the clinical manifestations of tetanus.

3.1.3. 3. Pathogenesis:

Pathogenesis:

- C. tetani usually enters body through a wound.
- In the presence of anaerobic conditions, the spores germinate and produce toxins.
- Toxins disseminated via blood and lymphatics.
- Toxins act at CNS sites including peripheral motor end plates, spinal cord, and brain, and in the sympathetic nervous system.
- The toxin interferes with the release of inhibitor neurotransmitters, Leading to muscle contraction and spasm.



Neurotoxins produced by Clostridium tetani(tetanospasmin), affects muscle

3.1.3. 3. Clinical signs:

- **Spasms** of **head muscles** (**lockjaw**)
- **In horses**, the **ears** are **erect**, the **tail stiff** and extended
- The **third eyelid prolapsed**
- **Walking**, **turning**, and **backing** are **difficult**

- Spasms of the neck and back muscles a "**sawhorse**" stance
  - **Prolonged muscular** action causes sudden, powerful, and painful contractions of muscle groups (this is called **tetany**), these episodes can cause fractures and muscle tears
  - **If respiratory muscle is involved** – apnoea

- Sweating is common , increased heart rate, rapid breathing, and congestion of mucous membranes

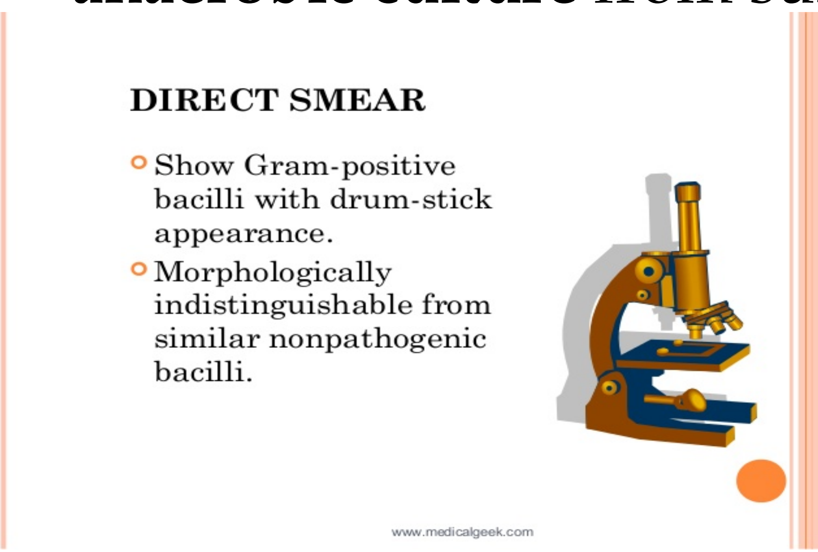
- **Spasms of head muscles (lockjaw)**

3.1.3. 4. Diagnosis:

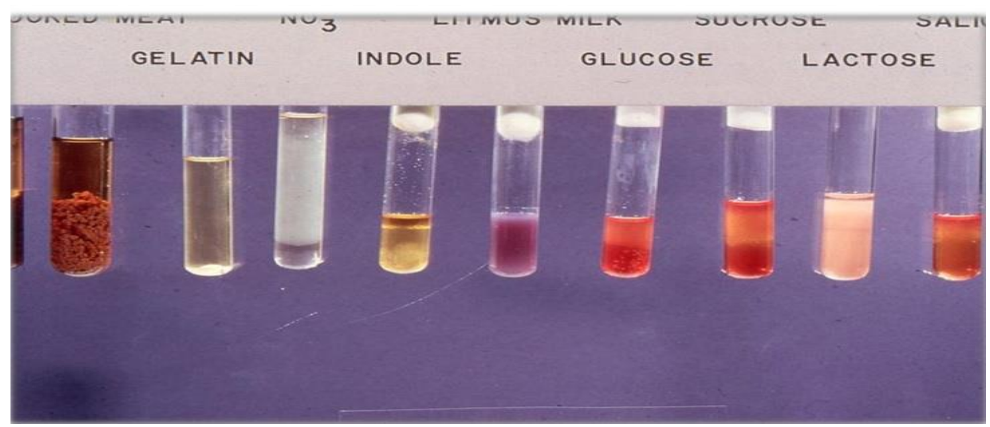
- The **clinical sings** and **history of recent trauma** are usually adequate for a diagnosis of tetanus

- **Diagnosis is done clinically** based on the presence of **generalized muscular rigidity** and or **spasm**

- The **diagnosis is confirmed by demonstrating the presence of tetanus toxin in the serum** and **gram-stained smears** and **anaerobic culture** from suspected wounds



Biochemical reactions will characterise the C.tetani



3.1.3. 5. Treatment:

- The main principles in the treatment of tetanus are to eliminate the causative bacteria, neutralize residual toxin, relax the muscle tetany to avoid asphyxia, and maintain the relaxation until the toxin is eliminated or destroyed

Patient Manifests with

- A person suffering from tetanus undergoes convulsive muscle contractions of the jaw--called LOCKJAW
- The contractions by the muscles of the back and extremities may become so violent and strong that bone fractures may occur
- The affected individual is conscious throughout the illness, but cannot stop these contractions

Other supporting measures?

- Remove and destroy the source of the toxin through surgical exploration and cleaning of the wound (debridement).
- Bed rest with a nonstimulating environment (dim light, reduced noise, and stable temperature) may be recommended.

Keeping animals in dark and quiet places may be helpful

- Respiratory support with oxygen, endotracheal tube, and mechanical ventilation may be necessary.

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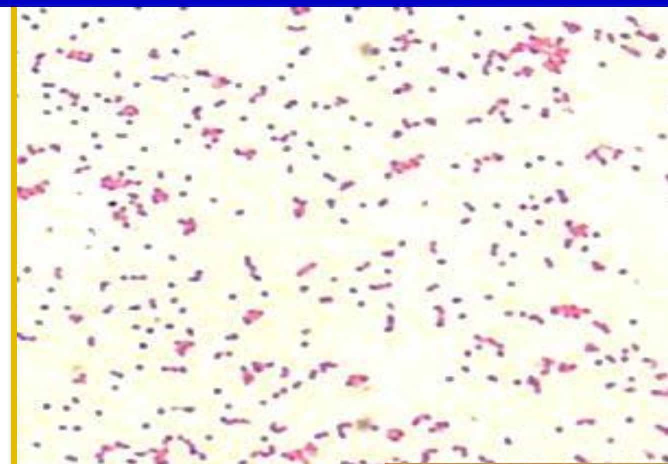
3.1.3. 5. Prevention & control:

- Proper hygiene and cleanness at castration and other surgical procedures
- Surgical wounds should be well drained and disinfected, and kept free of contamination by dirt
- Nails and wires should not be allowed to lie around premises where animals are kept
- **Tetanus is completely preventable** by active tetanus immunization (prevention can be achieved by active immunization by tetanus toxoid (TAT)
- Immunization is thought to provide protection for 10 years
- Clinical tetanus does not produce immunity to further attacks(therefore, even after recovery patients must receive a full course of tetanus toxoid)

3.1.4. Brucellosis



3.1.4. Brucellosis

- **Brucellosis** is a **bacterial disease** affecting animals (cattle, swine, sheep, goats, camels, dogs) and humans

- In general, the principal manifestations of brucellosis are reproductive failure, such as abortion or birth of unthrifty newborn in the female, and Orchitis and epididymitis with frequent sterility in the male

- Persistent (lifelong) infection is a characteristic of this facultative intracellular organism, with shedding in reproductive and mammary secretions

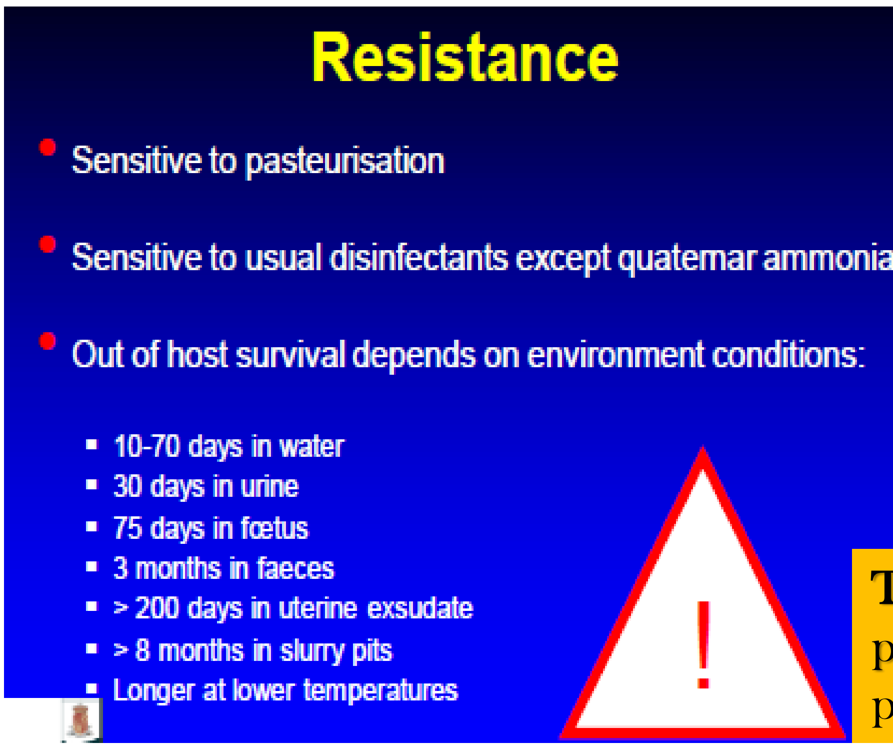
- It is a highly **contagious zoonosis** caused by ingestion of **unpasteurized milk** from infected animals, or **close contact** with their secretions

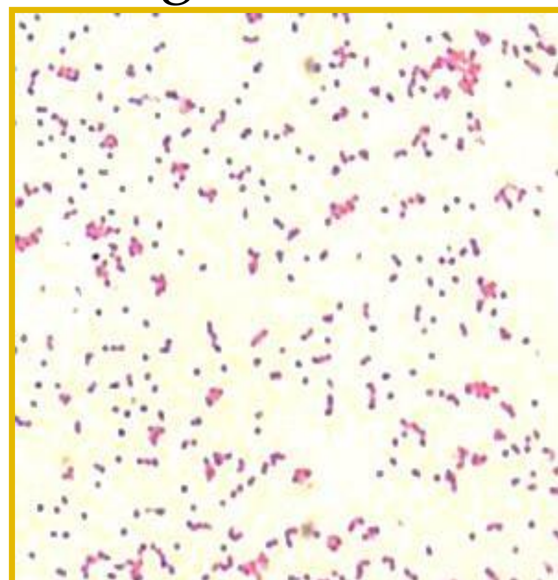
- Brucellosis is also an important zoonosis causing debilitating disease in humans

- The disease is commonly known as **Undulant** or **Malta fever** in humans and **Bang's disease** in animals

3.1.4.1. Aetiology (The Organism)

- **Brucellosis** is caused by various species of the genus ***Brucella***:-
  - Gram negative coccobacilli or short rod bacteria
  - Facultative anaerobe intracellular organism





The organism is able to **withstand drying**, **particularly** when organic material is present and **can survive in dust** and **soil**

Brucella Spices:

- Brucella spices cause life-long infection with intermittent shedding
- **Six named species occur in animals**

Species	Biovar/Serovar	Natural Host	Human Pathogen
B. abortus	1-6, 9	cattle	yes
B.melitensis	1-3	goats, sheep	yes
B. suis	1, 3	Swine	yes
	2	Hares, wild boar	(Yes)
	4	reindeer, caribou	yes
	5	rodents	yes
B. canis	none	dogs, other canids	yes
B. ovis	none	sheep	no
B. neotomae	none	Desert wood rat	no

B. abortus

B.melitensis

B. suis

B. canis

B. ovis

B. neotomae

(1)Transmission in Animals:

- **Ingestion** of infected tissues or body fluids (discharge during parturition, vaginal discharge, colostrum…)
- **Contact** with infected tissues or body fluids
- **Venereal**
- **Fomites:-**
  - ➢ Brucella can be spread on fomites including feed and water

(2) Transmission in Humans:

- **By ingestion:**
  - Raw milk and unpasteurized dairy products
  - Rarely through **undercooked** meat
- **By contact** of infected material with the conjunctiva (mucous membrane) and broken skin
  - Blood, urine, vaginal discharges, aborted fetuses, placentas
- **Aerosol:**
  - Laboratory, abattoirs
  - Pens, stables
- **Inoculation during vaccination:**
  - *B. abortus* strain 19, RB-51
  - *B. melitensis* Rev-1
- **Person-to-person** transmission **rare**

Third trimester abortions with *Brucella abortus*

Clinical signs in pregnant heifers and cows

- Abortions, later and later
- Placental retention (even without abortion)
- Metritis and/or infertility
- Mastitis (frequently inapparent)
- Artritis, knee hygroma

Clinical signs in bulls

- Orchitis
- Infecondity
- Artritis, knee hygroma

Clinical signs in calfs

- Intra-uterine deathy (abortion)
- Terms reached stillborn calfs
- Live calfs being ill from birth on
- Apparently healthy calfs, still live long germ carriers

Clinical signs in Small ruminants:

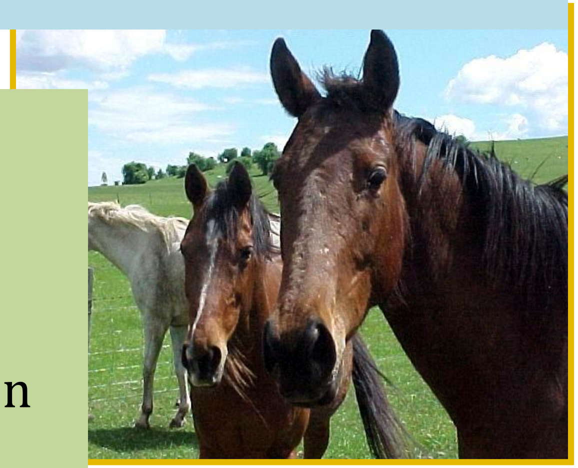
- ***B. melitensis*** causes late term abortions
  - Retained placenta
  - Birth of dead or weak lambs/kids
- ***B. ovis*** causes abortions, fertility problems
  - Orchitis, epididymitis

Clinical signs in Horses:

- Whithers infected ,not reproductive tract(uterus)

- *B. abortus* most common
  - Susceptible to
    B. suis
- **Fistulous Withers** or Poll Evil

- Suppurative/pyogenic infection on whither called poll evil/fistular whither
- Pus from open wound on whither



Clinical signs in Humans

- Can affect any organ or organ system
- All patients have a cyclical fever
- **Variability in clinical signs**
  - Headache,
  - Weakness,
  - Depression,
  - Weight loss,
  - Fatigue,

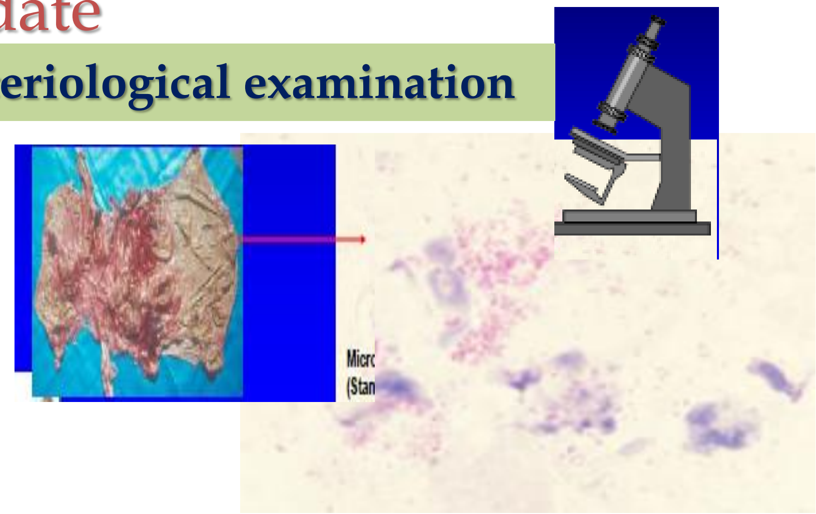
Populations at Risk?

- **Occupational disease:**
  - Cattle ranchers/dairy farmers
  - Veterinarians
  - Abattoir workers
  - Meat inspectors
  - Lab workers
- **Hunters**
- **Consumers:** (Unpasteurized dairy products)

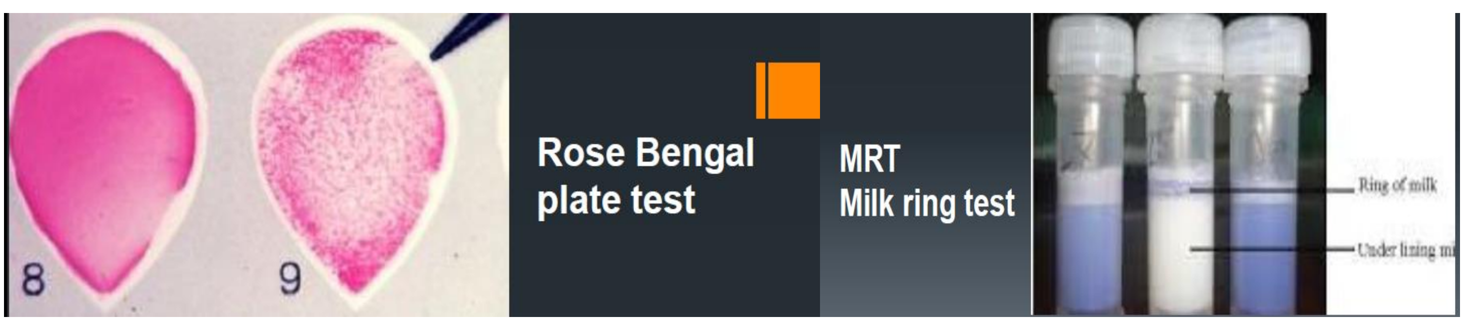
3.1.4.4. Diagnosis

- **Culture:**
  - Isolation and characterization of the organism from blood, organs and lymph nodes of the fetus, the placenta, milk, vaginal mucus, or uterine exudate

Bacteriological examination



- **Serological tests:**
  - Rose Bengal Plate Test
  - Complement fixation
  - Milk Ring Test
- **PCR**



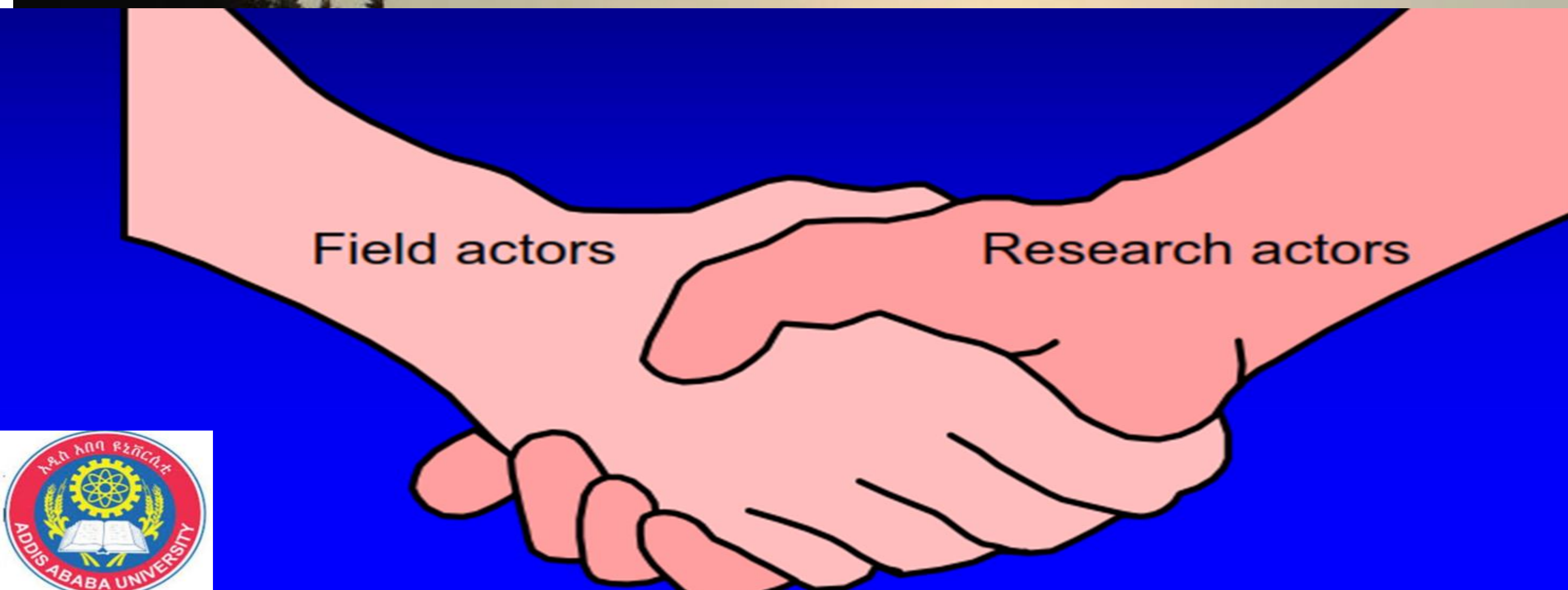
3.1.4.5. Prevention and Control

- **Education** about risk of transmission:
  - Farmers, veterinarians, abattoir workers, butchers, consumers, hunters
- **Wear proper** clothing if dealing with infected animals/tissues
  - Gloves, masks, goggles
- **Avoid consumption** of raw dairy products
- **Immunize** in areas of high prevalence
- **Eradicate reservoir**
  - Identify, segregate, and/or cull infected animals

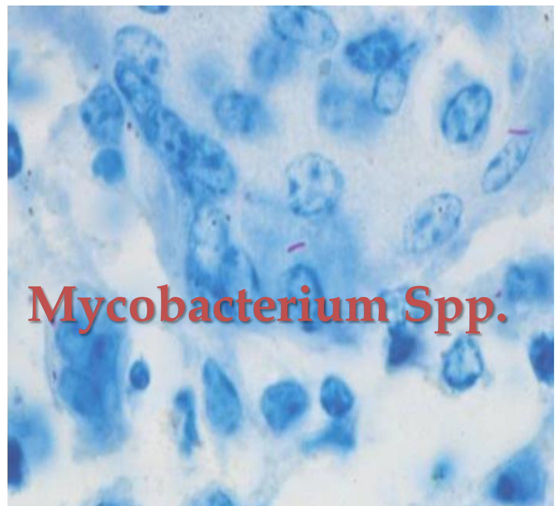
In wildlife: many other species are affected !!!

…and thus control of brucellos
is more difficult in wildlife


Thank you


Field actors
Research actors

3.1.5. Tuberculosis



3.1.5. Tuberculosis (TB)

- **Tuberculosis** (**TB**) is **a chronic highly contagious disease** of **both animals** and **humans** **characterized** by the **growth** of **nodules** (**tubercles**) in the **tissues**, **especially in the lung** and it is caused by **Mycobacterium species**

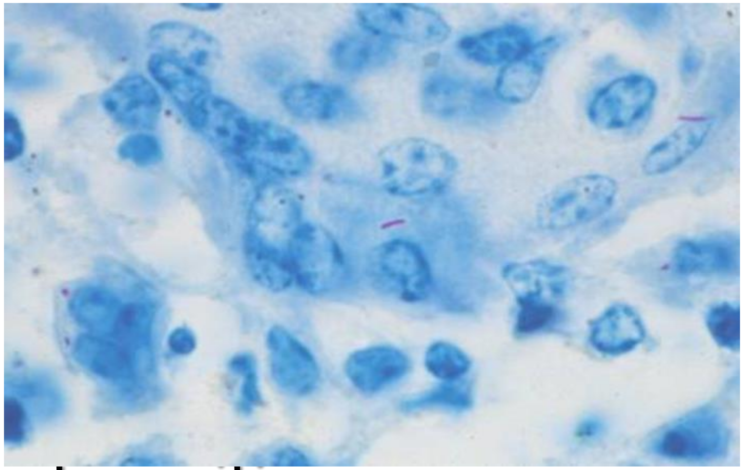
- The **name Tuberculosis** comes from the **nodules**, called **‘tubercles’**, which form in the **lymph nodes** (**tissues, e.g. lung**) of affected animals

- **Tuberculosis** (**TB**) remains an important disease of **cattle**, **wild animals**, and is **a significant zoonosis**

3.1.5.1. Etiology

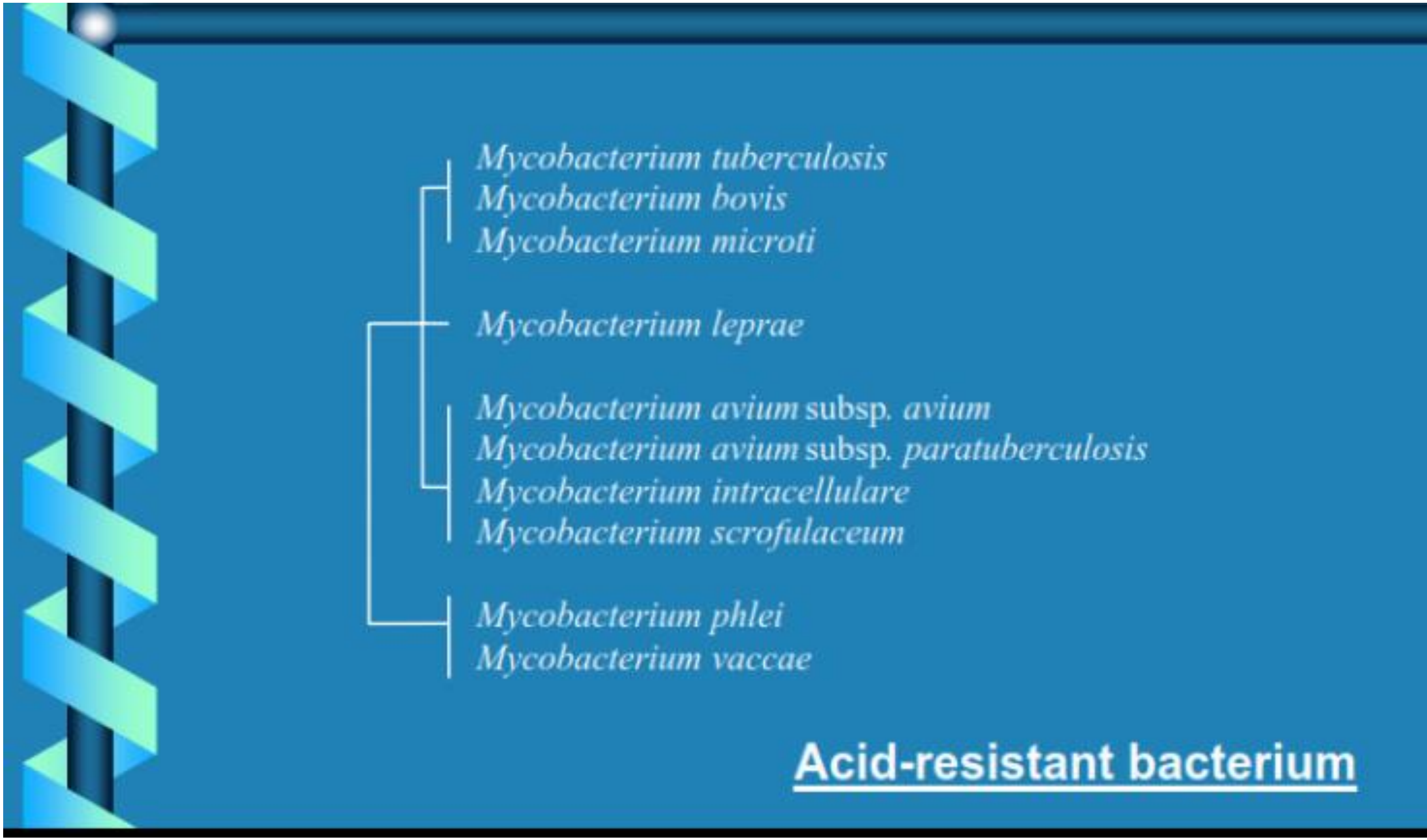
Caused by **Mycobacterium species**:

- **Unique bacteria** [i.e. **cell walls** contain a lot of waxy material (**mycolic acid**), which inhibits the uptake of nutrients into the bacterial cell, and these factors contribute to the **slow growth rate**]
- **Immobile**, slow growing rod-shaped, **due to their special staining characteristics under the microscope**, which is **mediated by mycolic acid** in the **cell wall**, they are called **acid-fat organisms**
- Mycobacteria do not grow outside of a host except in cultured media



- Require oxygen for growth, very heat sensitive

- **Tuberculosis** is caused by *Mycobacterium bovis* (**cattle**), *Mycobacterium avium* (**poultry**) and *Mycobacterium tuberculosis* (**man**) species which is **an acid fast bacterium**



Three Types of Tuberculosis

- *Mycobacterium* bovis (bovine)
  - ✓ **Causes Bovine Tuberculosis**
- *Mycobacterium* avium (bird)
  - ✓ **Causes Avian Tuberculosis**
- *Mycobacterium* tuberculosis
  - ✓ **Causes Human Tuberculosis**

Mycobacterium bovis

- Bovine TB can be transmitted from livestock to humans, deer and other animals
- No other organism has as great a host range as bovine TB
- Bovine TB can infect all warm-blooded vertebrates



Mycobacterium avium

- **Can affect all species of bird**
- **Can affect hogs and cattle**

Mycobacterium tuberculosis

- **Primarily affects humans**
- **Can be transmitted to hogs, cattle, and dogs**

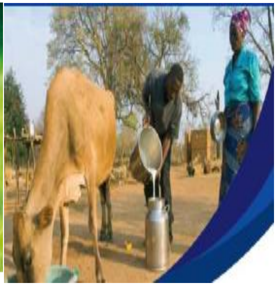
Bovine Tuberculosis

- It is **a form of tuberculosis in cattle** caused by *Mycobacterium bovis*, which is closely related to the **bacteria** that cause human and avian tuberculosis

- **Although cattle** are considered to be the true hosts of *Mycobacterium bovis*, the disease has been reported in many other domesticated and non-domesticated animals

It is ZOONOTIC TUBERCULOSIS

- The disease is **contagious** and spread by contact with infected domestic and wild animals



- It often affects sites other than the lungs (**extra pulmonary**), but in many cases is clinically indistinguishable **from TB** caused by *Mycobacterium tuberculosis*

3.1.5.2. Transmission

- **The usual route of infection** (most common means of transmission) is by **inhaling infected droplets** which are expelled from the lungs by **coughing**

- **Young animals** and **humans** can contract the disease by drinking raw milk from infected dams (cows)

- Infected cattle are the main source of infection for other cattle

- Infection by ingestion is possible at pasture when feces contaminate the feed and communal drinking water and feed troughs but a large infective dose is required

- **Organisms excreted in (Source of infection:)**

    - Animals may retain the microorganism for many years in the capsule, which may break down and release microorganisms into the surroundings, thus setting off an outbreak in a susceptible herd

    - Exhaled air
    - Sputum
    - Feces
    - Milk
    - Urine
    - Vaginal and uterine discharges
    - Discharges from open peripheral lymph nodes

Who is at risk?

- Animals kept in close contact with other infected animals in enclosed areas like barns are at greatest risk for exposure to bovine TB.

- Housing; high density, poor ventilation, stressors, immunosuppress or diseases are the factor increasing incidence of the tuberculosis

- Housing predisposes to the disease, as does high stocking intensity and a large number of animals on a farm so that the disease is more common and serious where these forms of husbandry are practiced

- The closer the animals are in contact the greater is the chance that the disease will be transmitted

3.1.5.3. Epidemiology

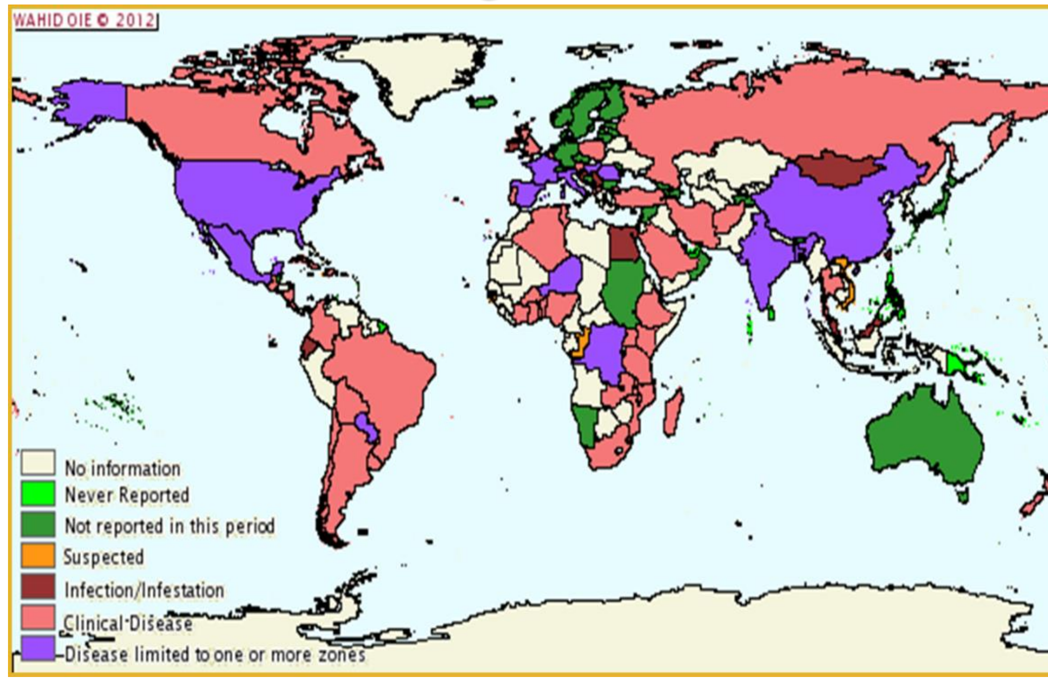
- All species, including humans, and age groups are susceptible to *Mycobacterium bovis*, with cattle, goats, and pigs most susceptible and **sheep** and **horses** showing **a high natural resistance**

  ✓ Isolations have been made from wild animals (buffaloes, boars, deer, antelopes, etc)

- The presence of the disease is usually signaled by detection in carcasses at abattoirs

- TB is found throughout the world

- The disease is more prevalent in most of Africa, parts of Asia and of the Americas



- Bovine TB is endemic in animal populations throughout much of the world

3.1.5.4. Pathogenesis

- **Tuberculosis** spreads in the body by two stages, (1) **the primary complex** and (2) **post primary dissemination**

(1). The primary complex consists of the lesion at the point of entry and in the local lymph node

- A lesion at the point of entry is common when infection is by inhalation
- When infection occurs via the alimentary tract, a lesion at the site of entry is unusual,(the only observable lesion is in the pharyngeal or mesenteric lymph nodes)

- A visible primary focal lesion develops within 8 days of entry being effected by the bacteria
- Calcification of the lesions commences about 2 weeks later

- The developing necrotic focal lesion is soon surrounded by **granulation tissue**, monocytes, and plasma cells and the pathognomonic '**tuberculosis**' is established

(2). Post-primary dissemination from the primary complex may take the form of acute miliary tuberculosis, discrete nodular lesions in various organs

- Depending upon the sites of localization of infection, clinical signs vary, because the disease is always progressive, there is the constant underlying toxemia which causes weakness, debility, and the eventual death of the animal

3.1.5.5. Clinical Signs

- TB usually has a prolonged course, and symptoms take months or years to appear (i.e. early stage may be asymptomatic)
- **The usual clinical signs include:**
  - In most cases progressive emaciation
  - Chronic cough due to bronchopneumonia (moist cough)
  - Weakness,
  - Loss of appetite,
  - Weight-loss,
  - Fluctuating fever,
  - Intermittent hacking cough,
  - Diarrhea,
  - Large (enlarged) prominent lymph nodes
- However, the bacteria can also lie dormant in the host without causing disease

3.1.5.6. Post Mortem Lesions

- **Bovine tuberculosis** is characterized by the formation of granulomas (tubercles) where bacteria have localized

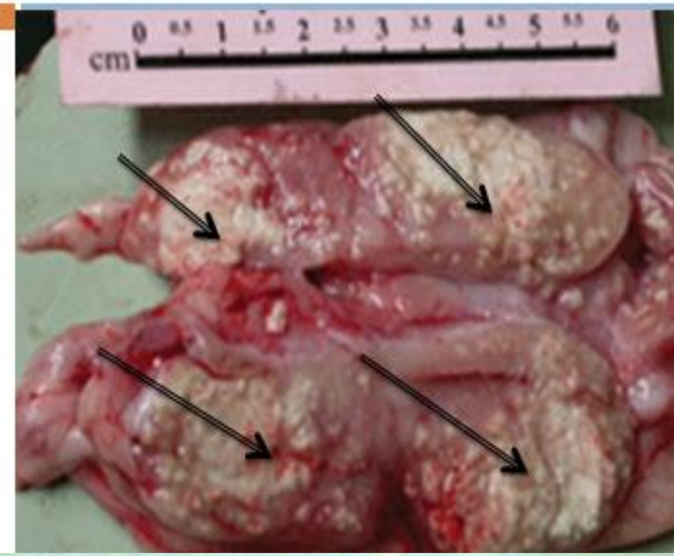
- These granulomas are usually (i.e. the appearance of granulomas are):
  - Yellow
  - Caseous
  - Calcified
  - May resemble abscesses

- In cattle, tubercles are found in the lymph nodes, particularly those of the head and thorax

- They are also common in the lung, spleen, liver and the surfaces of body cavities

Multi focal lesions of TB in lung

Milliary TB lesions in medistinal lymph nodes of goats



Lesions: Caseous lesions of *M.bovis* in lungs

Pericardial granuloma

3.1.5.7. Diagnosis

- The standard method for detection of TB is the **tuberculin test**, where a small amount of antigen is injected into the skin, and the immune reaction is measured

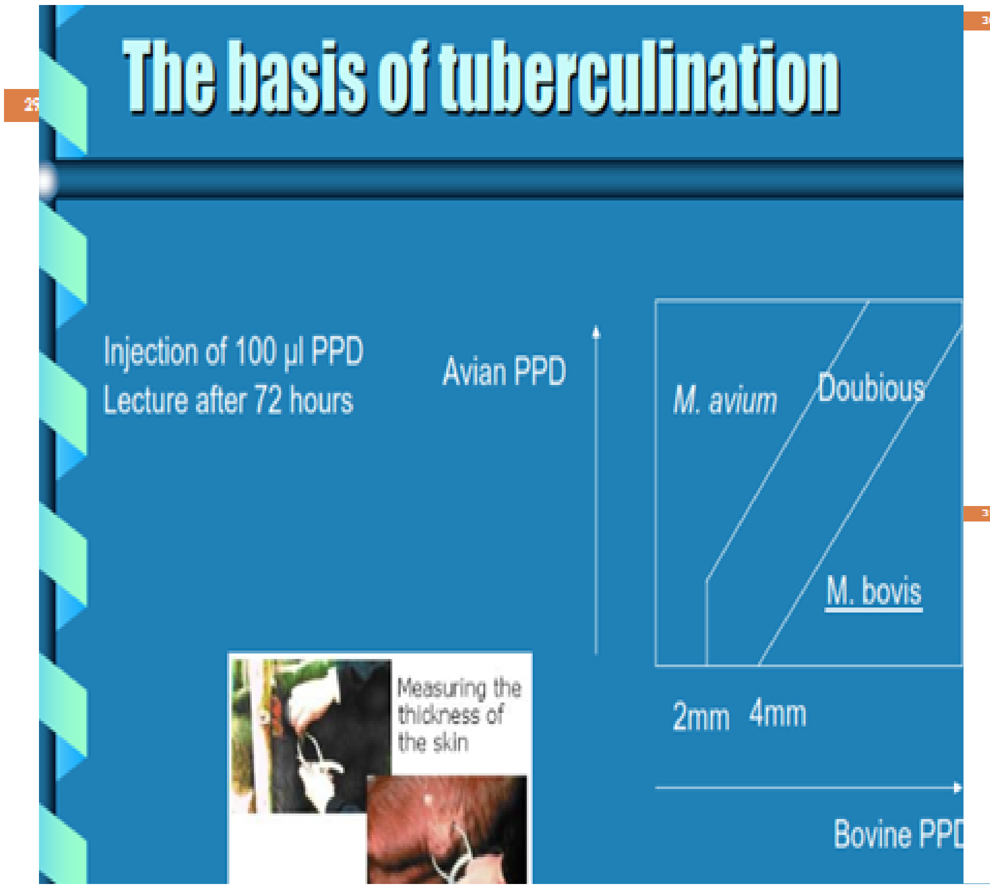
- **In live cattle**, tuberculosis is usually diagnosed in the field with the tuberculin skin test

- In this test, tuberculin [intradermal injection of bovine tuberculin purified protein derivative (PPD)] is injected intradermally; a positive test is indicated by a delayed hypersensitivity reaction (swelling)

- The tuberculin test can be performed using bovine tuberculin alone [Single intradermal (SID) test], or as a comparative test that distinguishes reactions to *M. bovis* from reactions to *M. avium* (i.e. Avian and bovine tuberculin are injected simultaneously into two separate sites on the same side of the neck, 12 cm apart and one above the other)

- The reaction is read between 48 and 96 hours after injection with a preference for 48-72 hours for maximum sensitivity and at 96 hours for maximum specificity, and a positive reaction constitutes a diffuse swelling at the injection site

Tuberculin test



SID - interpretation

Official lecture: Clinical signs	Official lecture: Thicknees (mm)	RESULT	CONTEXTUAL INTERPRETATION
-	X < 2	-	· Clinical picture · Lesions · Germs isolation · Infected herd
-	2 < X < 4	± ?	
-	X > 4	+ ?	
+		+	

Please place all « + » or « +/- » bovines in quarantine

CID - interpretation

Official lecture: Clinical signs	Official lecture: Thicknees (mm)	RESULT	CONTEXTUAL INTERPRETATION
-	Bo < 2	-	· Clinical picture · Lesions · Germs isolation · Infected herd
-	Bo < Av	-	
-	Bo – Av < 4	± ?	
-	Bo – Av > 4	+	
+		+	

Please place all « + » or « +/- » bovines in quarantine

- **Finding gross lesions is not conclusive evidence** that the animal is infected with the disease, thus further testing is required

- Thus the tissue samples collected during the necropsy are examined for **histopathological** (microscopic) lesions

- Most common lesion associated with bovine TB is the granuloma

- **Definitive diagnosis** is made by growing the bacteria in the laboratory (culture), a process that takes at least eight weeks

- Special stain, called acid-fast stain, allows for bacteria to be visualized

- PCR

3.1.5.8. Differential Diagnosis

Differential Diagnosis

- Bovine pleuropneumonia
- *Pasteurella*
- *Corynebacterium pyogenes*
- Aspiration pneumonia
- Traumatic pericarditis
- Caseous lymphadenitis
- Melioidosis
- Chronic aberrant liver flukes

3.1.5.9. Prevention and Control

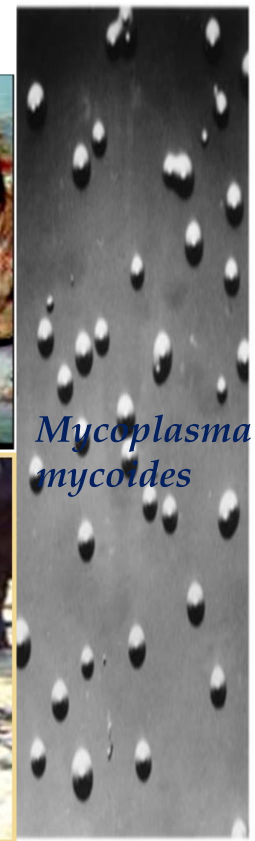
- The standard control measure applied to TB is test and slaughter
- Eradication of bovine tuberculosis has been virtually achieved in many countries
  - The methods used have depended on a number of factors but ultimately the test and slaughter policy has been the only one by which effective eradication had been achieved
- Pasteurization of milk of infected animals to a temperature sufficient to kill the bacteria has prevented the spread of disease in humans
- Treatment of infected animals is rarely attempted (treatment not advised) because of the high cost, lengthy time and the larger goal of eliminating the disease
- Vaccination is practiced in human medicine, but it is not widely used as a preventive measure in animals

3.1.6. Contagious Bovine Pleuropneumonia (CBPP)

- Bacteria (**Mycoplasma**) are the causative agent of contagious bovine pleuropneumonia (CBPP)

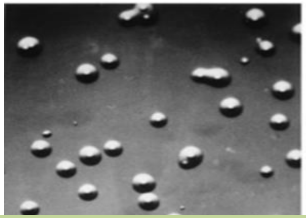
- **CBPP** is extremely infectious in cattle, and causes lung and occasionally joint disease

- Natural hosts are bovine and zebu and is not communicable to other species



3.1.6. 1. Etiology

- *Mycoplasma mycoides subsp. mycoides small-colony* type (MmmSC type) bacteria is the causative agent of contagious bovine pleuropneumonia (CBPP)
- *Mycoplasma mycoides subsp. mycoides large-colony* type is the causative agent of contagious caprine pleuropneumonia (CCPP) and does not affect cattle
- Characteristics:
  - **Absence** of a cellular wall
  - Absence of peptidoglycan precursors
  - Resistant to penicillin
  - Sensitive to environment
  - Variable morphological aspect: coccoid to filamentous (i.e. the organisms are pleomorphic and some forms are filterable)
  - Does not survive in meat or meat products



- MmmSC type is very similar culturally and antigenic ally to the causative organisms of CCPP but the two can be differentiated culturally and biochemically

3.1.6. 2. Transmission:

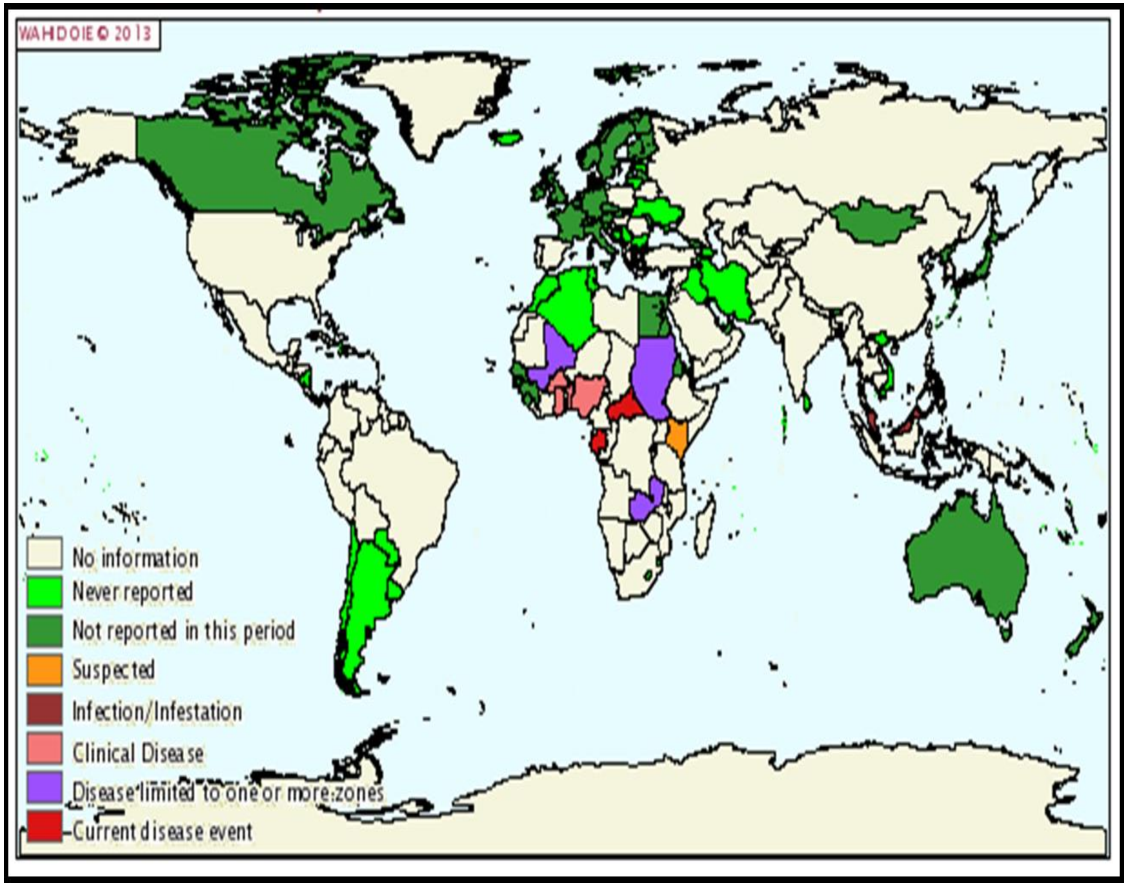
- **Aerosol**
  - Primary route of transmission
  - Breathing in infected drops from coughing animal
- **Direct contact**
  - Introduction of carrier most common cause of outbreaks
  - Infection from cow to unborn calf has been known to occur

- Introduction of carrier animal
  - Most common cause of outbreaks
- Aerosol (close contact)
- Direct contact
  - Saliva, urine, fetal membranes, uterine discharges
  - Transplacental
- Humans are not susceptible

- **CBPP is transmitted primarily by two different routes:**
  - ✓ **One is by aerosol transmission** (close proximity is necessary for transmission, which occurs primarily through breathing in infected droplets from a coughing animal)
  - ✓ **The second route is direct contact** (direct contact by the introduction of a carrier animal into a susceptible herd is the most common cause of outbreaks)

3.1.6. 3. Epidemiology:

- Under natural conditions, CBPP occurs in cattle of the species both **Bos taurus** and **Bos indicus** including buffalo, bison
- CBPP is widespread in Africa (i.e. African disease) and occurs in some countries of Asia and Europe
- CBPP in Africa was causing greater losses in cattle than any other disease
- Long Prepatent period (1 to 3 months)
- Inapparent carriers are a major source of infection (lungers)



Species Affected

- Cattle
- Asian buffalo
- Captive bison
- Yak

- Humans are not susceptible

- **The focus of infection is often provided by recovered 'carrier' animals in which a pulmonary sequestrum preserves a potential source of organisms for periods as long as 3 years**

- The focus of infection is often provided by recovered 'carrier' animals in which a pulmonary sequestrum preserves a potential source of organisms for periods as long as 3 years

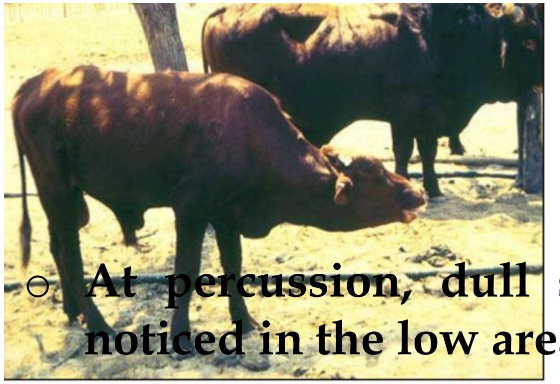
- Of recovered animals, as many as 25% may be carriers (recovered animals capable of giving CBPP to other cattle without being sick themselves)

- The chance of sickness increases with close confinement, and can reach 100% in susceptible herds

- Death rate ranges from 10-70% and can be affected by secondary factors in overall health, such as poor nutrition and a parasite burden

3.1.6. 4. Clinical signs:

- There is considerable variation in the severity of clinical disease from hyperacute to acute to chronic and subacute forms
- Acute Infections
  - Incubation period is highly variable, ranging from 10 days to six months
  - Moderate fever with respiratory, pulmonary and pleuretic symptoms
  - Polypnea (Increased breathing rate)
  - Elbows turned out,
  - Arched back,
  - Head extended
  - Tongue protruded
  - Open mouth
  - Cough, lack of appetite, lack of energy, fever



- At percussion, dull sounds can be noticed in the low areas of the thorax

Clinical Signs: Chronic Infections

- Less obvious signs of pneumonia
  - Coughing with exercise
  - Extreme weight loss; recurrent mild fever
  - Recover after several weeks
- Calves infected when they are born
  - Arthritis in several joints
  - May not show signs of pneumonia
- Healthy appearing animals may spread CBPP

Center for Food Security and Public Health
Iowa State University 2006

Clinical Signs: Chronic Infection

- ✓ Depressed
- ✓ Reluctant to move
- ✓ Thin

Emaciation, depression

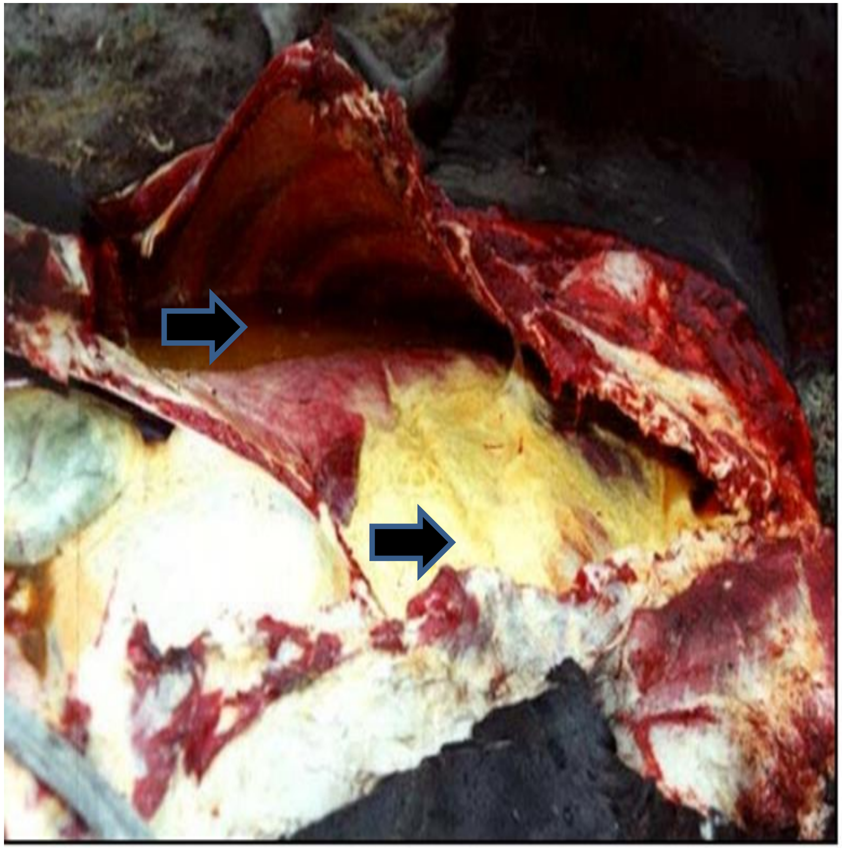
Center for Food Security and Public Health, Iowa State University, 2011

- ***In calves***
  - Pulmonary tropism is not the general rule
  - Arthritis with swelling of the joints
- Pulmonary symptoms in adults and arthritis in young animals alert the clinician to a diagnosis of CBPP

5/26/2019

3.1.6. 5. Post Mortem Lesions (Necropsy Findings)

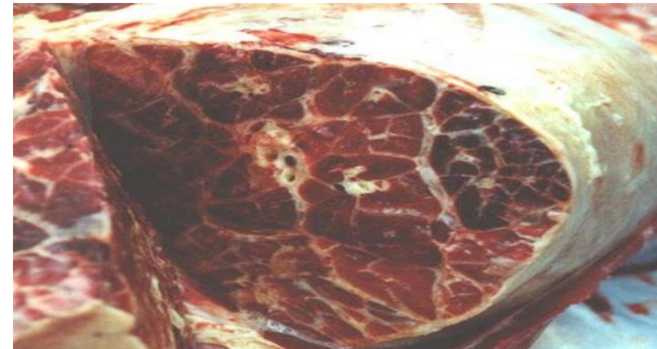
- **Lesions** are confined to the thoracic cavity and lungs and the lesions are usually unilateral



- The pleural cavity may contain large quantities of clear, yellow-brown fluid (up to 30 liters) containing pieces of fibrin (this fluid is ideal for culture of the organism)

- Caseous fibrinous deposits are present on the parietal and visceral surfaces of the lungs (i.e. fibrinous pleurisy: thickening and inflammation of the pleura with fibrous deposits)

- A characteristic "marbled" appearance of the affected lungs is caused by the presence of both acute and chronic lesions in the interlobular septa
    - i.e. Interlobular oedema, marbled appearance due to hepatisation (hepatized lung) and consolidation at different stages of evolution usually confined to one lung
- Consolidation of the lungs with a typically marbled appearance is characteristic
- Encapsulated sequestra containing necrotic tissue can be found even in recovered animals, and the animal may become a carrier



3.1.6. 6. Diagnosis:

- **Clinical signs:** not very specific (i.e. since there can be many causes of severe pneumonia in cattle, contagious bovine pleuropneumonia is difficult to diagnose based on clinical signs alone)

  - Difficult to distinguish from other respiratory diseases in cattle
  - Animals with CBPP frequently present with unilateral pneumonia, and in a herd with signs of pneumonia in adults and polyarthritis in calves, CBPP should be considered

- **Necropsy (Post mortem lesions):** are more specific and often helpful in diagnosis

Diagnosis: Laboratory

- Culture *Mycoplasma mycoides* subsp. *mycoides* can be directly identified by culture
- Immunological tests
- PCR
- Serology
  - Complement fixation
  - Competitive ELISA
  - Immunoblot
  - Latex agglutination

- Lab. Samples (culture)
  - **Pleural fluids**, lung lesions, lymph nodes, lung tissue exudates

Center for Food Security and Public Health, Iowa State University, 2011

3.1.6. 7. Differential Diagnosis:

Differential Diagnosis

- Bovine pasteurellosis
- Hemorrhagic septicemia
- Theileriosis (East Coast fever)
- Rinderpest
- Traumatic pericarditis
- Bronchopneumonia resulting from mixed infections

Center for Food Security and Public Health, Iowa State University, 2011

3.1.6. 8. Treatment:

- Treatment is recommended only in endemic areas because elimination of the organism may not be possible and carriers may develop

- Antibiotic treatment is generally not effective as it can result in extensive tissue damage and sequestration of the organism, although tylosin has been reported to be moderately effective

- **Commonly used antibiotics are:**
    - Macrolids (erythromycin, tylosine, etc)
    - Tetracyclines (Oxyttc)
    - Aminoglycosides (Streptomycin)

- As soon as an outbreak is suspected, slaughter and necropsy of a suspect animal is advisable

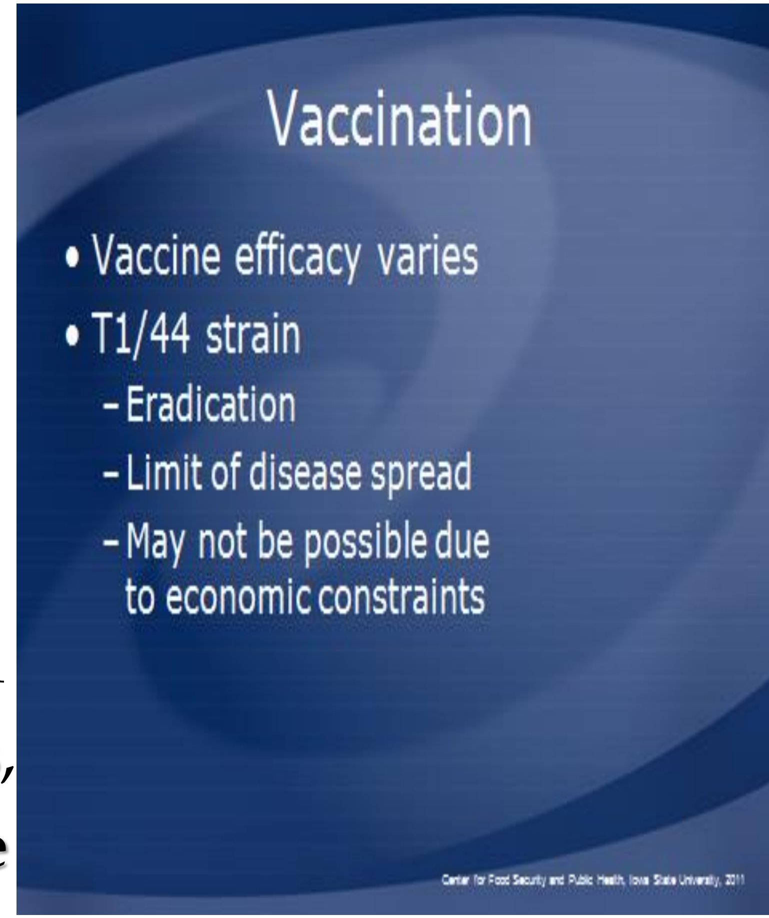
3.1.6. 9. Prevention & control:

- As soon as an outbreak is suspected, animals that were exposed to animals with CBPP must be quarantined and infected and exposed animals will likely be slaughtered

- (i.e. Quarantine of exposed and infected animals is recommended along with restricted movement, testing, and slaughter of infected animals)

- The animals and contaminated materials will need to be disposed of properly

- **Immunization with an attenuated vaccine** (**T1/44 strain**) is helpful in disease eradication

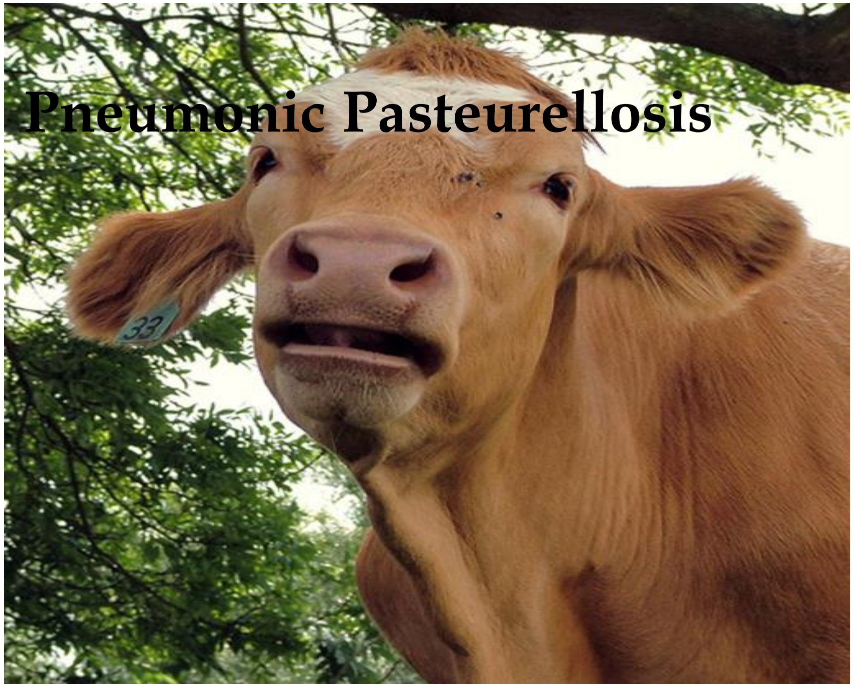


- In areas where cattle cannot be confined, the spread of infection can be curbed by vaccination

- However, many of the countries in which CBPP is a serious problem have desperate economic situations, and vaccination may not be possible

- To prevent CBPP from entering your farm, all animal movement on and off your premise must be stopped

3.1.7. Pasteurellosis



3.1.7. Pasteurellosis

- **Pasteurellosis is a group of diseases** affecting **different species of animals** (cattle, sheep, poultry, horse, pig and etc) caused by; *Pasteurella multocida* and *Pasteurella haemolytica*

- i.e. The groups of microorganisms known as pasteurellae are responsible for a number of different diseases in cattle, sheep, goats, pigs and poultry generally termed as **pasteurelloses**

- The most important ones are **Hemorrhagic septicemia** (cattle and other animals), **Pneumonic Pasteurellosis** or shipping fever (sheep and cattle) and Avian Pasteurellosis or Fowl cholera in **poultry**

3.1.7.1. Aetiology

- **Pasteurella spp.: commensals** of the upper respiratory tract (naso-pharnyx) of many animal species
- **Pasteurella** is gram negative, cocco-baccilli, **biporal staining** with blue color organism
- *Pasteurella multocida* is a heterogeneous species of gram-negative bacteria, there are a number of immunologically distinct types (serotype) of this spp. (*Pasteurella multocida*)
  - These have been classified as types **1** (or **B**), 2 (or **A**), **3** (or **C**), and **4** (or **D**) and there is a loose relationship between the serotype and the host species
  - *Pasteurella multocida* is responsible for two major disease conditions of cattle: Hemorrhagic Septicemia (caused only by type 1 or B) and Pneumonic Pasteurellosis (**2** or **A**)

- **Pneumonic Pasteurellosis of cattle**, commonly associated with infection by *Mannheimia* (formerly *Pasteurella*) *haemolytica* biotype A serotype 1(i.e. *Mannheimia* (*Pasteurella*) *haemolytica* biotype A serotype 1 is the most common cause of the pneumonia)

- **Eleven serotypes** of *Mannheimia* (*Pasteurella*) *haemolytica* have been demonstrated

- Pasteurella spp. are usually secondary causes of diseases

Name of the disease	Species affected	***Pasteurella spp.***
Septicemic Pasteurellosis **(Hemorrhagic Septicemia)**	Cattle	*Pasteurella multocida* type 1 (B)
Pneumonic Pasteurellosis **(Shipping fever pneumonia)** commonly associated with infection by *Pasteurella haemolytica* and also *Pasteurella multocida type A*	Cattle	▪ *Mannheimia* (formerly *Pasteurella*) *haemolytica* biotype A serotype 1 and ▪ *Pasteurella multocida* biotype A
Pasteurellosis	Sheep and goats	*Mannheimia haemolytica*
Pasteurellosis	Pigs	*Pasteurella multocida*

3.1.7. A. Septicemic Pasteurellosis (Hemorrhagic Septicemia)

- **Per acute,** highly fatal septicemic **disease of cattle** caused by ***Pasteurella multocida*** serotype 1(B)

- It is characterized by **Petechial hemorrhages all over the** serous membranes

Hemorrhagic septicemia

- Highly contagious bacterial disease of bovine
- Characterized by edematous swelling in neck region, pneumonia and wide spread of hemorrhage in visceral organs
- Caused by *Pasteurella multocida*

- **Haemorrhagic septicaemia** (**HS**) is a major disease of cattle and buffaloes characterised by an acute, highly fatal septicaemia with high morbidity and mortality

3.1.7.A.1. Transmission

- *Pasteurella multocida* is transmitted by **direct contact** with infected animals and on fomites

- **Cattle** and **buffalo** become infected when they **ingest or inhale** the **causative organism**, which probably originates in the nasopharynx of infected animals

    - The saliva of affected animals contains large numbers of pasteurella during the early stages of the disease

- In endemic areas, up to 5% of cattle and water buffalo may normally be carriers

- The **worst epidemics** occur during the rainy season, in animals in poor physical condition

- **Stresses** such as **a poor food supply** are thought to increase susceptibility to infection, and close herding and wet conditions seem to contribute to the spread of the disease

- **Rainy conditions** & **high humidity** facilitate transmission

3.1.7.A.2. Epidemiology

- **Hemorrhagic septicemia** occurs in cattle, yaks, camels, and water buffalo and, to a much smaller extent, pigs and horses

- It is considered economically important throughout the world including Africa

- **Animals of all ages** are susceptible but the most susceptible age group **is 6 months to 2 years of age**

- Both morbidity and case-fatality rates vary between 50% and 100%, and animals that recover require a long convalescence

- Outbreaks of the disease are often associated with wet humid weather during the rainy season

3.1.7.A.3. Pathogenesis

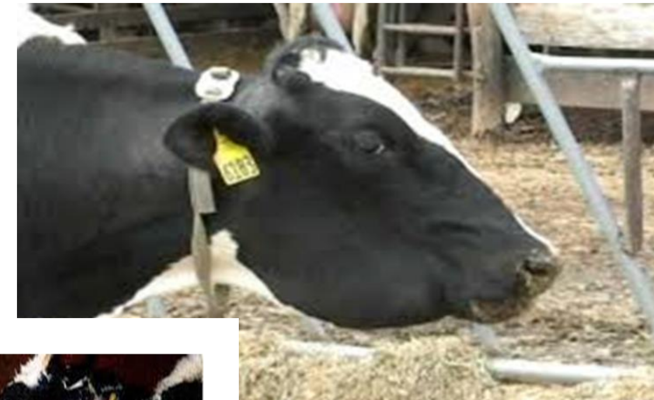
- The **portal of entry of infection** is thought to be the **tonsils**
- A fulminating **septicemia** occurs, which is associated with the capsular material of the organism
- The effects of the septicemia are most severe in the respiratory tract, heart, and gastrointestinal tract
- In cattle and **buffalo**, there is rapid translocation of bacteria from the respiratory tract to the blood, liver, and spleen, suggesting that the bacteria are able to invade via the mucosal epithelial layers

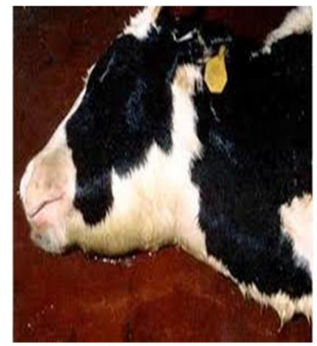
Hemorrhagic septicemia

Pathogenesis

- The organism is a normal inhabitant in the nasopharyngeal mucosa.
- Impaired local or systemic defense mechanism (stress, transportation, bad environment, crowding)
- Invasion → Proliferation of the m.o → of the mucosa to blood → Septicemia

3.1.7.A.4. Clinical Findings





- **Incubation period:** 3 to 5 days,
- **Most cases** in cattle and buffalo are acute or peracute
- **Dullness** & **reluctance** to move
- **Profuse salivation** and a serous nasal discharge (**respiratory distress occurs**)
- Severe depression and **sudden death** about 24 hours
- **Warm**, **painful swellings** about the throat, **dewlap**, **brisket** or perineum (i.e. **oedematous swellings** become apparent in the pharyngeal region; these **swellings spread** to the ventral cervical region and brisket)
- **Severe dyspnea** may occur if the respiration is obstructed
- **Mucous membranes** are congested

3.1.7.A.5. Necropsy Findings

- **Petechial hemorrhages** all over the serous membranes
- Accumulation of bloody stained fluid (serosanguinous fluid) in body cavities
- Acute hemorrhagic gastroenteritis
- **Edema of the lungs** and lymph nodes
- Generalized congestion, thickening of the interlobular septa of the lung may be prominent
- Enlarged and hemorrhagic lymph nodes of the thoracic region

3.1.7.A.6. Diagnosis

- Some characteristic epidemiologic and clinical features aid in the recognition of **Hemorrhagic septicemia** (HS)
  - Of particular significance is **a history of earlier outbreaks** and **a recent failure to vaccinate**
  - The season of the year, rapid course, and high herd incidence & **case fatality**, with fever and oedematous swellings in the throat, neck & brisket indicate typical
- Characteristic necropsy lesions support the clinical diagnosis;
- Confirmation requires (laboratory tests) the isolation and characterization of the pathogen using conventional and molecular techniques
  - **Gram/ Methylene blue staining**: blood or tissue smears reveal a gram–negative, short bacillus or ovoid with bipolar staining

3.1.7.A.7. Treatment, Control & Prevention

Treatment:

- **CAF** is the best drug which acts on gram-negative bacteria
- Oxytetracycline at high dose has been also be effective in pigs
- **Sulfadimidin**

Control: Vaccination

- For cattle *Pasteurella multocida* type-B killed organisms (Killed vaccine aluminum adjuvated)
    - 20 billion germs/ml killed by formalin (0.5% final concentration) & precipitated by 1% aluminum potassium sulphate (**oil adjuvant vaccine** (OAV) given at 2ml SC gives solid immunity up to 1 year

3.1.7.B. Pneumonic Pasteurellosis (Shipping Fever)

- **Pneumonic Pasteurellosis**: disease complex resulting from invasion of lung by *Mannheimia* (formerly *Pasteurella*) *haemolytica* & *Pasteurella multocida*, which are commensal organisms of tonsils & nasopharynx and the disease occur when respiratory tract defense mechanisms broken

- **Pneumonic Pasteurellosis** is **characterized** by **fibrinous bronchopneumonia**
  - The Pasteurella spp. are the main cause of the pneumonia but viruses or mycoplasmas may act synergistically to allow the bacteria to be pathogenic
  - There is often a history of stressors such as:
    - Usually following **transportation** (shipping fever)
    - **Mixing of groups of cattle** from different sources
    - Confinement of cattle
    - Ineffective housing and ventilation

- **Pneumonic Pasteurellosis of cattle**, commonly associated with infection by *Mannheimia* (formerly *Pasteurella*) *haemolytica* biotype A serotype 1, and *Pasteurella multocida* biotype A (i.e. *Mannheimia haemolytica* the **primary** agent involved in **Pneumonic Pasteurellosis**)

- Pasteurellosis of pigs, this is usually associated with infection by *Pasteurella multocida* and is mainly **pneumonic in form**

- Pasteurellosis of sheep and goats is usually associated with infection by *Mannheimia haemolytica* and, although it is often **pneumonic in form**, a septicemic form of the disease is common, especially in lambs

Name of the disease	Species affected	*Pasteurella spp.*
Septicemic Pasteurellosis (Hemorrhagic Septicemia)	Cattle	*Pasteurella multocida* type 1 (B)
Pneumonic Pasteurellosis (Shipping fever pneumonia)	Cattle	▪ *Mannheimia* (formerly *Pasteurella*) *haemolytica* biotype A serotype 1 and ▪ *Pasteurella multocida* biotype A
Pasteurellosis	Sheep and goats	*Mannheimia haemolytica*
Pasteurellosis	Pigs	*Pasteurella multocida*

3.1.7.B.1. Transmission

- **Transmission** occurs by the **inhalation of infected droplets** coughed up or exhaled by infected animals
- Infection originate from clinical cases or recovered carriers in which the infection persists in the upper respiratory tract
- The disease occurs when cattle are closely confined in inadequately ventilated barns, or when overcrowded in trucks and trains, or held for long periods in holding pens in feedlots
- **In animals at pasture**, the rate of spread may be much slower

3.1.7.B.2. Epidemiology

- **Pneumonic Pasteurellosis** is a common disease world wide
- The disease occurs **most commonly in young growing cattle** from **6 months to 2 years of age** but all age groups are susceptible
- The **morbidity** may reach 35%, **the case fatality rate** may range from 5-10%, and the **population mortality rate** may vary from 0.75-1 %
- The disease occurs in outbreaks 7-10 days after stressful transportation
- The mixing of cattle from different sources is an important risk factor

3.1.7.B.3. Pathogenesis

- The organisms are part of the normal flora of the upper respiratory tract, **colonize** first the upper respiratory tract then the lower respiratory tract

- Under normal conditions the bovine lung is relatively free of pasteurella organisms

- **Impaired defense mechanism** (transportation) **proliferate** in nasopharynx then invade the lung

- Alveolar macrophages will effectively clear pasteurella organisms from the alveoli by phagocytic mechanisms,

  - **However**, the **Pasteurella organisms** release endotoxins (**leukotoxin**) and (**cytotoxins**) that affects leukocytes & platelets, which causes rapid lysis & death of WBCs and **finally forms** fibrinous bronchopneumonia lesion & acute fibrinous pleuropneumonia

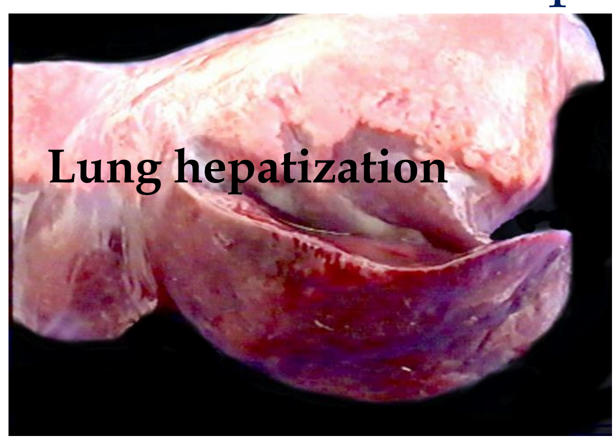
3.1.7.B.4. Clinical signs

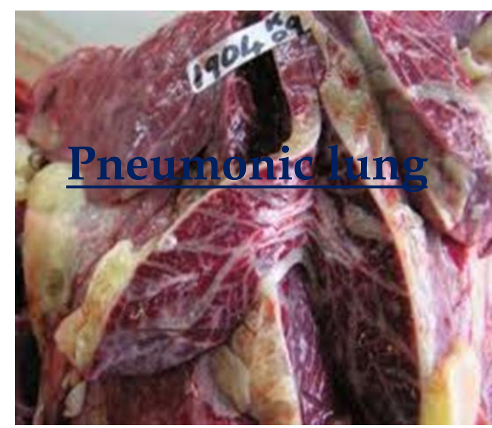
- The disease usually occurs within 10-14 days after the animals have been stressed
- Sudden death
- Depression and rapid and shallow respiration
- Coughing, which becomes more pronounced and frequent if animals are urged to walk
- Mucopurulent nasal discharge, **crusty nose**, and **an ocular discharge**
- lowered or drooped head & ears
- Labored & rapid breathing
- Anorexia
- Fever
- A mild diarrhea in some cases

3.1.7.B.5. Necropsy Findings

- Marked pulmonary consolidation (i.e. Reddish black to grayish brown consolidated areas in the lungs)
- Gelatinous thickening of the interlobular septa
- Areas of necrosis with white boundaries &deep central red zone
- The lung is firm and the cut surface usually reveals an irregular, variegated pattern of red, white, and gray tissue due to hemorrhage, necrosis, and consolidation
- Classical lesion is sever fibrinous bronchopneumonia

(Courtesy College of Veterinary Medicine, University of Illinois.)





3.1.7.B.6. Diagnosis

(1). Bacterial culture [culture tracheal aspirates, lung lesion at necropsy, milk (mastitis)]& **species identification** by biochemical tests

- All species can be isolated on blood agar (haemolysis)

- **Blood smears** (septicemia)

(2). Serological tests (indirect haemagglutination is the commonest)

(3). Molecular Diagnosis

3.1.7.B.7. Differential Diagnosis

- Contagious Bovine pleuropneumonia (CBPP)
- Verminous pneumonia
- Sporadic conditions, such as lung abscesses & aspiration pneumonia

3.1.7.B.8. Treatment

Antimicrobial therapy:

- About 85-90% of affected cattle recover within 24 hours if treated with antimicrobials
  - Drugs like Oxytetracycline, Tilmicosin, Sulfonamides, and Penicillin can be used
  - Broad-spectrum antimicrobials are used most commonly

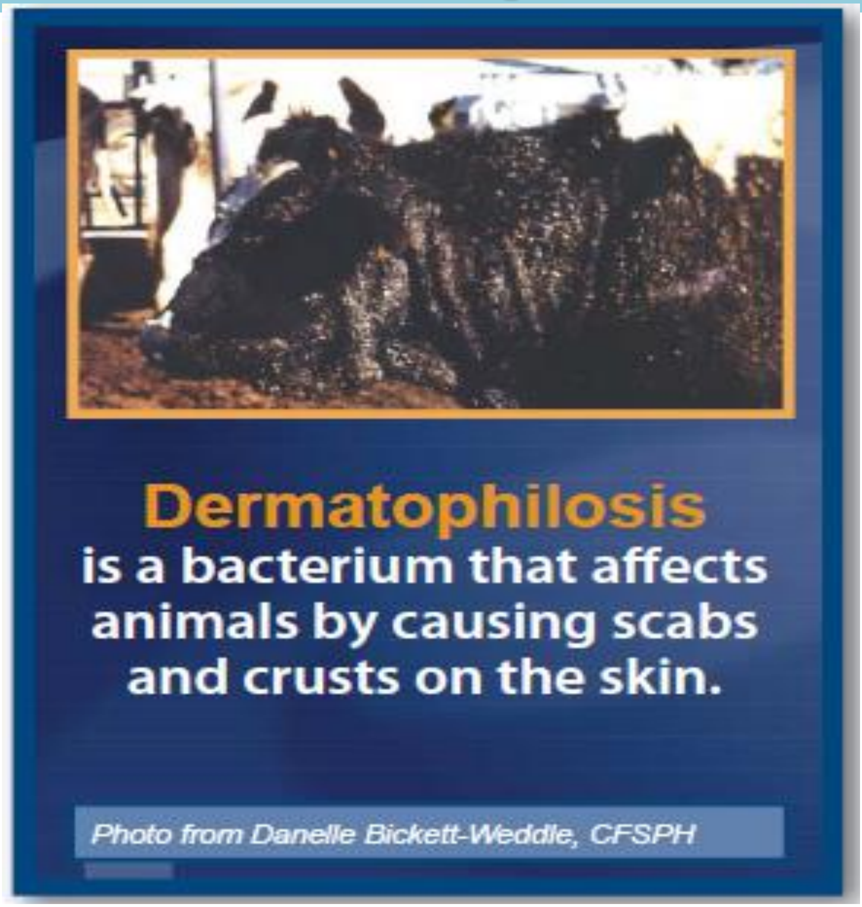
Anti-inflammatory agents:

- Corticosteroids and nonsteroidal anti inflammatory drugs (NSAIDs) are used as an ancillary treatment for severe cases

3.1.7.B.9. Control

- Control **depends on** management, the use of vaccines and antimicrobials
  - **Weaning at least 2 weeks** before shipment was also considered a desirable practice
  - **Vaccinating the weaned-calf** for all the commonly anticipated diseases before weaning
  - Vaccination should be twice at a 14-day interval with the *Mannheimia haemolytica* bacterial extract and genetically attenuated leukotoxin vaccine (for feedlot cattle)
  - **Chemoprophylaxis:** antimicrobials (**oxytetracycline**) are used for the **control** of **pneumonic Pasteurellosis**, particularly in cattle that have just been introduced

3.1.8. Dermatophilosis



DERMATOPHILOSIS (MYCOTIC DERMATITIS, CUTANEOUS STREPTOTRICHOSIS, SENKOBO DISEASE OF CATTLE, LUMPY WOOL OF SHEEP)

The disease in sheep is commonly called mycotic dermatitis, in cattle cutaneous streptotrichosis, although other local names exist including Senkobo skin disease in Central Africa, Kirchi in Nigeria, and Saria in Malawi. Dermatophilosis is a name common to the disease in all species.

What is Dermatophilosis and what causes it?

- Dermatophilosis is a skin disease caused by a bacterium called *Dermatophilus congolensis*
- The **disease can affect many species** of domestic and wild animals and **occasionally** humans

3.1.8.1. Transmission

- **Dermatophilosis** can affect cattle, **sheep**, **goats**, horses, and **less frequently** pigs, dogs, and cats

- Dermatophilosis can occur in animals of all ages but is more common in the young

- The organism that causes Dermatophilosis is found on the skin of diseased animals and also carrier animals that show no signs

Epidemiology Organism present in minor carriage lesions on face and feet. Serious disease occurs when body skin is broken by shearing or insect bites, or macerated by prolonged wetting, coupled with management practices that promote transmission. The disease has significant importance in cattle in tropical areas and occurs mainly in sheep and horses in high rainfall areas in temperate climates.
In tropical areas ticks promote severe infection in cattle by suppression of immune function

- **Spread of the disease occurs by direct contact** between animals or through exposure to contaminated surroundings (fomites) or by biting insects (vectors), particularly flies and ticks

- **Factors that break-down the natural protective barriers of the skin** such as prolonged wetting by rain, high humidity, and high temperature allow the bacteria to spread

3.1.8.2. Clinical findings

Cattle

The early lesion is a pustule and the hair over the infected site is erect and matted in tufts (**paintbrush lesions**) with greasy exudate forming crumbly crusts which are hard to remove. These develop to scabs which are greasy and fissure at flexion points, and finally to scabs that are hard, horny, and confluent. The scabs vary in color from cream to brown, are 2–5 cm in diameter and are often in such close apposition that they give the appearance of a mosaic. In the early stages the crusts are very tenacious and attempts to lift them cause pain. Beneath the crusts there is granulation tissue and some pus. In the later stages, the dermatitis heals and the crusts separate from the skin, are held in place by penetrating hairs, but are easily removed.

cattle

Lesions occur on the neck, body, the back of the udder and may extend over the sides and down the legs and the ventral surface of the body. Commonly they commence along the back from the withers to the rump and extend halfway down the rib cages. In some animals the only site affected is the flexor aspect of the limb joints or the inguinal area or between the forelimbs.

In young **calves**, infection commences on the muzzle, probably from contact with the infected udder or because of scalding by milk in bucket-fed calves, and may spread over the head and neck.

- Sheep: hard crusts distributed over backline palpable in fleece
- Cattle and horses: non-pruritic crusting dermatitis

3.1.8.3. Diagnosis, Treatment

Diagnosis:

- **Diagnostic confirmation** based on organisms in scrapings or biopsy sections, culture (i.e. Bacteriology - affected skin and draining lymph node)
- **Histology** - formalin-fixed samples of these tissues

Treatment:

- Antibiotic treatment at high dose for a single treatment is effective in reducing the proportion of active lesions in an affected flock
- Antibiotics that are effective include procaine penicillin combined with streptomycin at a dose of 70 000 units/kg and 70 mg/kg, respectively, erythromycin at 10 mg/kg, **long acting tetracycline** at 20 mg/kg and combination of **lincomycin** and **spectinomycin** at a dose of 5 mg/kg and 10 mg/kg, respectively

3.1.8.4. Differential Diagnosis & Control

DIFFERENTIAL DIAGNOSIS

- Ringworm
- Staphylococcal dermatitis/folliculitis
- Scabies
- Pediculosis
- Fleece rot – sheep.

Control:

- The best methods to control infection are isolating infected animals, culling those that are chronically ill, and controlling external parasites (flies, ticks)
- Additionally, affected animals can be given antibiotics or treated externally (i.e. use of topical bactericides to prevent infection of shear cuts, and of skin in risk periods)
- Avoidance of skin trauma and of management practices that promote transmission
- Acaricides i n cattle
- There is no vaccine for Dermatophilosis

Group Assignment

- Prepare **PowerPoint presentation** (maximum of 10 slides) of **10 minutes** presentation
- The presentation is followed by 10 minutes question and **answer session**
- Each group will be evaluated on content, presentation and accuracy of answers
- **Date of Presentation: 12 June, 2019**

Group_1 **Mastitis**	Haimanot Girma (1902/18) Zewuditu Fittu (1909/06) Demeke Getachew (1898/06)
Group_2 **Paratuberculosis**	Fikru Hunde (1900/06) Tariku Dilecha (1905/06) Yeshiharg Abebe (1907/06)
Group_3 **Actinobacillosis**	Zahara Mohamed (1908/06) Endashaw Tadesse (1899/06) Getachew Deresu (1901/06)
Group_4 **Actinomycosis**	Ali Shiferaw (1896/06) Seefu (1903/06) Baisa Fekensa (1897/06


Thank you

COLLEGE OF VETERINARY MEDICINE

COURSE---FUNDAMENTALS OF FARM ANIMAL DISEASE / VLT 4141

TARGET STUDENTS --- VLT—III

Prepared by Abdi F. (Asst. prof)

email-abdikoo68@gmail.com or abdi.feyisa@aau.edu.et

Learning objectives:

At the end of this session you are able to:

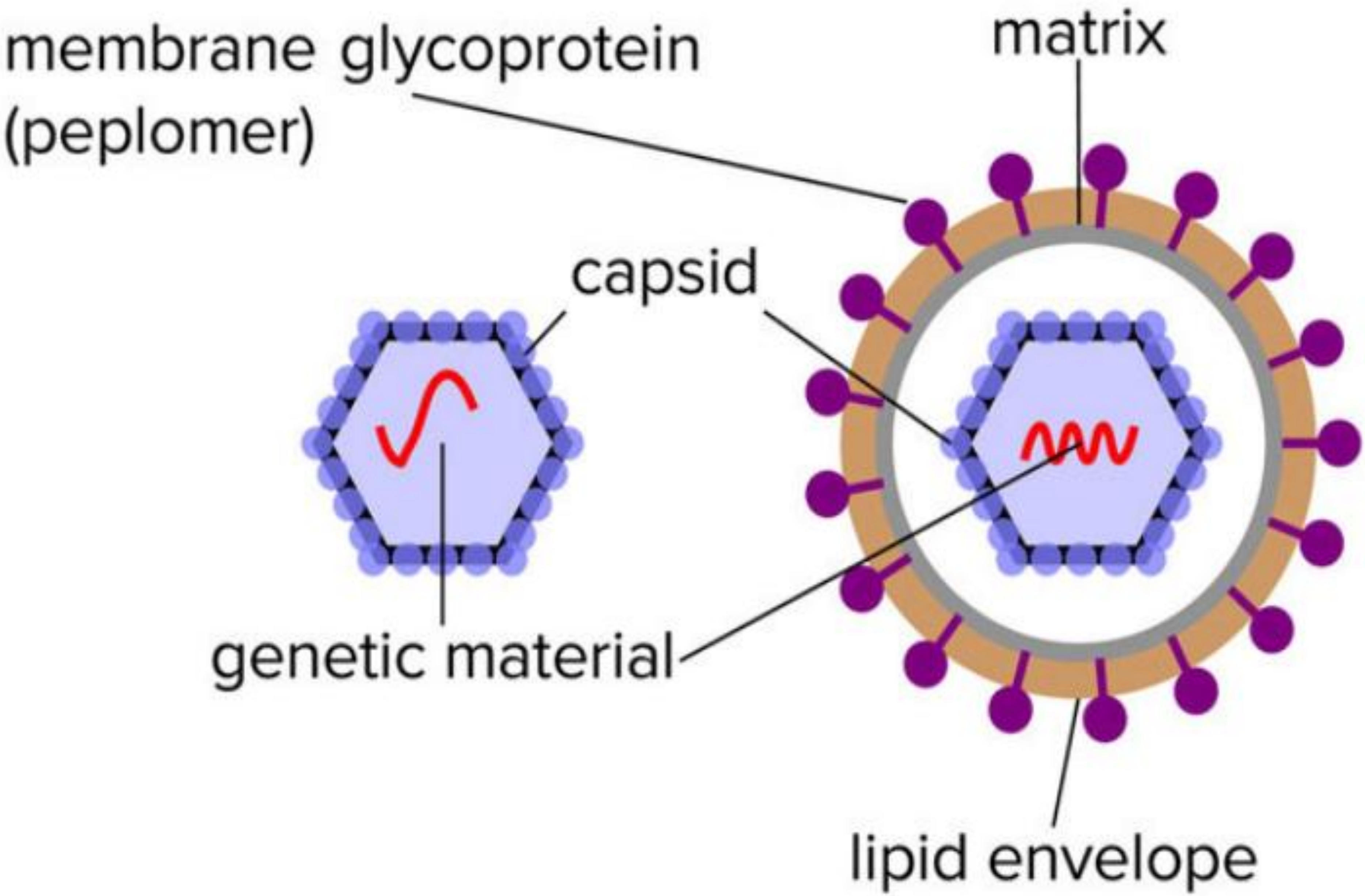
- Describe common and economically significant viral diseases of farm animals (medical history and clinical findings)
- Identify representative samples to be collected for the proof of causality for each diseases
- Describe appropriate laboratory diagnosis/ tests for each diseases
- Collect samples and process in laboratory

VIRAL DISEASES OF FARM ANIMALS

INTRODUCTION

What is virus ?

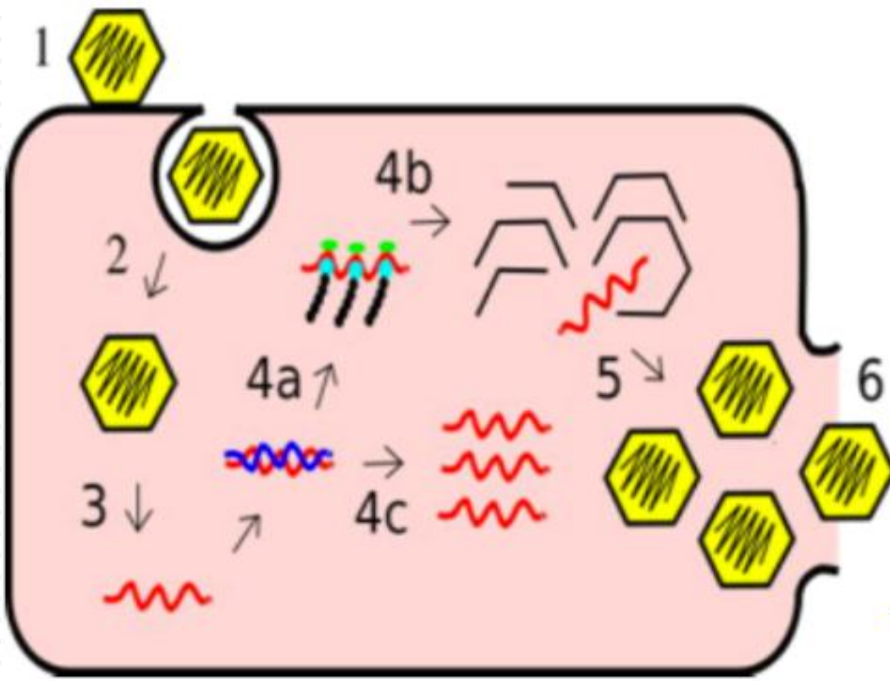
- Viruses are **obligate intracellular small infectious agent**
- Made of a core of nucleic acid (DNA or RNA) surrounded by a protein coat or capsid and in addition some have envelopes.
- Unlike bacteria or fungi virus cannot replicate on inert media; **viable host cells** are required for replication.
- This present a greater therapeutic challenge than do bacteria.
- Drugs that target viral diseases must penetrate host cells; in doing so, they are likely to disrupt normal cellular activities


membrane glycoprotein
(peplomer)
matrix
capsid
genetic material
lipid envelope

Int cont'd

Viral replication:

- penetration into susceptible host cells
- Un coating of viral nucleic acid
- Synthesis of early regulatory proteins
- Synthesis of RNA or DNA
- Synthesis of late regulatory proteins
- Assembly (maturation) of viral particles
- Release from cells



Int cont'd

- Most infectious diseases (*including viral infections*) are a major limiting factor in animal producing parts of the world alike Ethiopia.
- For instance: rinderpest (eradicated from Ethiopia), foot-and-mouth disease (FMD), Lumpy skin disease, sheep pox goat pox and Peste des petis ruminants are all viral infectious diseases having limitations on livestock production and lead to
  - shortages of meat, milk, draught animals, and manure,
  - Necessitate import from developed countries ,
  - impose trade embargo on affected countries

discourage domestic livestock production.

Int cont'd

- What viral disease of farm animals do you know ?

- Mention some representative samples and appropriate Lb. DX/tests to be done


USDA

1. Foot and mouth diseases (FMD)

- Foot-and-mouth disease (FMD) is a **severe**, clinically **acute, vesicular disease of cloven-hoofed animals** including domesticated ruminants, pigs and wild life.

- FMD is **endemic** in large areas of Africa (**including Ethiopia**), Asia and South America and has shown an extraordinary ability to cross international boundaries and cause **epidemics** in previously free areas,

- Is among **OIE Listed disease** (potential for rapid and extensive spread **within and between** countries and can cause severe economic impact).

FMD Cont'd

Etiology- it is caused by foot-and-mouth disease virus **(FMDV)**, which is classified within the **Aphthovirus genus** as a member of the **Picornaviridae family**.

- **FMDV** has **seven distinct serotypes** with indistinguishable clinical effects, namely type **O, A, C**, Southern African Territories **(SAT) 1, SAT 2, SAT 3 and Asia 1.**
- Where serotype **A, O, C, SAT-1 and SAT-2** are currently recognized in Ethiopia.
- Each serotypes have distinct **topotypes and lineages.**
- There is no cross-immunity between serotypes and even within the same strain . **This presents difficulties to vaccination programs.**

FMD Cont'd

Sources of infection and transmission;

- Vesicular fluid or epithelium
- Meat and by product in which PH has remained above 6.0
- Incubating and clinically affected animals
- Breathe, saliva, feces and urine, milk and semen
- Carrier animals: particularly cattle and water buffalo(virus persist on orophrynx for up to 30mths in cattle or longer in buffalo, 9mth in sheep)

FMD Cont'd

Transmission;

1. Direct and indirect contact.

A. Direct contact

- Is the **most common** mechanism of spread of **FMD**
  - **mechanical transfer** of the virus from infected to susceptible animals,
    - **through cuts or abrasions or through the mucosae,** or
  - infection by the deposition of droplets or droplet-nuclei **(aerosols)** in the respiratory tract of recipient animals.

FMD Cont'd

B. Indirect contact

- via contaminated personnel, vehicles, and all classes of fomites.
- Iatrogenic

FMD Cont'd

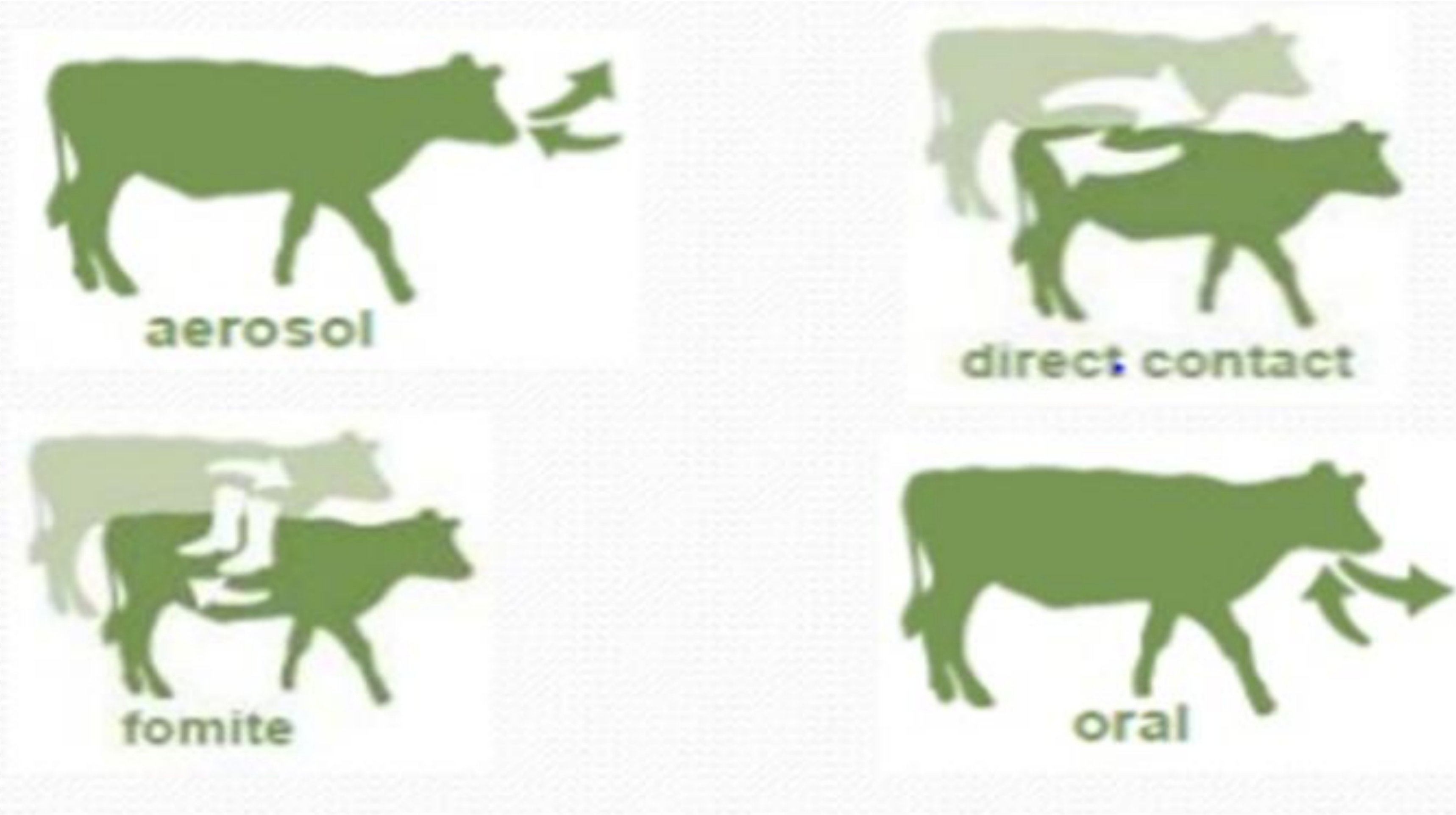
2. Airborne transmission.

- Droplet and droplet nuclei may be extended to **long-range airborne transmission by the aid of wind.**
  - significant when pigs are the source of infection because, they liberates the largest quantities of airborne virus.

3. Transmission by the oral route.

- Feeding of **contaminated animal by products and**
- Contaminated feeding and water **troughs** with excretions,

FMD Cont'd



FMD Cont'd

Pathogenesis

- **Previremic phase:** primary exposure to live virus results in the establishment of infection and accumulation of FMDV in the non cornified epithelium of the **pharyngeal area** **(also known as primary site of viral replication).**

- **Viremic phase:** Virus then spreads through regional lymph nodes and via the bloodstream to **cornified epithelium** of the **mouth and feet, the dorsum of the snout of pigs, and the teats**, resulting in several cycles of **viral amplification** and spread. Characteristic lesions develop at these sites

- **Postviremia / convalescent:** phase including resolution of clinical disease that may result in **long-term persistent infection**.

FMD Cont'd

Clinical signs of FMD

- The first signs of illness usually appear within 2 to 14 days.

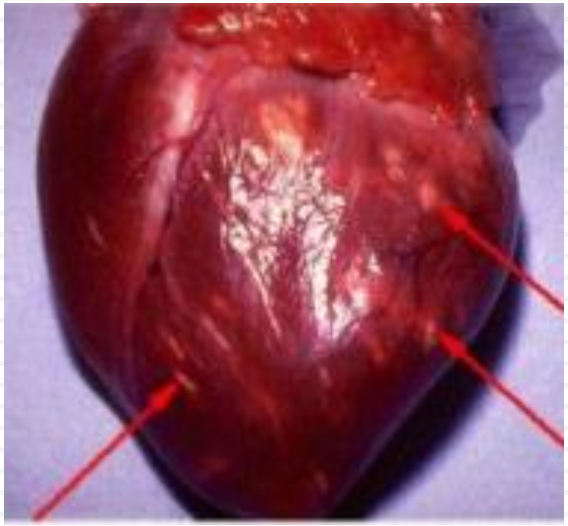
- Animals with FMD typically have a **high fever (40° to 41° C) and blisters** on the **tongue and lips, in and around the mouth, on the mammary glands, and around the hooves**.

- These blisters, called **vesicles** later turn into **red areas** called **erosions**.
- Pain and discomfort from the **vesicles and erosions** lead to other symptoms such as;
  - **depression,**
  - **anorexia,**
  - **excessive salivation,**
  - **lameness, and**
  - **reluctance to move or stand.**

FMD Cont'd

- Most affected animals will not die from FMD, but the disease leaves them weakened and unable to produce meat and milk the way they did before.

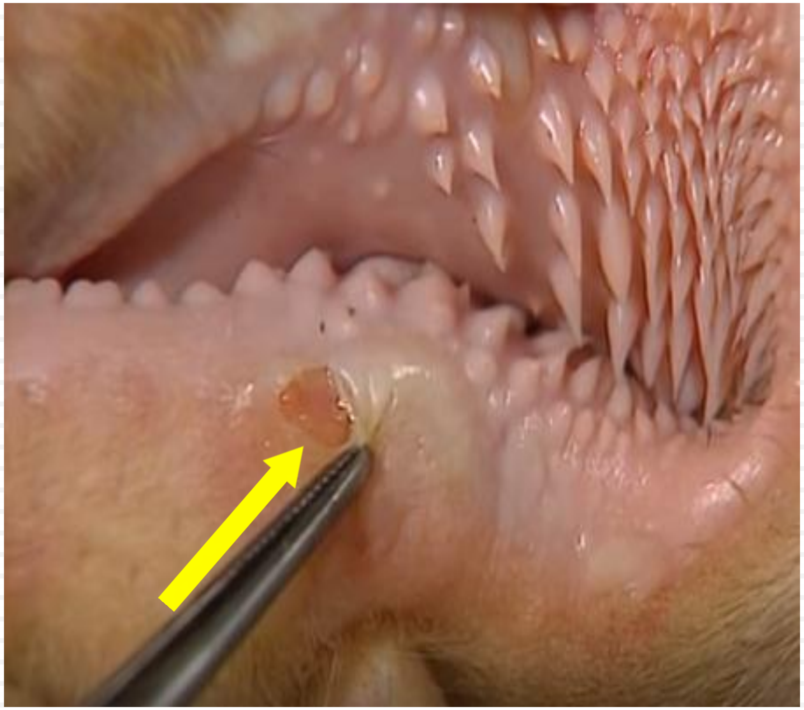
- Often young animals will die due to myocarditis some times called "tiger heart"



Necrosis of heart muscle of young animal "tiger heart"

FMD Cont'd

Vesicle on the snout of pig



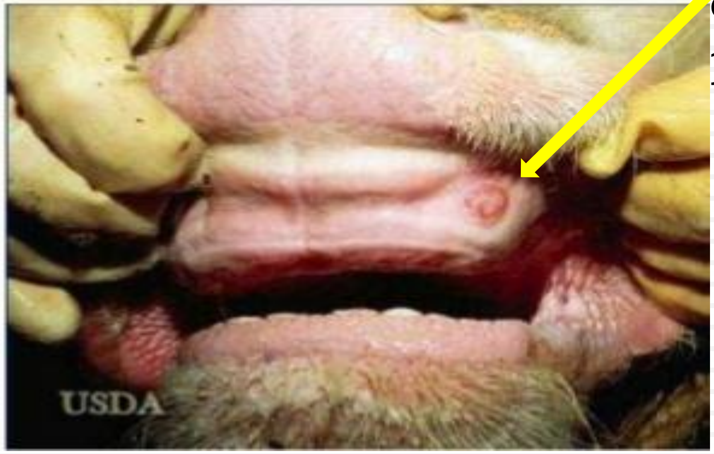
Vesicular fluid

Ruptured vesicle at the end of teat

FMD lesion in the inter digital space

Erosive lesions on the tongue

FMD Lesion on the dental pad

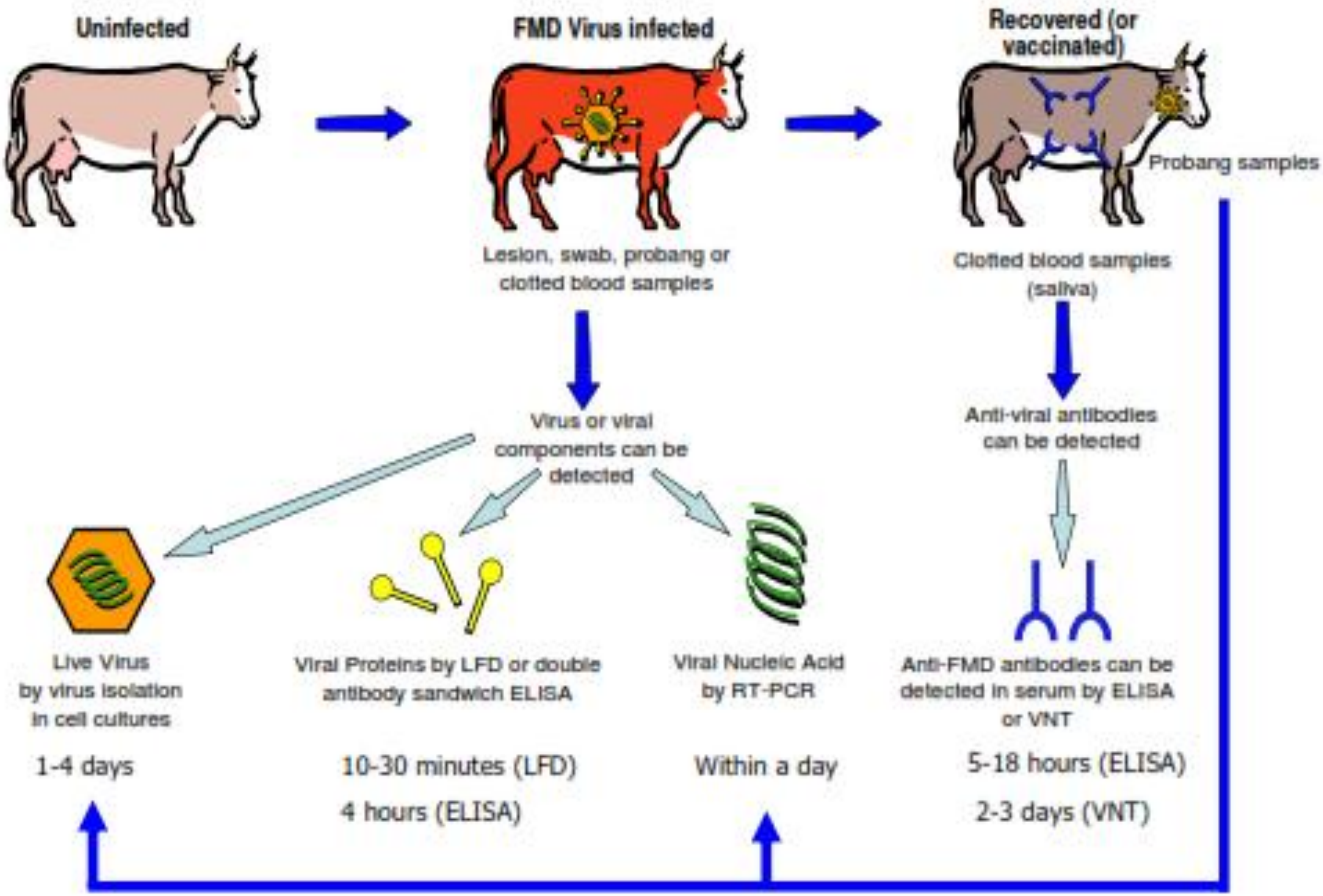


FMD Cont'd

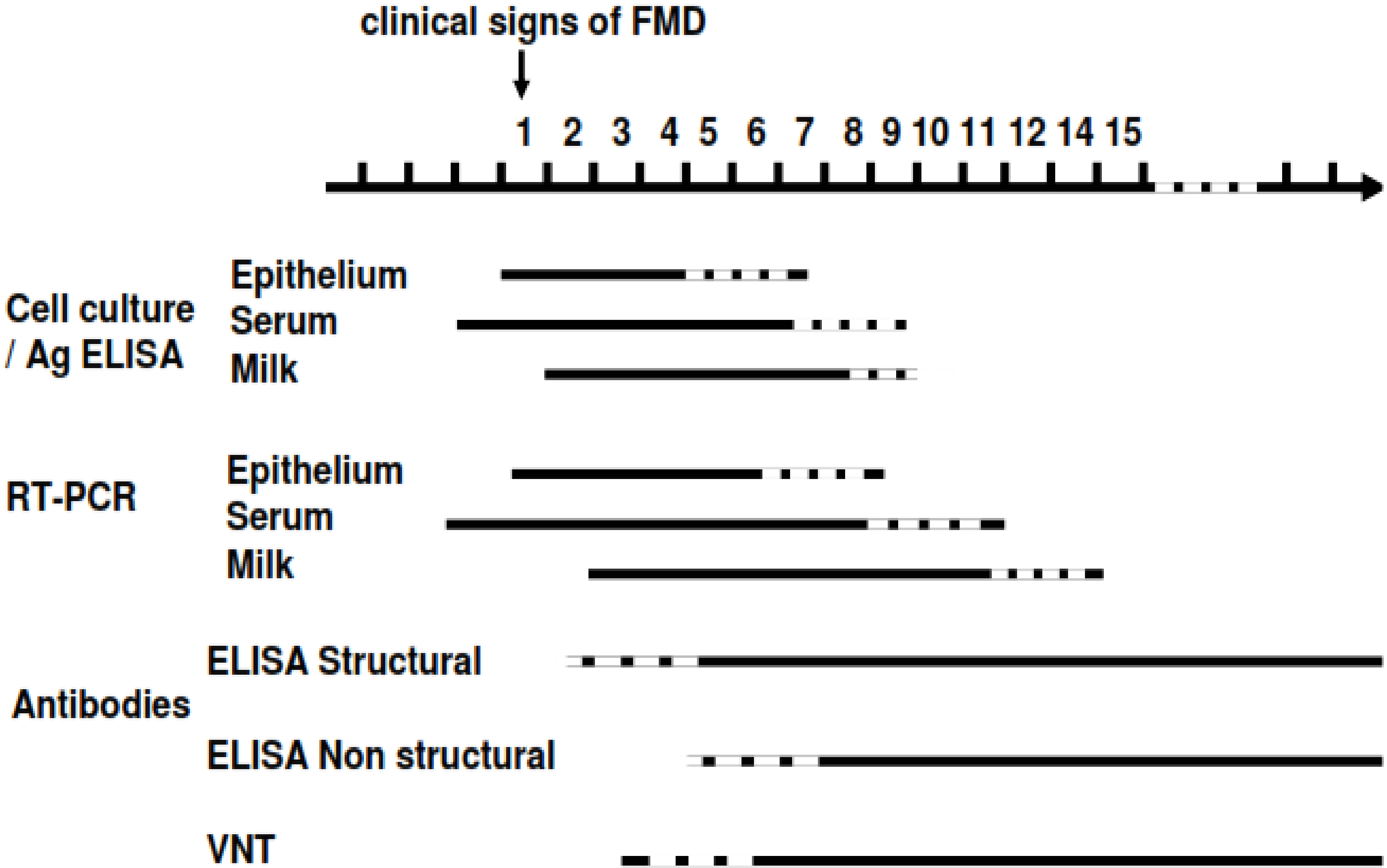
Differential Diagnosis

- viral vesicular diseases, including
  - swine vesicular disease
  - vesicular stomatitis

Principals of FMD Diagnosis



FMDV diagnosis: Window of detection by different techniques/tissues



FMD Cont'd

1. Representative samples;

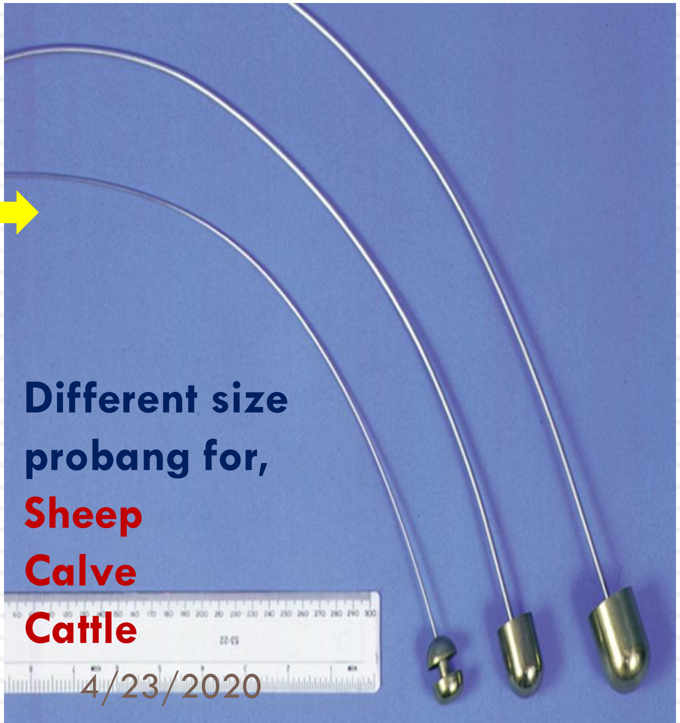
- Surrounding epithelial surfaces (tongue, buccal cavity and foot) (Up to 2cm2 or 1g is ideal)
- Fresh vesicular fluid Samples (not more than 5 ml).
- Swabs from mucosal surfaces (oral or nasal swabs)
- Blood
- Milk
- Oropharyngeal samples
- Heart and other organs in fatal cases

FMD Cont'd

2. Materials required for collection, preservation and transportation;

- Transport medium composed of equal amounts of glycerol and 0.04 M phosphate buffer, pH 7.2 to 7.6, or glycerol and phosphate buffered saline (PBS) for laboratory tests,
- Ice carrier
- Probang
- plain tube

an instrument used to collect op sample



FMD Cont'd

3. Laboratory diagnosis /tests

A/ Identification of agents (AG) in tissue or fluids

- Sample:[epithelium & vesicular fluid]

1. Virus isolation or cell culture: takes 1-4 days

- Suspected sample inoculated into primary cell culture or cell line or unweaned mice (2 to 7 days old) → Examined for cytopathic effect (CPE) after 48 hours. If no CPE is detected the procedure repeated for another 48 hours.
  - **BHK-21 cell line, primary bovine thyroid (BTY) cells and pig kidney cells are used**
- neutralization of the virus by known antisera makes the technique highly efficient and specific.

FMD Cont'd

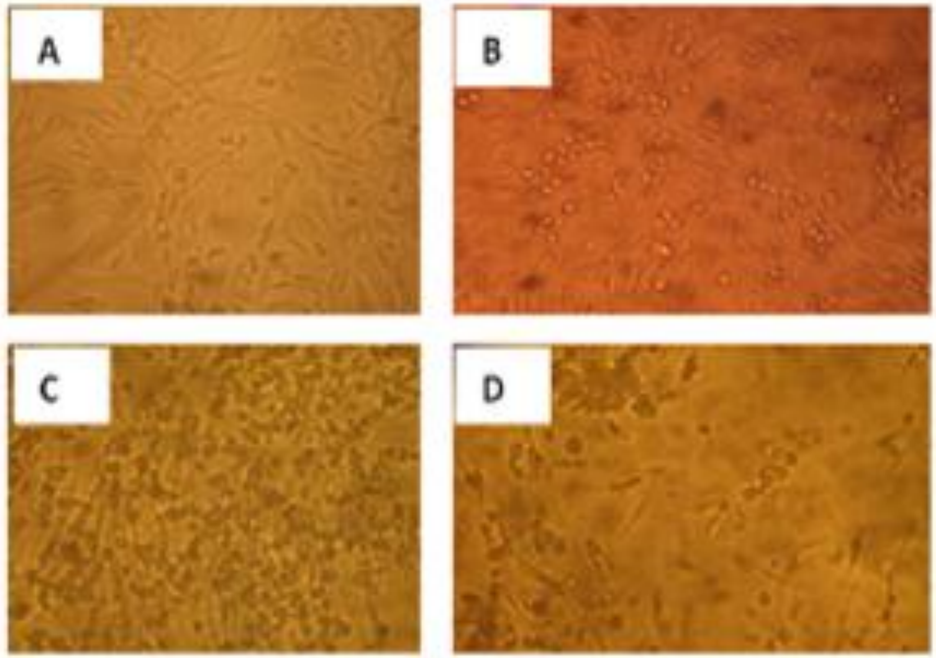


Figure Normal BHK-21 cell line (A). FMD infected BHK-21 cell (stage 1: initiation of infection and cell rounding started, photo taken after 12 hours,40x). (B).FMD infected BHK-21 cell (stage 2, almost 100% cell infected, photo taken after 24 hours of infection,40x). (C).FMD infected BHK-21 cell (stage 3). (D).

FMD Cont'd

2. Immunologic methods:

- Enzyme-linked immunosorbent assay (ELISA):

  - Sandwich ELISA is the preferred test carried out for the detection and typing of specific FMDV antigens in epithelial tissue suspensions (directly on samples from field materials).

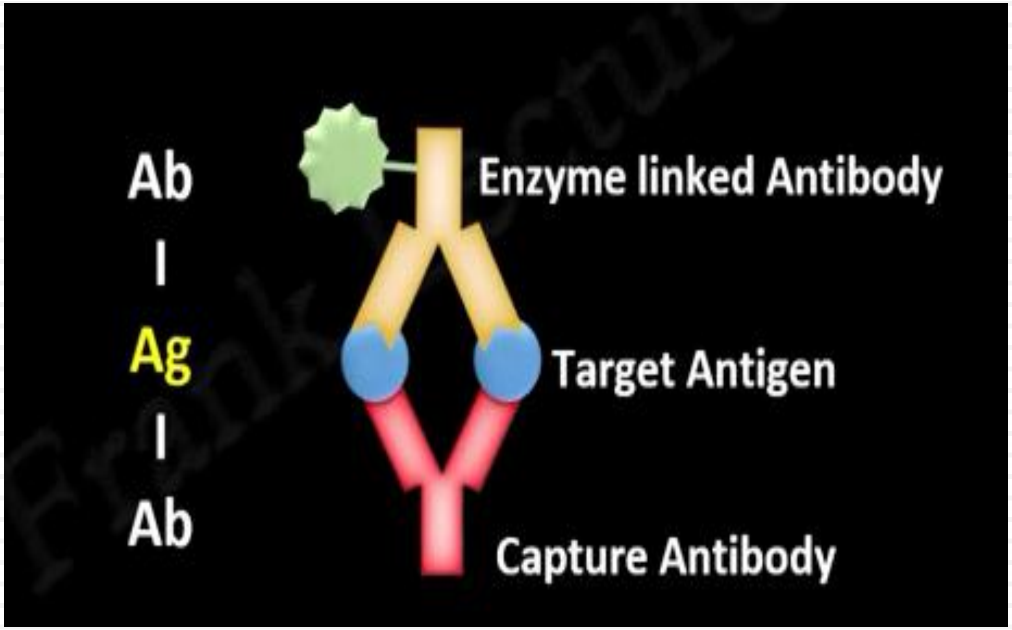
  - it is usually accompanied by concurrent cell culture isolation and the application of ELISA to any samples showing a cytopathogenic effect.

  - ~ takes up to 4hrs

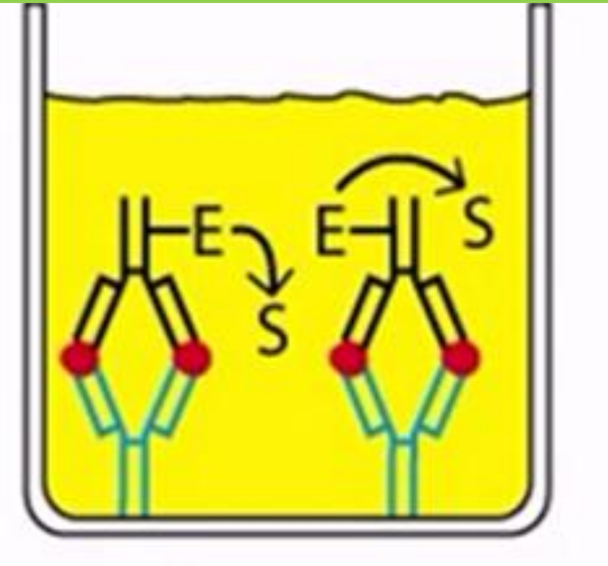
Sandwich ELISA

- Principle- detect anti-gene in suspected samples via Ab-Ag-Ab—rxn; enzyme substrate rxn and color production

Components of sandwich ELISA



+ve samples after Substrate added



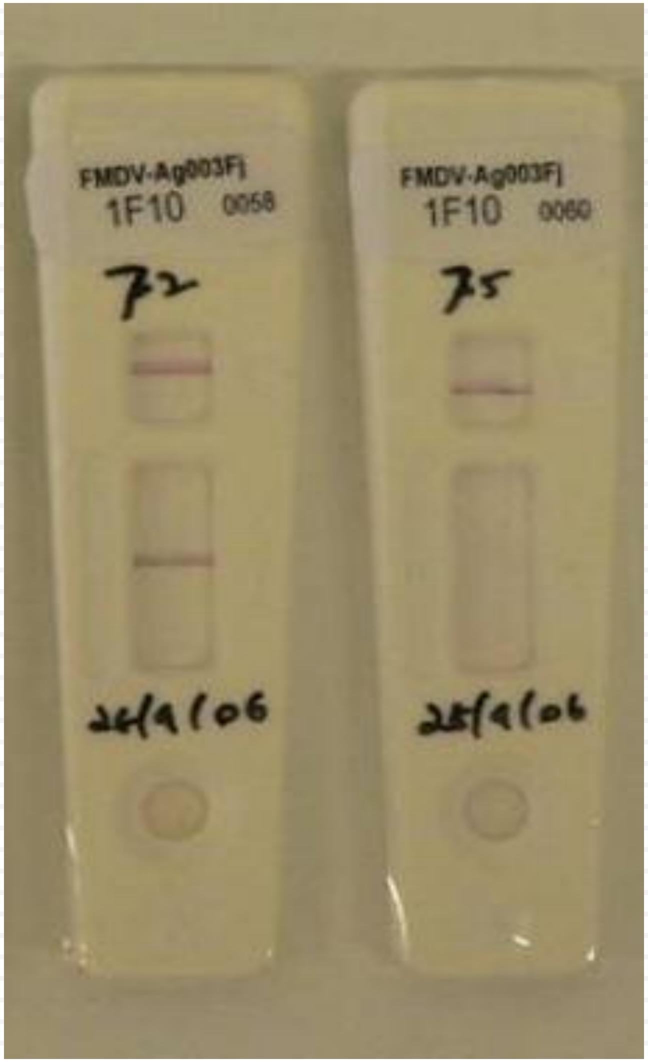
FMD Cont’d

- lateral flow immunochromatographic (LFI) strip test

  - This ***‘Pen-side’ test*** is developed to diagnose FMD **antigen** based on a **monoclonal antibodies** that reacts against FMDV of all seven serotypes.

    - Used to test epithelial suspensions or vesicular fluid.
    - The procedure takes **only 10 minutes** and can be **done on–field**.

FMD Cont’d



FMD Cont'd

3. Nucleic acid recognition methods:

- These include **reverse transcription polymerase** chain reaction (RT-PCR) which **amplifies** fragments of **FMD genome** in samples and can be **used for typing**. It is more sensitive than ELISA. **Takes ~5hrs.**

FMD Cont'd

Components of VNT;
1. **Serum sample**
2. **Ag/ virus of known titer**
3. **Cell line/cell cultures**

B/Antibody detection

- **Virus neutralization test (VNT):**
  - **Sample: [blood/serum]**

- Antibody to FMD virus can be detected by the ability of a serum to prevent a CPE (cytopathic effect) when virus of known titer is added to cultures of susceptible cells in the presence of the serum.

- The VNT is now largely used as a confirmation test for sera found positive by ELISA and for import/export certification when importing countries specify the use of the VNT.

- it requires 48-72 hours to complete

FMD Cont'd

Table: 1 A summary of representative samples to be collected and preferred test s for FMD diagnosis

Samples to be collected	Transport/Collection	Tests which can be performed
1.Epithelium/Vesicular Fluid/Vesicular Swabs	Phosphate buffered gelatin saline (PBGS) or PBS	PCR, Antigen ELISA, Virus Isolation
2. Serum	Plain evacuated tube	PCR, Antibody ELISA, VNT
3. Oral & Nasal Swabs	Phosphate buffered gelatin saline (PBGS) or PBS	PCR, Antigen ELISA, Virus Isolation

Group Assignment

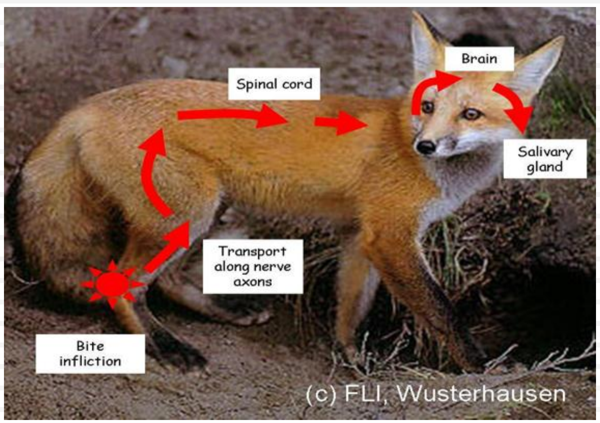
1. **Short Review on DIVA tests** used to differentiate between NS and S- proteins of FMD (Max pages=5)

Date of submission and presentation

Tuesday, March 10, 2020;Afternoon@2PM

Rabies

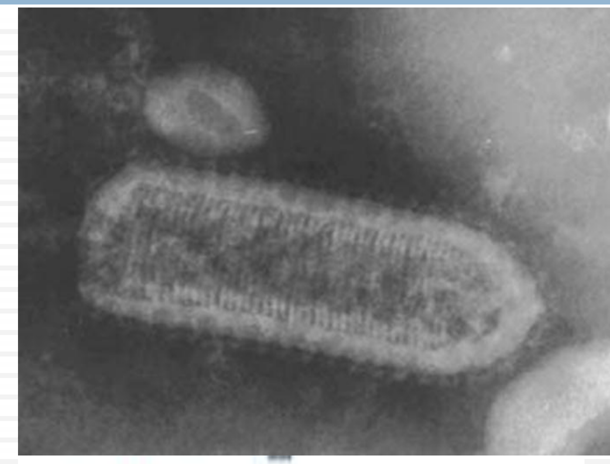
- Rabies is an **acute progressive viral encephalomyelitis** that **principally** affect carnivores and bats, although mammal can be affected.



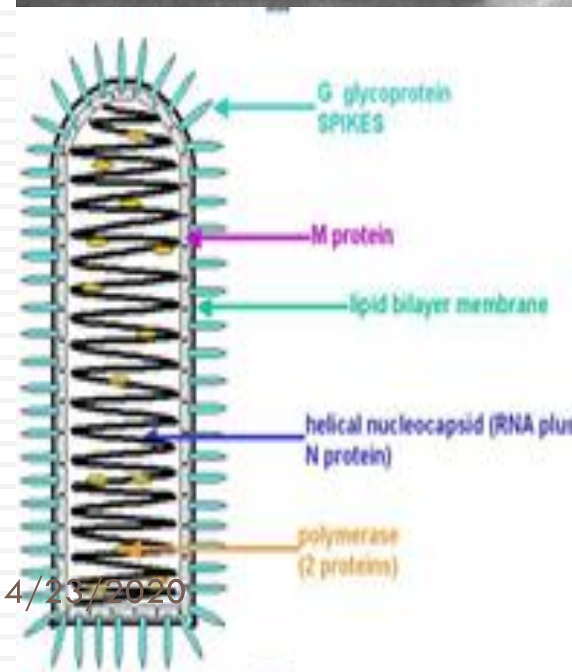
- It is almost **invariably fatal** once the clinical signs develop.
- Africa and Asia recorded **95%** of fatal cases of rabies.
- Globally **dog is the most important reservoir** particularly in developing countries

Rabies cont'd

Causative agents



- It is caused by **enveloped Single stranded RNA genome** viruses belonging to **lyssavirus genus** within **Rhabdoviridae family**
- **Bullet-shaped (75 x 180 nm)**
- Rabies viruses have about15 species; three of them were identified in Ethiopia so far;
  - **Rabies virus (genotype 1)**----the most common world wide and causative agent of **classical rabies**.
  - **Mokola virus feline origin** and
  - **Lagos bat virus dog origin**
- The virus is fragile and susceptible to most disinfectants



Rabies cont'd

Transmission

- The virus is predominantly transmitted by **infected saliva through bite or scratch,**
- Through **broken skin or mucus membranes** (eyes, nose and mouth)

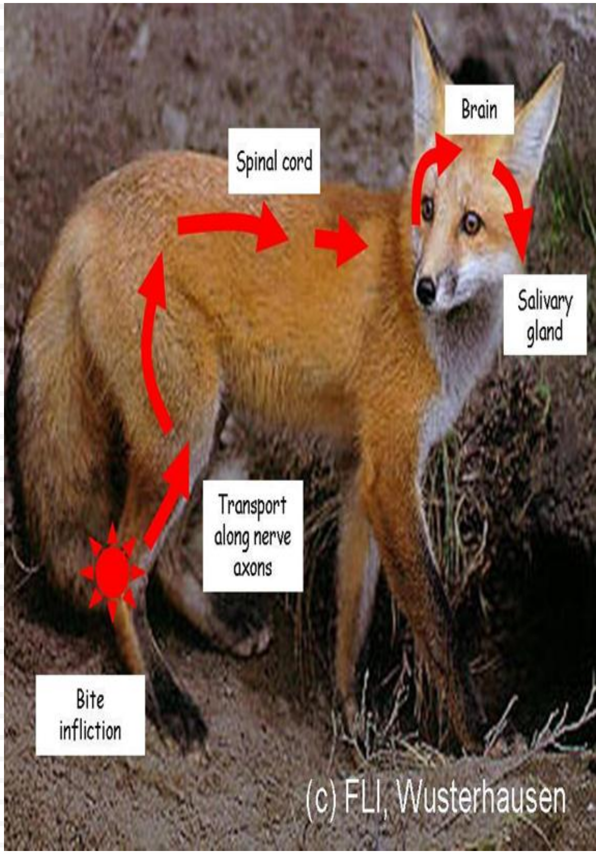
Rarely ;

- laboratory exposure (inhalation)
- Organ transplantation from infected individual
- aerosol transmission in bat cave

Rabies cont'd

Pathogenesis and clinical signs

- It is highly neurotropic
- once the rabies virus enters the body, it begins to multiply in the area near the entry site (bitten muscle),
- viral attachment to peripheral nerves to the spinal cord and ascending to the brain,
- The virus travels via peripheral nerves to salivary glands,
- Virus sheds intermittently in the saliva



Rabies cont'd

Clinical signs

- The most reliable signs, regardless of species, are
  - acute **behavioral changes** and
  - **unexplained progressive paralysis**

'furious' form

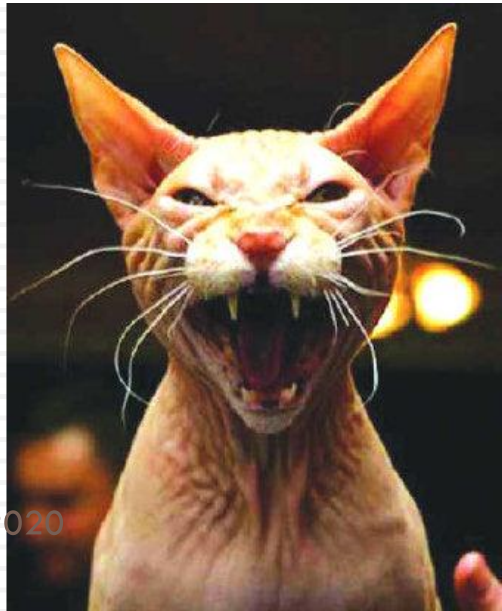
- nervousness,
- restlessness
- exaggerated response to stimuli.
- Self infliction of injury at the site of bite
- attack everything.
- Wondering here and there

Rabies cont'd

Dumb form

- the dog sit with its mouth open.
- unable to eat

(as if bone in its throat).



- The clinical signs of rabies in cats are similar to those in dogs.

- Rabid cats have a greater tendency to hide in secluded places and are often more vicious

Rabies cont'd

Diagnosis of rabies

1. Detecting virus (inoculation tests);

A/ Mouse inoculation test (MIT) is inoculation of suspected sample **intracerebral** into young mice for virus amplification

- required samples: hippocampus, cerebral cortex, cerebellum and pons-medulla
- Death within 28 hrs will be considered as positive.

Rabies cont'd

B/ Rapid tissue culture infection test (RTCT)- Inoculation of sample onto cell cultures (e.g., Mouse neuroblastoma cells (MNA) and baby hamster kidney (BHK) cells)- Faster and cheaper than mouse inoculation test;

- Sensitivity-comparable to MIT; no mice sacrificed
- Requires training and manpower, as well as cell culture systems and fluorescence microscopy facilities; sensitive to toxic and bacterial contamination; amplification of live virus may require adequate biosafety (safety cabinets and BSL-3 laboratory)

Rabies cont'd

2. Detecting viral RNA

a. Reverse-transcriptase PCR (RT-PCR)

b. Real-time reverse-transcriptase PCR (RT-qPCR)

- international health organizations do not currently consider molecular assays to be reference techniques for the post-mortem diagnosis of RABV in humans and animals
- however these tests are used to diagnose rabies ante-mortem (before death) in humans.
  - Saliva samples or skin biopsies taken at the nape of the neck (being careful to include hair follicles)

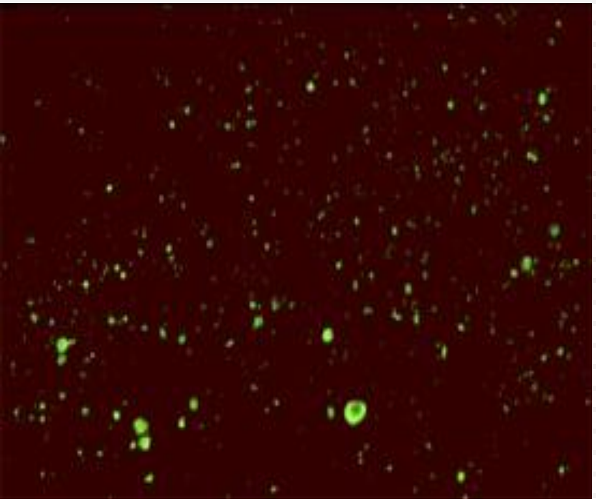
Rabies cont'd

3. Detecting viral antigens/proteins

A. Direct fluorescent antibody test (DFAT)

- Gold standard for fresh or fixed brain samples
- DFAT uses fluorescein isothiocyanate (FITC)-labeled anti-rabies antibody.
- Rabies-suspect brain tissue will be incubated with FITC and visualized using a fluorescence microscope.
  - Positive (bound) fluorescent-apple-green areas will be seen under the microscope.
  - Negative (Unbound) **dark areas** will seen.
- Results are available within 1–2 h and are expressed as positive or negative.

Rabies cont'd



Positive DFAT

Fluorescence microscope

Negative DFAT

Rabies cont'd

B. Rapid immunodiagnostic test (RIDT)

- Is **immunochromatographic** assay based on monoclonal antibodies to capture rabies antigens
- Highly sensitive and specific but usually less so than DFAT;
- Usable on **brain and saliva samples** from animals;
- Results obtained rapidly
- Need for further validation before either OIE or WHO can recommend its use

Rabies cont'd

Rapid immunodiagnostic test (RIDT)

Diseases caused by capripoxvirus

- Capripoxvirus (CaPVs) is one of the genera under the **poxviridae family** which is responsible for the most economically significant diseases of **domestic ruminants in developing countries.**
- Capripoxvirus (CaPVs) is double stranded DNA (ds DNA).
- It is comprised of Lumpy Skin Disease Virus (LSDV), Sheep Pox Virus (SPPV), and Goat Pox Virus (GTPV).

Diseases caused by capripoxvirus

- They are the only DNA virus which completes their replication cycle in the **cytoplasm of the host cells**

- There is **DNA cross-hybridization** between species of **CaPV** which account for **serologic cross-reaction and cross-protection.**

- Although they are considered host specific, natural and experimental **cross-infection** of CaPVs is possible in other hosts. But no natural infection of sheep and goats with LSDV has been described so far.

Diseases caused by capripoxvirus

LUMPY SKIN DISEASE (LSD)

- LSD is among the significant diseases of CaPVs and caused by LSDV for which **Neethling strain is the prototype**.

- It is featured by circumscribed, few to **multiple nodular lesions covering different parts on the skin** which sometimes involve underlying subcutis and musculature.

- LSD has been **limited to domestic cattle** although there were natural case records in **water buffalo** (*Bubalis bubalis*).

Diseases caused by capripoxvirus

- LSDV is remarkably stable for long periods in the environment, scabs, nodular lesions and air dried hides.

Diseases caused by capripoxvirus

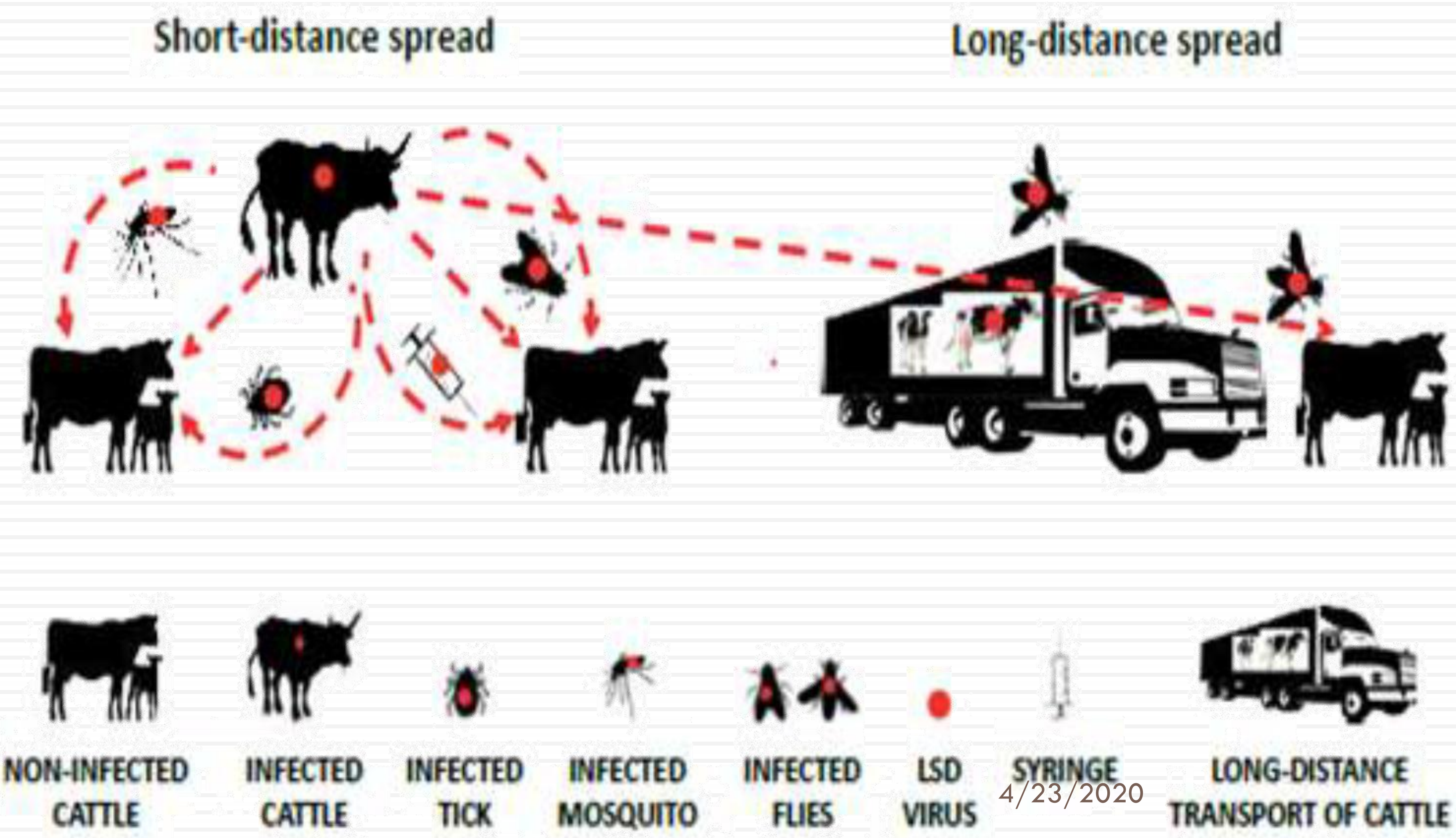
Sources of infection and transmission

- The **Principal sources** of infection to healthy animal is **the skin lesions**.
- The virus is also evacuated via blood, nasal and lachrymal secretions, saliva, semen, and milk of infected animals

Transmission;

- The significant means of LSDV transmission is **mechanical** by **hematophagus arthropod vectors such as mosquitoes, biting flies and ticks.**

Diseases caused by capripoxvirus



Diseases caused by capripoxvirus

Clinical manifestations

- The generalized clinical signs of LSD may explode from 7 to 14 DPI under experimental conditions whereas in natural cases it takes 2 to 5 weeks.

Diseases caused by capripoxvirus

- In severe cases that may persist for 7–12 days, there is;
  - continuous high pyrexia (40–41.5°C),
  - severe depression,
  - anorexia and
  - Typical circumscribed several (more than hundreds) nodular skin lesions covering the whole body.
  - Later the nodules may disappear or persist as hard lumps or become moist, necrotic, and slough or ulcerated.
  - Regional lymph nodes become enlarged (up to 10 times than their usual size), edematous and congested


A
B
C
D

Characteristic LSD lesion

Diseases caused by capripoxvirus

Differential diagnosis

- **Pseudo lumpy skin disease** which is caused by bovine herpes virus-2 (BHV) has related skin lesions with LSD and requires laboratory confirmation to distinguish.
- Other diseases related with skin lesion

Diseases caused by capripoxvirus

Laboratory diagnosis

- The diagnosis of LSD can be established based on the typical clinical signs combined with laboratory identification of the virus antigen or antibody.

Representative samples

- Skin biopsy (nodular lesions)
- Serum/blood samples

Diseases caused by capripoxvirus

Laboratory techniques

- Viral isolation
  - preferable secondary cell for culture- vero cell, bovine dermis cells or lamb testes cells
  - characteristic CPE and intracytoplasmic inclusion bodies detected
- serum or virus neutralization tests- **gold standard test**
- real-time PCR methods fast and sensitive method in confirming clinical cases by demonstrating ***viral DNA*** in **blood and skin samples.**
- Histopathology- intracytoplasmic inclusion bodies detected in the cells from skin nodules which is pathognomonic.

Diseases caused by capripoxvirus

Sheep and goat pox diseases

- Are overwhelming diseases of sheep and goats in developing countries due to high morbidity and mortality.
- They are caused by **SPPV (sheeppox virus) and GTPV (goatpox virus)** and cross infection is possible.
- characterized by fever, generalized skin nodules (pox lesions), lesions in the respiratory and gastrointestinal tracts and lymph node enlargement.

Diseases caused by capripoxvirus

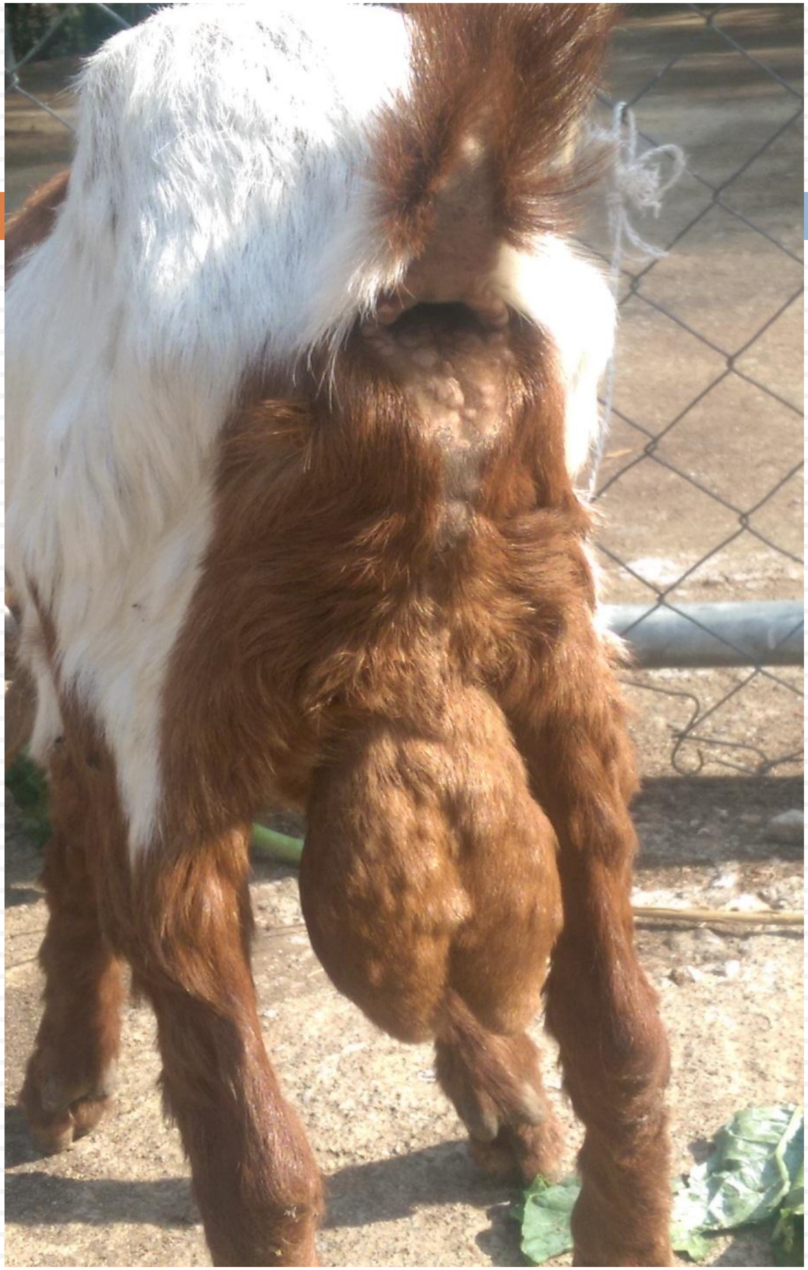
Transmission

- **by aerosol** following nasal secretion
- Direct and indirect contact with infected material
- mechanical transmission by insect vectors has also been **established experimentally.**

Diseases caused by capripoxvirus

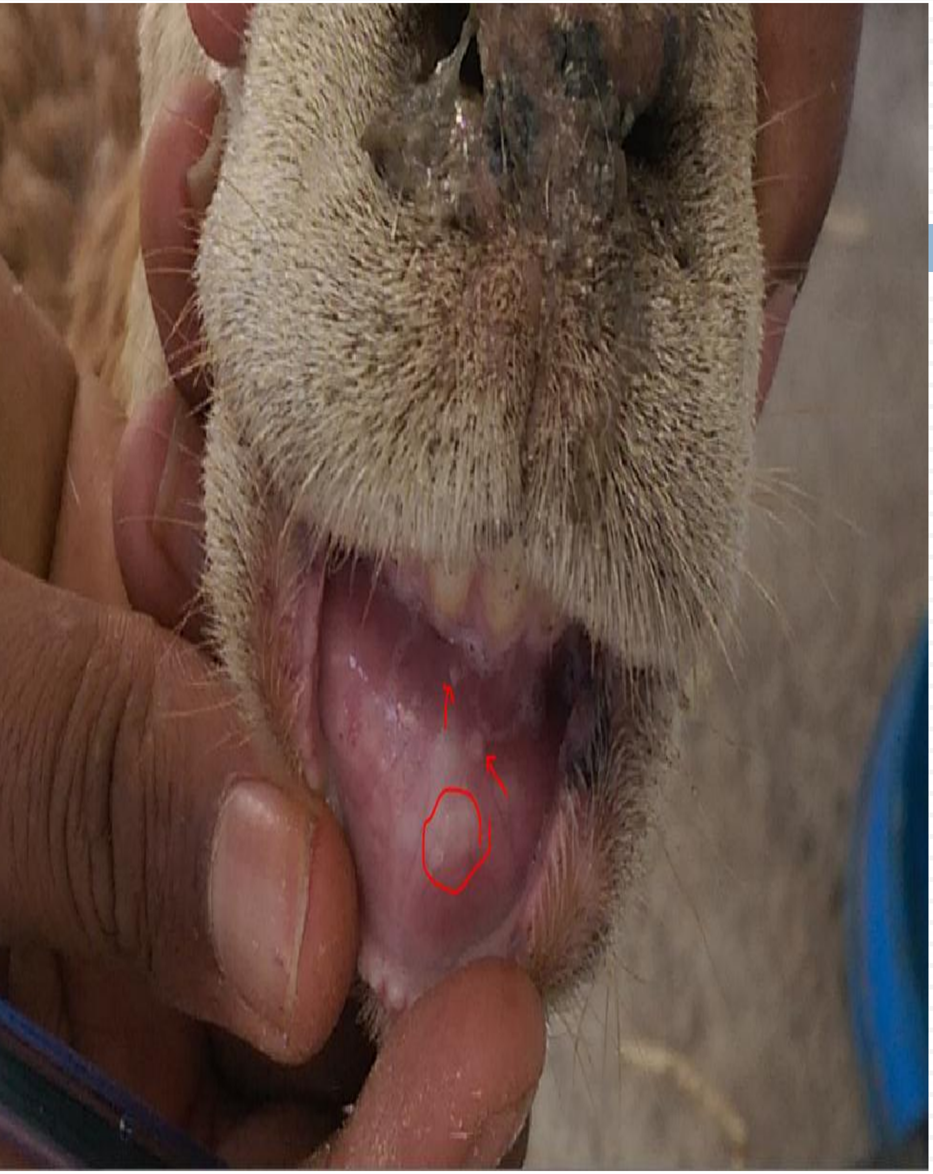
Clinical signs of SGP (Sheep and goat pox)

- SGP are **indistinguishable clinically**
- The incubation period of SGP 4-15 days in field condition
- Clinically they can be either malignant or benign.
- Malignant form - **common in lamb and kids**.
  - may die without observable pox lesion.
  - Fevers (40-42°C), dyspnea, ocular and nasal discharge and
  - pox lesion on unwooled skin (vulva, prenium, udder, nostril and mucous membranes of the mouth)



Diseases caused by capripoxvirus

- Benign form- common in adult
  - pox lesions occur particularly under the tail.
  - Lesions may be seen on the vulva, prenium, udder, nostril and mucous membranes of the mouth.
  - If lesion is present in the lung acute respiratory distress occurs



4/23/2020

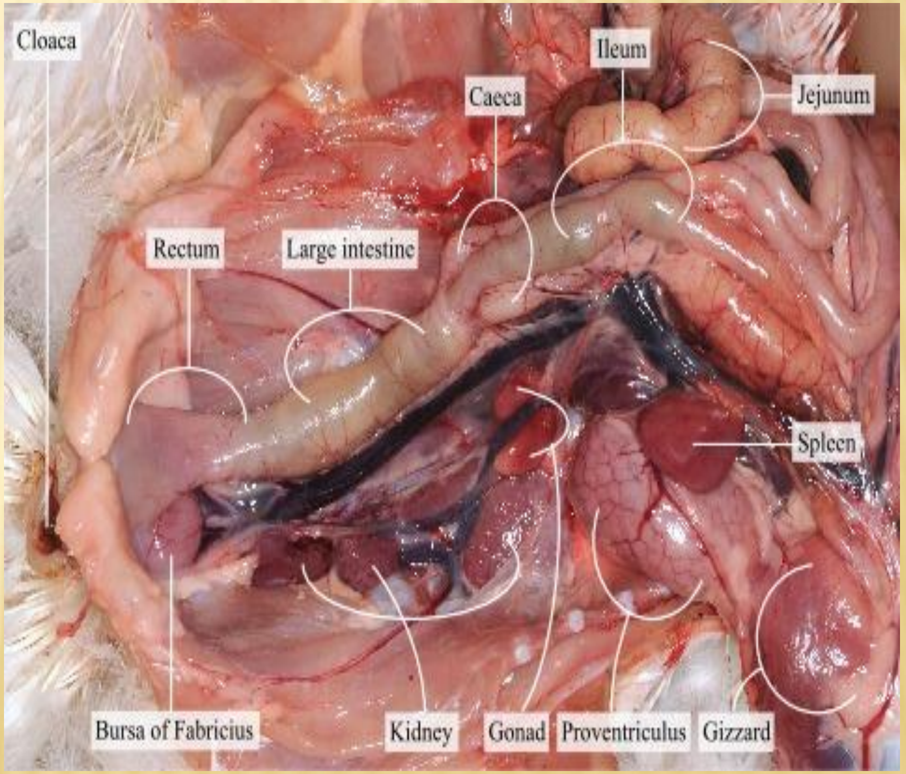
Diseases caused by capripoxvirus

Diagnosis

Required samples

- Skin biopsies
- vesicular fluid if available
- scabs
- lymph node aspirates
- whole blood

IMPORTANT VIRAL DISEASES OF POULTRY



Prepared by Abdi F.(DVM, MVSc, Asst. prof)

FOR-VLT YEAR III

What viral disease/s of poultry do you know


fieldcasestudy.com

1. Infectious bursal disease (IBD, Gumboro)

- It is an acute, highly contagious viral disease of **young chickens (18-40 days of age).**

- Manifested by inflammation and subsequent **atrophy of the bursa of Fabricius**, various degrees of nephroso-nephritis and immunosuppression.

- Has great economic significance in poultry producing countries world wide because of **heavy mortality and immunosuppression i.e. most deaths are associated with secondary infection.**

IBD cont'd

Cause and transmission:

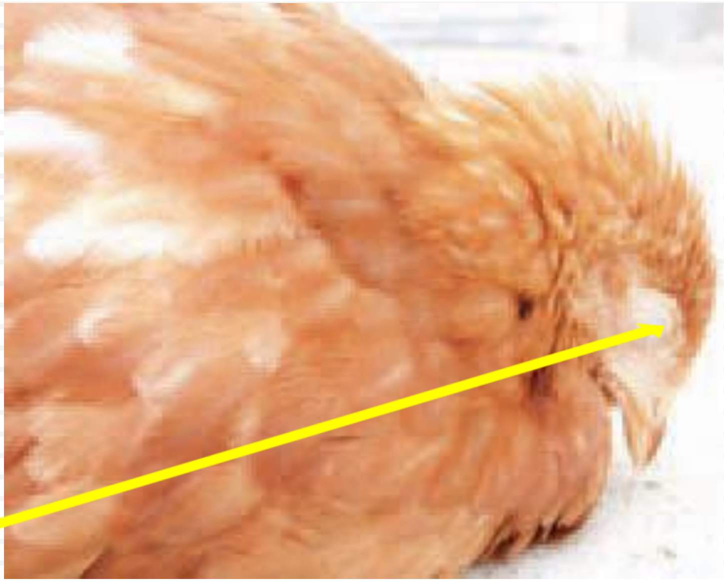
- Caused by **IBD virus** belongs to the **Birnaviridae family of RNA** viruses.
- Two serotypes (serotype1&2) are known to exist, but only **serotype 1 is pathogenic**.
- The virus have **an attraction to cells of bursa** and cause depletion of this organ.
- The virus **excreted via feces** for 10-14 days i.e. **most common route of infection is via oral** **but can also be spread via direct contact and inhalation**
- The virus is **stable in environment and remain infectious for months.**
- The incubation period is short and the first symptoms appear 2-3 days after infection

IBD cont'd

Clinical signs

Severe forms

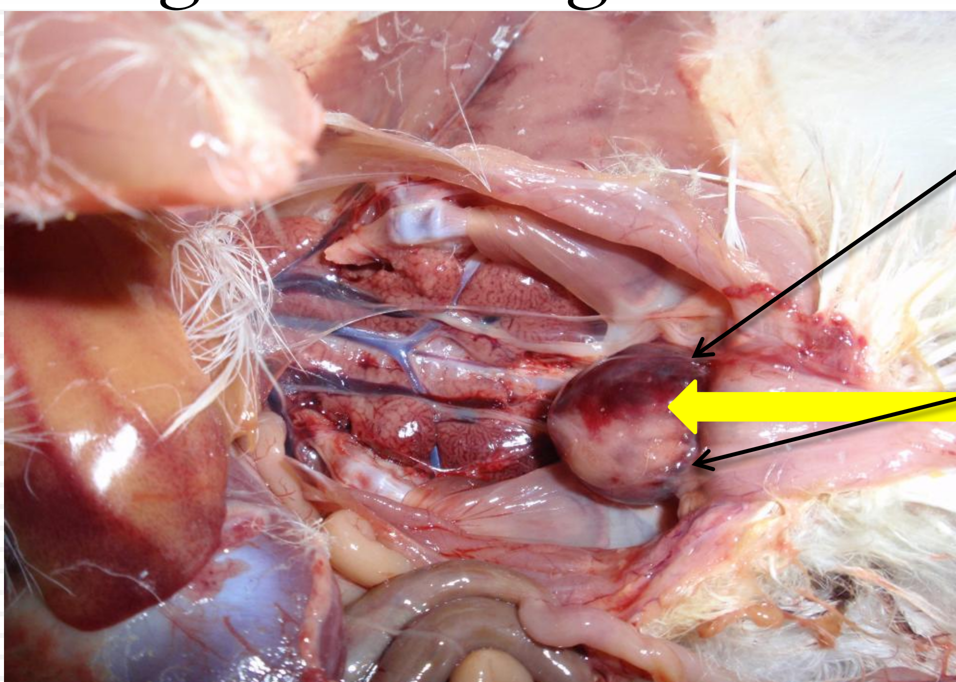
- Depression, **white watery diarrhea**
- The feathers around the vent are usually stained with feces containing plenty of **urates**
- **Vent pecking**
- Loss of appetite, ruffled feathers
- Unwillingness to move
- Trembling
- Closed eyes, lying down and death



IBD cont'd

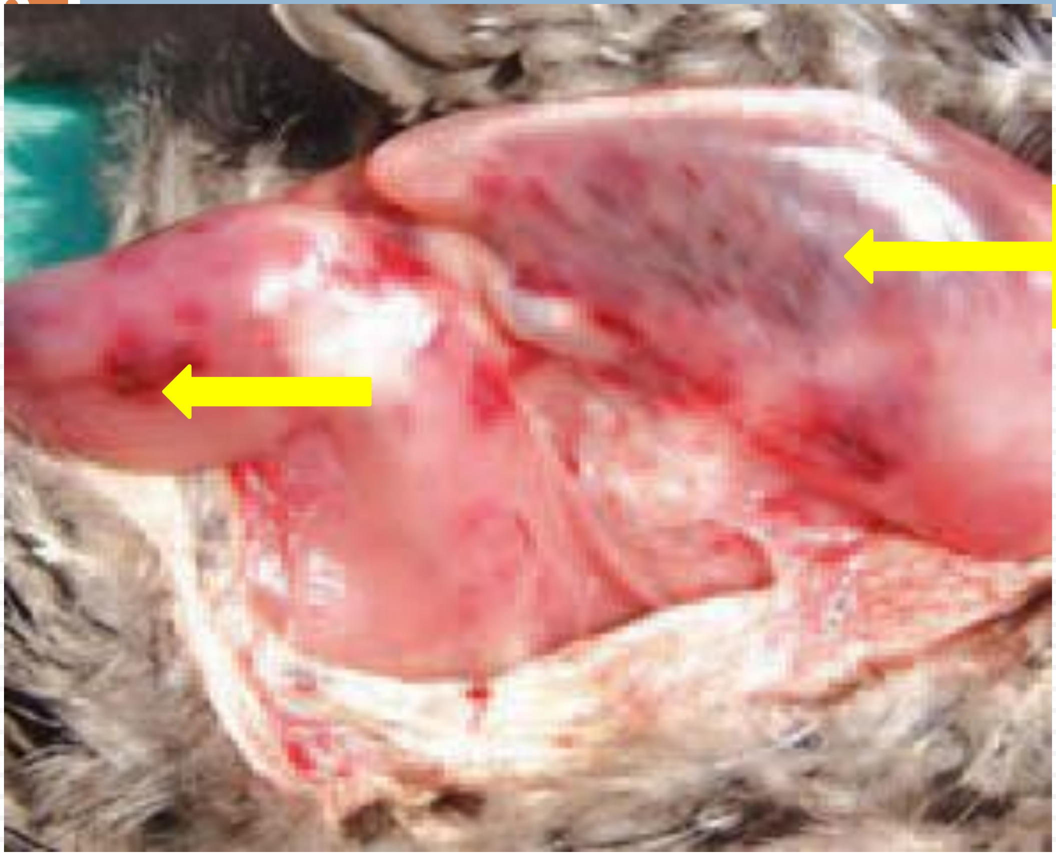
Postmortem findings

- Enlarged, hemorrhagic or edematous bursa of fabricius
- Kidneys are swollen and pale in appearance
- Hemorrhages in the thigh and breast muscle



Enlarged and edematous bursa

IBD cont'd



Hemorrhage in breast and thigh muscle

IBD cont'd

Diagnosis

- IBD can be diagnosed by a combination of characteristic signs and post-mortem lesions.

Laboratory confirmation

Virus isolation------------- READING ASSIGNMENT

- Detecting the presence of **viral antigen**
- Sample required----**bursa of Fabricius and spleen**
- Specific Pathogen Free (SPF) embryonated eggs or unvaccinated chickens

IBD cont'd

- Embryo mortality 2-4 days after the inoculation indicates the **sample is positive for IBD** and confirmed by the **virus neutralization (VN) test**.

AGID test (agar gel immunodiffusion) **READING ASSIGNMENT**

- Used to **detect viral antigen** in the **bursa of Fabricius**.
- A portion of the bursa is removed, homogenized, and used as antigen in a test against known **positive antiserum**.
- Useful in the early stages of the infection (before the development of an antibody response)

IBD cont'd

Ag-ELISA

- based on plates coated with IBDV-specific antibodies have also been described for the demonstration of IBDV antigens in **bursal homogenates.**

RT-PCR (reverse transcription polymerase chain reaction)

- used to detect viral genomic RNA in the **bursa of Fabricius**.

Histology of bursa tissue

2. Marek's disease

- Marek's disease is a **tumor-causing viral diseases** of chicken's characterized by marked growth of nerves, enlargement of liver, spleen and kidney's due to diffuse growth of certain cells.
- Mostly affect female birds
- Mostly begins in growers when they approach sexual maturity (b/n 17-20 weeks of age)

MD cont'd

Cause

- Caused by virus belonging to **herpes virus** group, intra nuclear (cell associated), **enveloped ds-DNA virus**
- Three types- **serotype 1**,2 and 3 0r (very harmful, harmful and mild)
- Once affected, chickens **remain infected until death** i.e. they continuously shed the agent and act as source of infection.
- The virus **concentrated in the feather follicles** and shed in the **dander** (scales from feather or dandruff) thus **inhalation** is the most important route of infection.
- The transmission is horizontal

MD cont'd

Clinical signs

- classical forms
  - Commonly affect nerves(brachial and ischiatic nerve) and characterized by **paralysis of wing and legs**
  - Affected chicken lies on their side with one leg stretched forward and the other back ward
  - Mortality is 10-15%

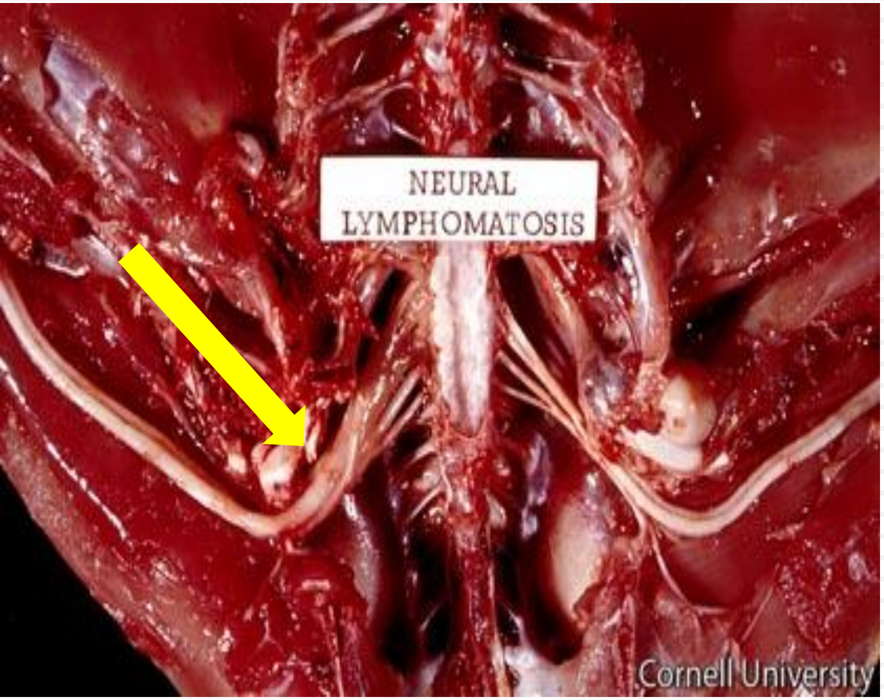
MD cont'd

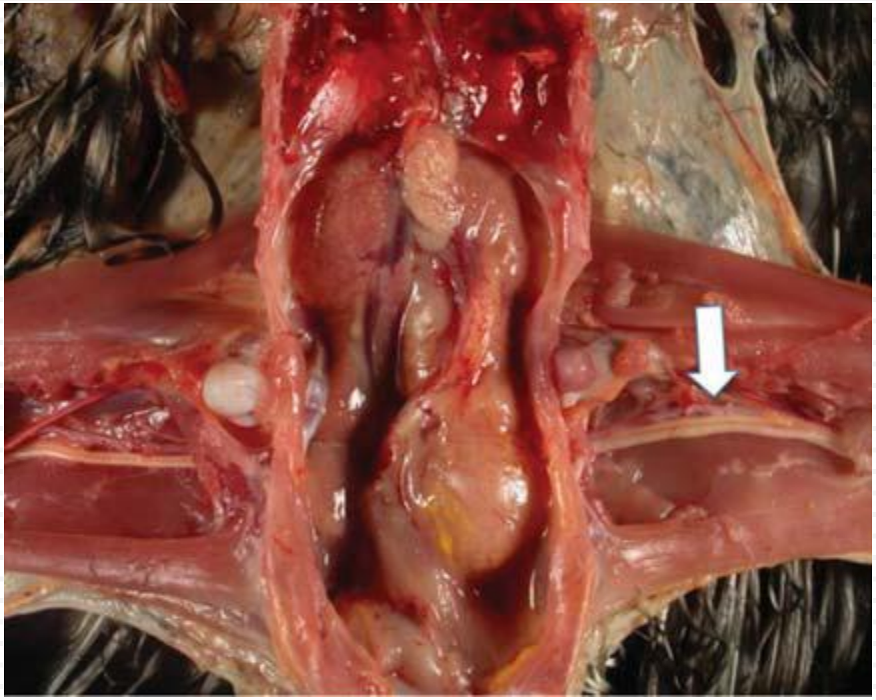
- Acute form
  - Mortality is high in this form (15-30%) and may reach 80% during an outbreak
  - Most chicken die without showing any symptom and some may depressed before death
  - Paralysis similar to Classical form may be observed in some chickens

MD cont'd

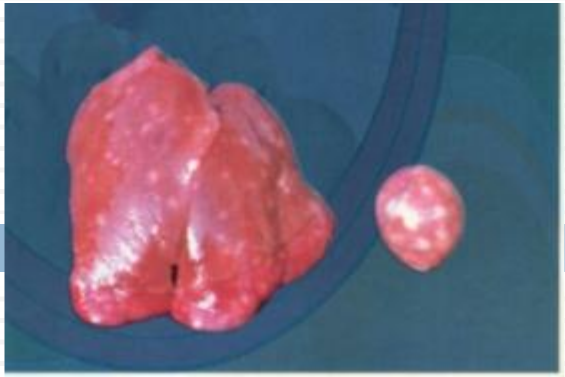
Post mortem findings

- Classical form
  - Marked enlargement of one or more nerves (ischiatic and brachial nerves commonly)

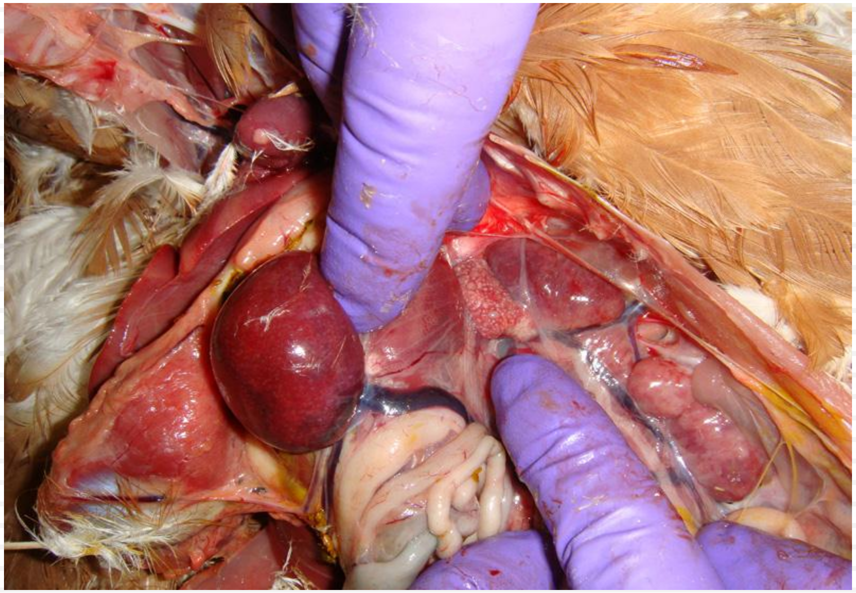




MD cont'd



- Acute form
  - Marked enlargement of internal organs such as lung, kidney, spleen, liver, gonads(tests and ovary), proventriculus and heart
  - Characteristically liver and spleen enlargement with white spot on their surface.



MD cont'd

Diagnosis

- Diagnosis is made based on clinical signs and gross or microscopic lesions
- MDV is detected by virus isolation and the demonstration of viral nucleic acid, antigen or antibodies.
- MDV **genomic DNA and viral antigens** can be detected in the **feather tips of infected birds** using **PCR** and the **radial immunoprecipitation test**, respectively.
  - These molecular diagnostic tests can be used for differentiating pathogenic and vaccine strains.
- **Serological tests:** Antibodies to MDV develop within 1–2 weeks of infection and are commonly recognized by the **AGID test, or the indirect immunofluorescent antibody test**.

3. Newcastle disease (NCD)

- Newcastle disease is an acute, rapid-spreading, contagious disease of **birds of all ages** characterized by lesions in the **respiratory tract, visceral organs, and nervous tissue**.

- It causes minor to severe mortality in susceptible flocks, depending on the pathogenicity of the virus.

NCD Cont'd

Cause

- Caused by New castle Disease virus, synonymous with **avian paramyxovirus serotype 1 (PMV-1),**
  - It is an enveloped RNA virus and the most important of the 11 known PMV serotypes as a pathogen for poultry.
- The original classification of NDV isolates into one of three virulence groups by chicken embryo and chicken inoculation as;
  - virulent (velogenic),
  - moderately virulent (mesogenic)

  or virulent NDV (vNDV),
  - low virulence (lentogenic) (loNDV) ---widely used as live vaccines (LaSota).

NCD Cont'd

Transmission

- Infected birds shed virus in **exhaled air, respiratory discharges, and feces.**
- Virus is shed during incubation, during the clinical stage, and convalescence period (limited).
- Virus may also be present in **eggs laid during clinical disease**
- Chickens are readily infected by **aerosols and by ingesting contaminated water or food.**

NCD Cont'd

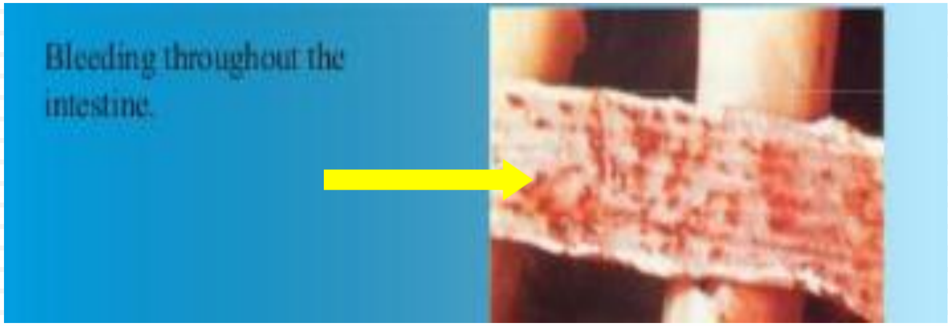
Clinical signs

- **respiratory signs** – gasping, coughing, sneezing and rales,
- **nervous signs** – tremors, paralyzed wings and legs, twisted necks(torticolis, opisthotonus), circling, spasms, and paralysis,
- **digestive signs** – watery greenish diarrhea,
- Comb and wattles may turn dark and bluish, and birds may develop swollen head(eyelid with abnormal accumulation of fluid) and neck,
- a partial or complete drop in egg production may occur. Eggs may be abnormal in color, shape, or surface, and have watery albumen,
- mortality is variable but can be as high as 100%.

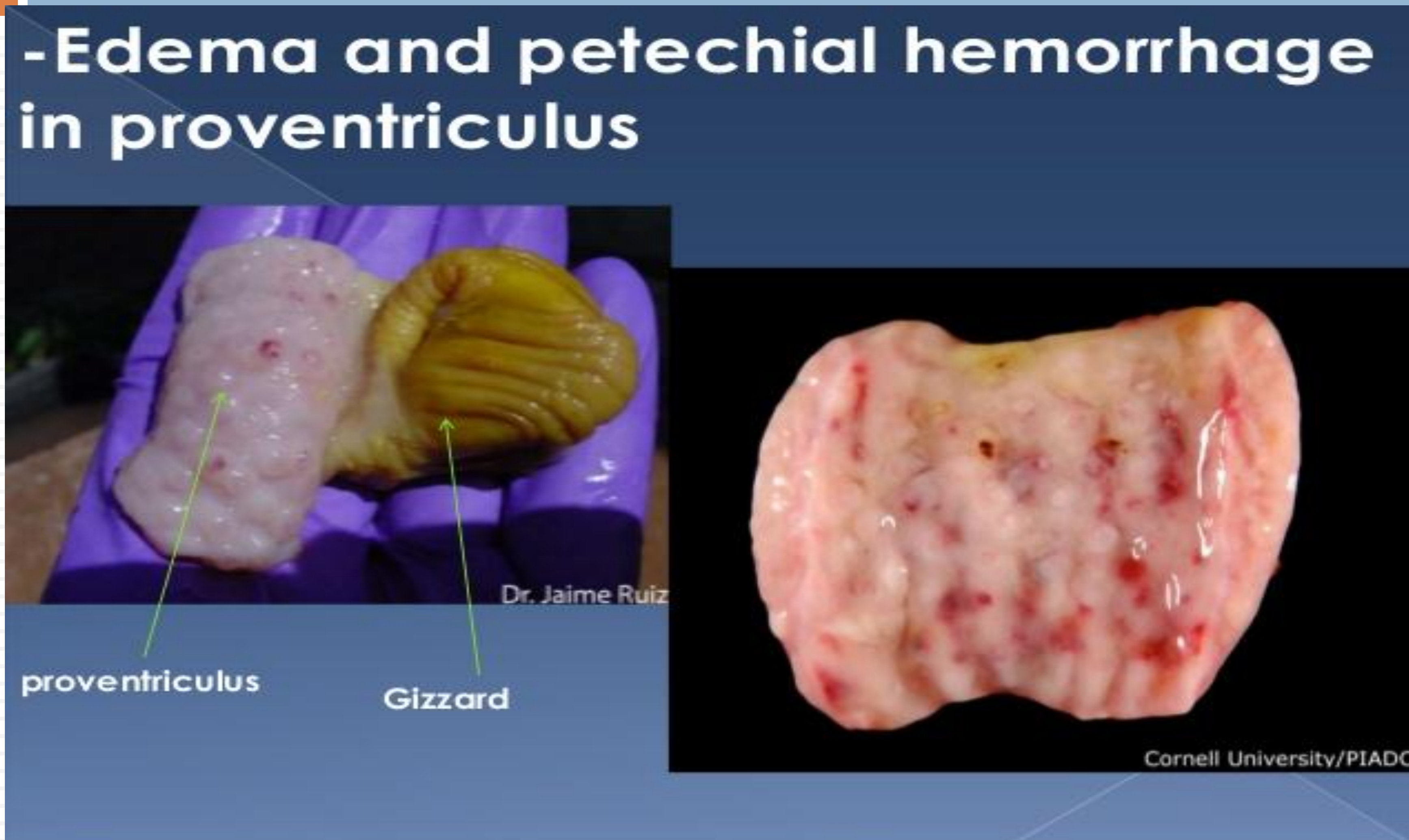
NCD Cont'd

Postmortem findings

- With the velogenic strain, there are varying degrees of **congestion and hemorrhages in visceral organs, including the proventriculus, ceca, and small intestines,**
- With the mesogenic form, hemorrhages may occur in the proventriculus and less commonly in the small intestines,
- There is **clear fluid present in the nasal passages, larynx, and trachea**



NCD Cont'd


-Edema and petechial hemorrhage in proventriculus
Dr. Jaime Ruiz
proventriculus
Gizzard
Cornell University/PIADC

NCD Cont'd

NCD Cont'd

Diagnosis

- Can be made based on clinical sign, necropsy and confirmation in laboratory.

Laboratory confirmation

- **Viral isolation**
  - inoculation of suspected sample into embryonated eggs
  - Specimens for attempting isolation of the virus should be selected from birds that **show early clinical signs** of the disease.
  - Samples should be taken from the **oro-nasal swabs**, **trachea, cloaca, brain, lung, kidneys, intestine (including contents), spleen, liver and heart tissues** from live or moribund

NCD Cont'd

- Following inoculation via the allantoic sac with Newcastle disease virus have different **mean death times** depending the pathogenicity of the virus;
  - velogenic (death in less than60 hours),
  - mesogenic (60–90 hours), or
  - Lentogenic (greater than 90 hours to kill embryo)

NCD Cont'd

- **Serological tests**
  - Clotted blood samples or serum
  - Enzyme-linked immunosorbent assay **(ELISA):** as whole virus is used as antigen, detects antibody to all of the virus proteins
  - commercial ELISA kits available to assess post-vaccination antibody levels


I am Ameraucana

Vector borne rickettsial and protozoal diseases of farm animals

Glossina morsitans
Tsetse fly
(WHO/TDR/Petana)

The cattle tick (*Boophilus*) carries a variety of diseases harmful to animals and humans.

Prepared by Abdi F.(DVM, MVSc, Asst. Prof.)

FOR-VLTS—SUMMER-YEAR-VI

- What do you know about vectors?
- Mention diseases caused by vectors
- How can we diagnose these diseases in suspected animals?
- Why does they are important?

Ehrlichiosis/Heartwater or cowdriosis

Ehrlichiosis/Heartwater or cowdriosis

- Heartwater is a non-contagious tick-borne disease of domestic and wild ruminants caused by an intracellular rickettsial pathogen previously known as *Cowdria ruminantium* but reclassified as ***Ehrlichia ruminantium*** .

- *Associated with **Amblyomma spp** of ticks*

- Heartwater is usually an acute disease and may be fatal within days of the onset of clinical signs due to hydropericardium and hydrothorax

Ehrlichiosis

Causative agent

- Caused by ***Ehrlichia ruminantium*** an obligate intracellular rickettsial agent belonging to the family *Anaplasmataceae* .

- The organism grows in **membrane-bound vacuoles** within the **cytoplasm of the host cell mainly endothelial cells** and to a lesser extent **neutrophils.**

Ehrlichiosis

Transmission and sources of infection

- *Amblyomma spp*. are the known vectors of *E. ruminantium* and are three-host ticks and transmit the agent while feeding on susceptible host;
  - *A. variegatum, A. hebraeum and A.cohaerens*
- The larvae and nymphs acquire infection by feeding on E. ruminantium-infected domestic or wild ruminants (source of infection for ticks).
- The ruminant host, once infected, may remain a carrier for up to 3.5 years and potentially for the rest of its life, thus serving as a reservoir of infection for ticks.

Ehrlichiosis

Clinical signs

- It is clinically characterised by **sudden onset of high fever**, which may be accompanied by **nervous signs** and may be followed more or less rapidly by **death**.
- disease usually develops within 10 to 30 days following infected tick bite.

Ehrlichiosis

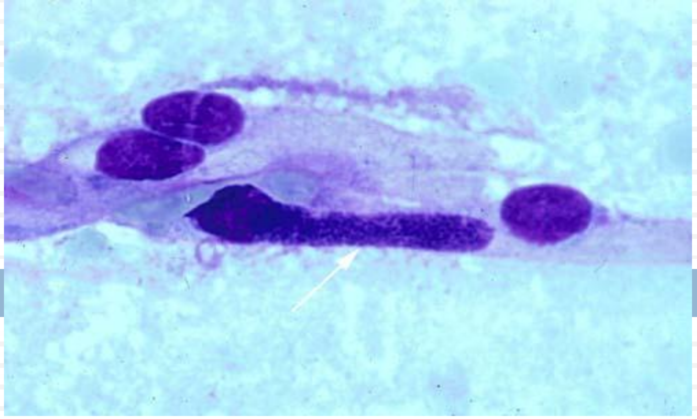
Forms of HW

- **Peracute form**---death with terminal convulsions occurs suddenly with little or no prior indication of clinical disease.
- **Acute form**--- are more common, high fever , anorexia, dyspnea, nervous signs and death within 2-6 days.
  - It comprises ataxia, chewing movements, twitching of the eyelids, circling, aggression, apparent blindness, recumbency, convulsions, and death

Ehrlichiosis

- **Sub acute form**--- the clinical signs are similar to those in the acute form, but less pronounced, and may be followed by death or recovery.
- **Mild or inapparent form**---the only clinical sign is transitory fever, which may not be noticed in the field, followed by recovery and development of immunity.
  - This form is common in neonatal animals, which possess an innate inverse age-related resistance to heartwater disease

Ehrlichiosis



Diagnosis

- Is the most **difficult part to confirm in live animals**
- Confirmatory diagnosis is based on identifying the rickettsia in **capillary endothelial cells** using a **Giemsa-stained squash** preparation of **brain or highly vascularized tissue at postmortem**.
  - The rickettsia occur as **blue to reddish** purple colonies or morulae of five to several hundred coccoid organisms (0.2 to 0.5 microns in diameter) in the cytoplasm of the cells close to the nucleus.

- Serological assays such as **IFAT, Immunoblotting and ELISA**, only provide information about previous exposure of an animal to infection and do not differentiate between strains of *E.ruminantium* **because of cross reaction unlike PCR-based techniques.**

Anaplasmosis

- Anaplasmosis is a disease of domestic and wild animals caused by the genus *Anaplasma* (Rickettsiales: Anaplasmataceae) which is **obligate intracellular gram-negative bacteria** found **exclusively within membrane-bound inclusions or vacuoles in the cytoplasm** of both vertebrate and invertebrate (tick) **host cells.**

- The genus includes *A. marginale, A. centrale, A. bovis, and A. ovis,* which are pathogens of ruminants; *A. phagocytophilum,* which affects a wide range of hosts, including humans, wildlife, and domesticated animals; and *A. platys,* which infects dogs.

Anaplasmosis

- *A. marginale* is the causative agent of anaplasmosis in cattle, buffalo, and wild ruminants, it is transmitted by ticks and other vectors,

  - It is an acute, fever producing disease in cattle and multiples by binary fusion in the red blood cells of hosts and cause severe anemia.

  - occurs at the edge of the red cells.

  - The incubation period of the disease is about 2 – 12 weeks.

Anaplasmosis

Sources and methods of transmission

- The source of infection is always the **blood of an infected animal.**
- **Persistent carriers** are the reservoir for herd infection.
- Transmission is **biologically by ticks** but can also occur transplacentally.
- Mechanical transmission is by **biting flies or blood contaminated fomites**.

Anaplasmosis

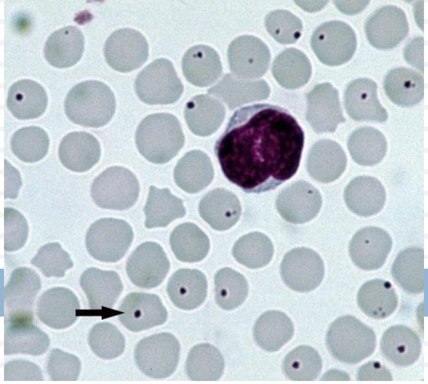
- Of the ticks, the one-host *Rhipicephalus* (*Boophilus*) spp. are of major importance in tropical and subtropical regions, and the three-host *Dermacentor* spp. are of major importance in the western United States.
- Tabanids are efficient mechanical vectors and can transmit infection for 2 hours after feeding.
- Sucking lice (Haematopinus spp. and Linognathus spp.) have been identified as potential vectors of anaplasmosis in cattle, goats, and buffalo

Anaplasmosis

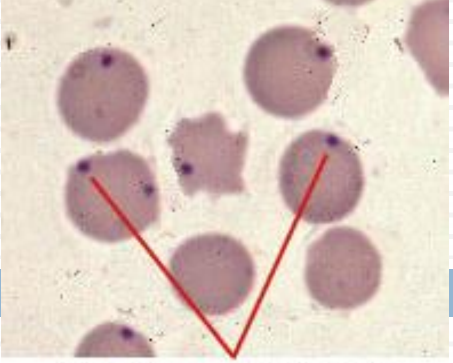
Clinical signs

- The number of infected erythrocytes increases logarithmically
- Clinical signs appear consistently when **40 to 50 % of RBCs** have been removed
- Progressive hemolytic anemia
- Icterus
- No hemoglobinuria (because extravascular hemolysis, in the reticulo endothelial cells; liver and spleen).
- Fever
- Weight loss
- Abortion
- Decreased milk production
- Death

Anaplasmosis



Anaplasma centrale



Anaplasma marginale

Diagnosis

- Is based on clinical signs and blood smear (thin-thick-giemsa stain) for **clinical cases**
- Serological:
  - Complement fixation
  - IFAT
  - ELISA
- PCR –best method for epidemiological investigation and can identify agents.

Babesiosis

- *Babesiosis is a* tickborne disease of animals caused by obligate, **intraerythrocytic** apicomplexan *Babesia* parasites.

- *B. bigemina* and *B bovis* are economically significant in cattle industries in tropical and subtropical areas.
- It also affects horses, sheep, goats, pigs, and dogs with varying degrees of importance throughout the world.
- Infection of a vertebrate host is initiated by inoculation of sporozoites into the **blood stream** while the tick takes a blood meal.
- Most babesial sporozoites directly invade circulating erythrocytes without a tissue stage of development.

Babesiosis

Table indicating Babesia species affecting livestock with their respective tick vector

	Babesia sp	Tick species	Country
Cattle	*Babesia bigemina*	*Boophilus annulatus, B. microplus, B. (annulatus) calcaratus, B. decoloratus; Rhipicephalus appendiculatus, R. bursa, R. evertsi; Ixodes ricinus;*	North America, Australia, South America, Africa
		Haemaphysalis punctata	Europe
	Babesia bovis	*Ixodes persulcatus,*	Former USSR
		I. ricinus;	Europe
		B. annulatus,	Iran
		B. microplus	Australia
	Babesia berbera	*B. annulatus (calcaratus); Rhipicephalus bursa*	Africa
Sheep and goats	*Babesia motasi*	*Dermacentor sylvarum; Rhipicephalus bursa; Haemaphysalis punctata; Ixodes rincus*	Europe
	Babesia ovis	*Rhipicephalus bursa;*	Former USSR
		Haemaphysalis bispinosa;	India
	Babesia ovata	*Haemaphysalis longicornis*	Japan
Horses	*Babesia caballi*	*Hyalomma dromedarii;*	Africa
		Dermacentor (reticulata) marginatus, D. pictus, D. sylvarum;	Former USSR and the Balkans, South America, Florida in the United States
		Hyalomma (excavatum) anatolicum, H. marginatum, H. volgense; Rhipicephalus bursa, R. sanguineus	Africa, the Balkans, South America, Australia
	Babesia equi	*Hyalomma dromedarii; Rhipicephalus evertsi, R. sanguineus; Dermacentor marginatus, D. pictus; Hyalomma anatolicum, H. marginatum, H. uralense; Rhipicephalus bursa, R. sanguineus*	

Babesiosis

Transmission

- The main vectors of *B bigemina* and *B bovis* are one-host ***Rhipicephalus (Boophilus)*** **spp** ticks, in which transmission occurs transovarially.
  - *Boophilus annulatus*
  - *B. microplus*
  - *B. decoloratus*
- mechanical transmission by insects or during surgical procedures has no practical significance.
- Transmission to the host occurs when **larvae** (in the case of *B bovis*) or **nymphs and adults** (in the case of *B bigemina*) feed on infected host.

Babesiosis

Clinical signs

- fever
- Anemia
- Jaundice
- Hemoglobinuria (**due to intravascular RBC’s destruction**)
- high case-fatality rate.
- CNS involvement due to adhesion of parasitized erythrocytes in brain capillaries can occur with **B bovis** infections.

Babesiosis

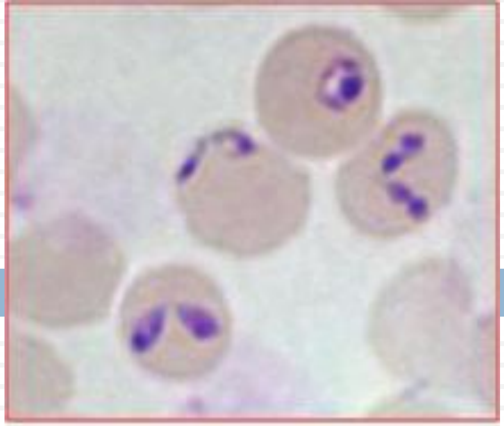
Postmortem lesions

- Particularly with *B bovis* enlarged and friable spleen, liver with an enlarged gallbladder containing thick granular bile; congested, dark-colored kidneys; and generalized anemia and jaundice.

- Most clinical cases of *B bigemina* have hemoglobinuria, but this is not invariably the case with *B bovis*. Other organs, including the brain and heart, may show congestion or petechiae.

Babesiosis

Diagnosis



Babesia bovis

Babesia bigemina

- Confirmatory diagnosis in live animal can be made by microscopic examination of Giemsa-stained thick and thin blood smears from **capillaries in the ear or tail tip**.
- Smears of heart muscle, kidney, liver, lung, brain, and from a blood vessel in an extremity (eg, lower leg) should be taken at necropsy.
  - Morphologically; *B bovis* is small, with the parasites in paired form at an obtuse angle to each other;
  - *B bigemina* is larger with paired parasites at an acute angle to each other.
  - Single forms of both parasites are also commonly seen.

Trypanosomsis

Trypanosomosis

- Trypanosomosis is a disease caused by flagellated protozoan parasites belonging to the genus *Trypanosoma,* family Trypanosomatidae.
- They **live in the blood and other body fluids** of vertebrate hosts, where some of them cause disease.
- With the help of the flagellum, trypanosomes swim within the vertebrate bloodstream and prosper despite being constantly attacked by the host immune system.
- The parasites can be transmitted mechanically, biologically and venereal (***the summary is indicated in the next Table***).

Trypanosomosis

Summary of the trypanosomoses of domestic animals and humans

Disease	Distribution	*Trypanosoma* spp.	Main vector
Animals			
Nagana or African trypanosomosis (most mammals)	Tropical Africa	*T. brucei brucei* *T. congolense* *T. vivax* *T. simiae*	*Glossina* spp. Other biting flies
Surra (horses, camels, buffaloes)	Africa, Asia, South and Central America	*T. evansi*	Biting flies
Dourine (horses and donkeys)	Africa, Asia, South and Central America	*T. equiperdum*	None (venereal transmission)
Nonpathogenic (cattle and sheep)	Worldwide	*T. theileri* *T. melophagium*	 Biting flies
Humans			
Rhodesian sleeping sickness	East, central, and southern Africa	*T. brucei rhodesiense*	*Glossina* spp.
Gambian sleeping sickness	Western and central Africa	*T. brucei gambiense*	*Glossina* spp.
Chagas' disease (also in dogs, cats, and pigs)	South and Central America, southern United States	*T. cruzi*	*Rhodnius* spp. *Triatoma* spp.

Trypanosomosis

African trypanosomosis/ Nagana

- Common in most mammals

Caused by

- T. Vivax
- T.Congolense
- T. brucei
- T. Simiae

Vectors – mainly Glossina sp/Tsetse flies

–other biting flies

Trypanosomosis

Pathogenesis:

- Infected tsetse inoculate metacyclic trypanosomes into the skin of animals,
- the trypanosomes reside for a few days and cause localized inflammation (chancres).
- They enter the lymph and lymph nodes, then the bloodstream, where they divide rapidly by binary fusion.
- In *T congolense* infection, the organisms attach to endothelial cells and localize in capillaries and small blood vessels.
- *T brucei* species and *T vivax* invade tissues and cause tissue damage in several organs.

Trypanosomosis

Clinical feature of nagana/tsetse caused trypanosomes

- The basic clinical syndrome appears after an incubation period of 8 to 20 days following the infective tsetse fly bite includes;
- Anemia,
- Fever, which is likely to be intermittent or cyclic for weeks,
- Affected animals are dull, anorexic, and listless,
- have a watery ocular discharge and lose condition,

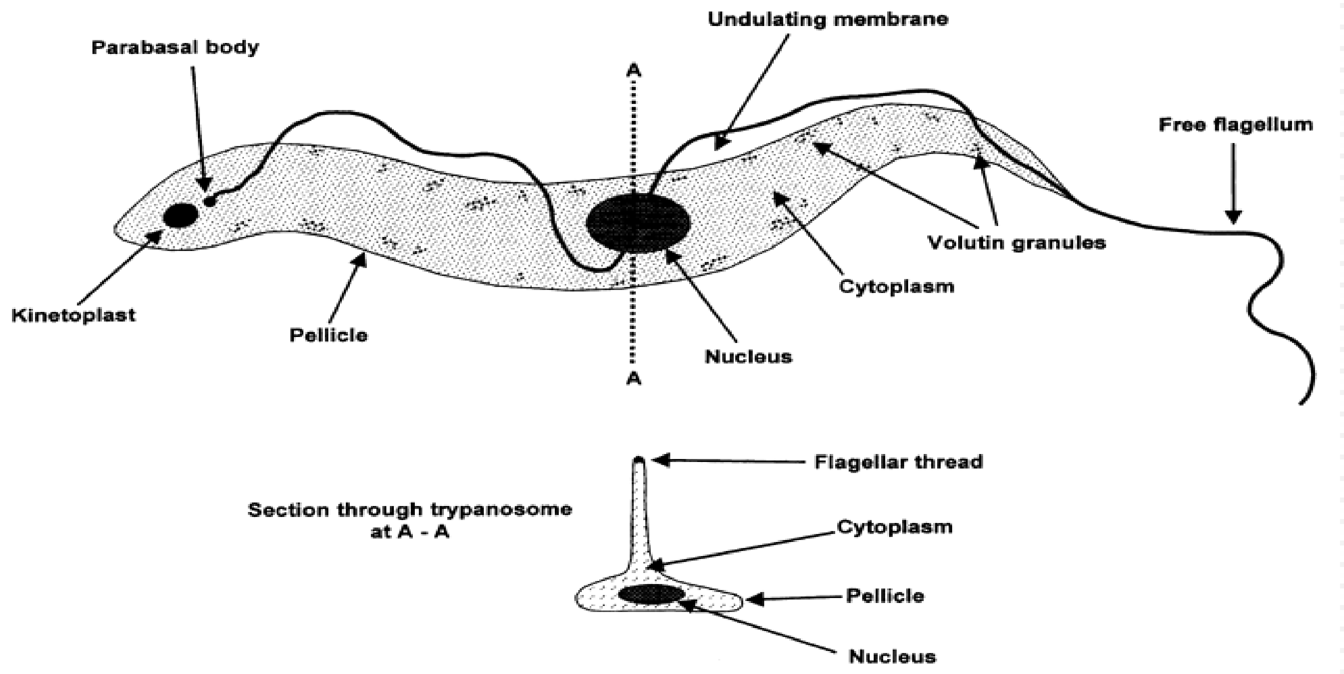
Trypanosomosis

- Superficial lymph nodes become visibly swollen,
- mucous membranes are pale, diarrhea occasionally occurs,
- Estrus cycles become irregular, pregnant animals may abort,
- The animal becomes very emaciated and cachectic and dies within 2 to 4 months or longer.

Trypanosomosis

Diagnosis

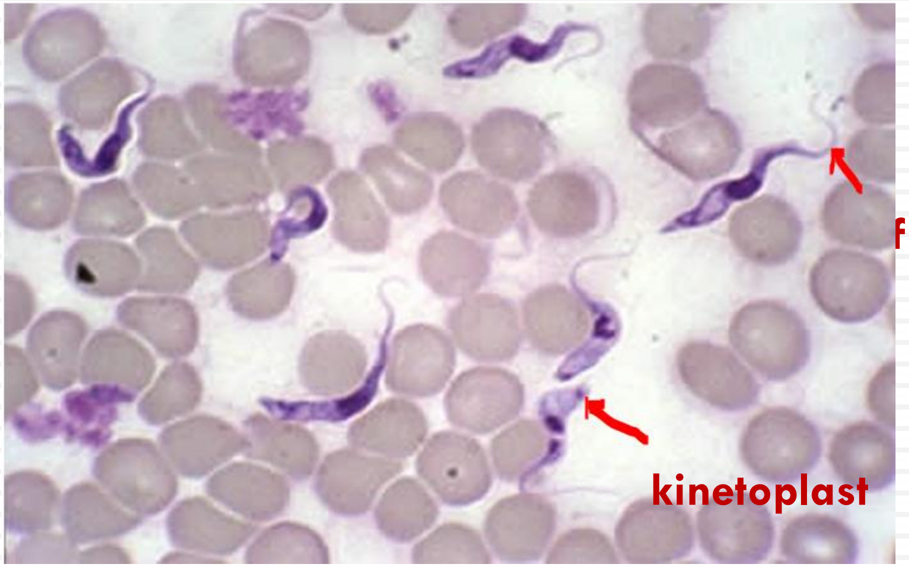
- They can be diagnosed in stained blood film
- The morphology and movement of the trypanosomes are characteristic for each species and are helpful in making a diagnosis.



Trypanosomosis

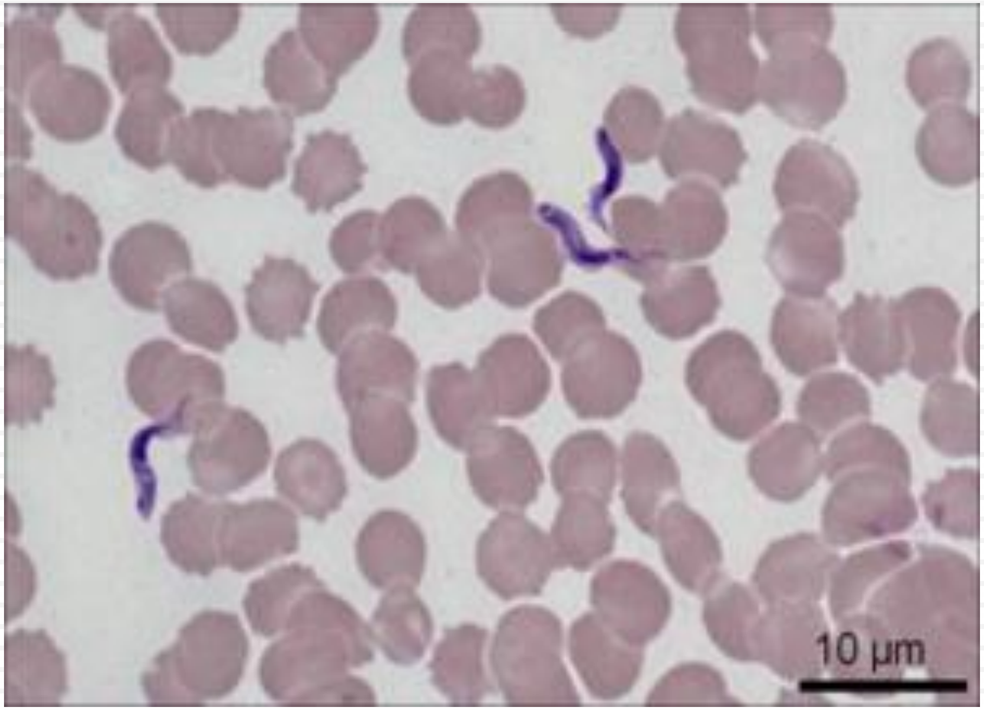
In acute infections

- *T. vivax* is usually numerous in blood samples and can be identified by its **very fast movement in wet films**. In stained smears, it is 20 to 26 μm long, slender, and monomorphic, with a rounded posterior end, **a terminal kinetoplast, and a long free flagellum**, but **no prominent undulating membrane**.



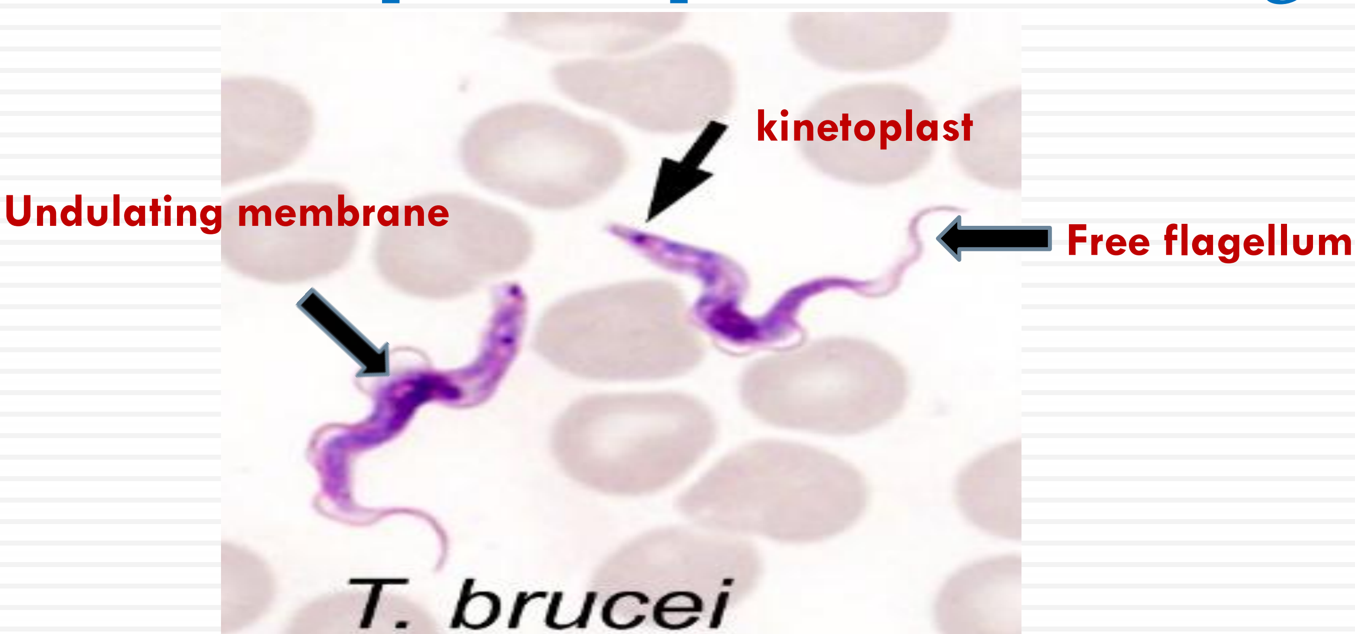
Trypanosomosis

- *T. congolense* is smaller, is sluggish in wet films, and often adheres to red blood cells by the anterior end. In stained smears, it is 9 to 18 μm long, with a marginal kinetoplast, no free flagellum, and no prominent undulating membrane.



Trypanosomosis

- *T. brucei* is large like *T. vivax*, but **its rapid movement is in confined areas of the wet film**. In stained smears, it is pleomorphic and may occur as long and slender forms up to 35 µm have **a long free flagellum**, pointed posterior end, **subterminal kinetoplast**, and **prominent undulating membrane**



Trypanosomosis

- ***Convectional parasite detection methods may not provide strong evidence in most indigenous areas and chronically infected animals but polymerase chain reaction (PCR) techniques can detect parasite nucleic acids and provide good information.***

    - For instance, a study conducted in Ethiopian involving 1524 animals, reported the overall prevalence of infection as 5.5% by conventional parasitological methods and 31.0% by PCR.

None infectious disease

- Ketosis
- Hypocalcaemia
- Acidosis
- Bloat

KETOSIS (Acetonaemia)

- Clinical KETOSIS is a metabolic disease of high-yielding milking cows associated with an inadequate supply of energy to sustain the high milk yields.
- majority of dairy cattle are in negative energy balance during early lactation,

AETIOLOGY

- Primary KETOSIS results during early lactation in high-yielding dairy cows when the cow cannot consume enough energy to supply her glucose requirements for lactogenesis
- Secondary KETOSIS is caused by diseases that depress food intake (e.g. LDA).
- They mobilize body reserves of fat and protein.
- The three principal ketone bodies produced are acetone, acetoacetate and β-hydroxybutyrate (BHB)

Risk factors include

- Inadequate energy content of the ration,
- Inadequate intake of the diet,
- Poor rumen function resulting from sudden changes in diet

CLINICAL SIGNS

- KETOSIS usually occurs within the first month after calving,
- Wasting form. This is the more common form with loss of appetite, refusal to eat concentrate feeds and a sudden drop in milk yield
- The faeces are often dark and firm, with a ‘waxy’ appearance
- The cow loses considerable body condition over 4–7 days.
- Temperature, pulse and respiratory rate are usually normal.


Treatment

- administering 400 ml of 40% glucose IV.
- Glucocorticoid therapy (e.g. dexamethasone)
- Multivitamin injections
- Predisposing causes should be corrected

Prevention/control measures

- Correct dry cow management should be implemented
- Any dietary changes should be made gradually.
- Cows should be grouped according to their nutritional requirements.

MILK FEVER

(HYPOCALCAEMIA, PARTURIENT PARESIS)

- A disease of cattle, sheep, and goats occurring around the time of parturition and caused by hypocalcemia
- characterized by weakness, recumbency, and ultimately shock and death.
- Plasma calcium concentration is normally maintained between **2.1 and 2.6 mmol/L or (8.5 -10.4 mg/dL)**
- Subclinical hypocalcemia **<1.8 mmol/L (7.5 mg/dL)**
- more severe hypocalcemia **<1.25 mmol/L (5 mg/dL)**

Etiology

- Hypocalcemia just before or after parturition
- The onset of lactation results in a sudden large demand on the calcium homeostasis.
- To maintain the normal concentration of calcium in the blood there must be increased absorption of calcium from the gut and/or mobilization from the skeleton.
- These processes take 2–3 days to become fully active and, if they fail, hypocalcaemia results.
- About 5-20% of adult cows are unable to maintain plasma calcium and consequently develop severe hypocalcemia

EPIDEMIOLOGY

- The disease occurs most commonly in high-producing adult lactating dairy cattle.
- It may occur at or before calving
- majority cases take place within 3 days subsequent to parturition.
- in exceptional situations (often very high-yielding cows during oestrus), several weeks to months after calving.
- most marked in cows at their 3rd to 7^{th} parturition
- the disease tends to recur at successive parturitions.

- Complete milking in the first 48 h after calving appears to be a precipitating factor
- there is a special susceptibility at estrus
- Episodes of subclinical hypocalcemia occur in up to 50% of adult cows during the first few weeks of lactation.
- Subclinical hypocalcemia is of major significance
- Starvation for 48 h also causes severe depression of serum calcium levels

Clinical signs

- usually occurs within 24 hours after parturition
- There is initial hyperaesthesia, with teeth grinding and coarse muscle tremors, stiffness of the limbs,
- Cows show ataxia and are reluctant to walk.
- The clinical signs progress to sternal recumbency

Typical case of hypocalcaemia which presented within 24 hours of calving. Note the head averted against the chest.

- Cows become comatose in lateral recumbency
- They are very weak and have an elevated heart rate (120 beats per minute).
- Eventually, ruminal tympany and/or paralysis of respiratory muscles causes death in untreated cattle.
- Potential complications of hypocalcaemia include uterine inertia (leading to dystocia and/or stillbirth),
- prolapse of the uterus and musculoskeletal damage.

DIFFERENTIAL DIAGNOSIS

- Acute toxic mastitis; physical injury/nerve paralysis;
- uterine rupture; haemorrhage caused by dystocia;
- acidosis/grain overload.

DIAGNOSIS

- Diagnosis is based on the cow’s history, clinical signs and response to intravenous calcium borogluconate solution

Treatment

- 400 ml of 40% calcium borogluconate solution (containing 12 g calcium),
  - warmed to body temperature,
  - should be administered by slow intravenous injection
  - (over 5–10 minutes) into the jugular vein
- Dairy cows should not be milked for 24 hours

Control

- Dietary management to reduce prepartum intake of calcium
- Calcium gel oral dosing before calving, at calving and 12 and 24 h after calving.

Acidosis

(CARBOHYDRATE OVERLOAD, RUMINAL LACTIC ACIDOSIS, GRAIN OVERLOAD)

Acidosis results from the sudden unaccustomed ingestion of large quantities of carbohydrate-rich feeds,

→ increased lactic acid production

→ a fall in rumen pH, which kills many cellulolytic bacteria and protozoa.

→ Acid-tolerant bacteria such as *Streptococcus bovis* survive, producing more lactic acid.

→ increase in rumen liquor osmolarity,

→ Lactate is absorbed into the circulation compromised rumen mucosa.

SIGNS

- Low rumen pH (below 5.0) reduces rumen motility, causing stasis and mild bloat.
- Colic signs
- They may fall and experience difficulty rising due to weakness
- Distended abdomen
- Tinkling sounds due to the sequestration of fluid and gas.
- There is profuse very fluid fetid diarrhoea after 12–24 hours
- Sometimes laminitis
- Death may follow within 24–48 hours despite treatment.

Treatment

- 72 g of sodium bicarbonate in 5 litres of saline
- Intravenous fluids that contain bicarbonate
- multivitamin preparations and antibiotic
- Antacid drenches including 500 g of magnesium hydroxide per 450 kg
- rumenotomy

Bloat

(RUMINAL TYMPANY)

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- Ruminal tympany is the accumulation of gas in the dorsal rumen following abnormal fermentation and indigestion.
- It may be of two types: free gas bloat or frothy bloat.

Free gas bloat

- The rumen becomes distended with gas, and pressure is exerted upon the diaphragm.
- Any condition causing oesophageal obstruction or interference with normal eructation can result in accumulation of free gas in the rumen.
- The cow's ability to belch may be affected by physical obstruction of the oesophagus; paralysis of the muscular wall of the rumen; and foaming of the rumen contents.

FROTHY BLOAT

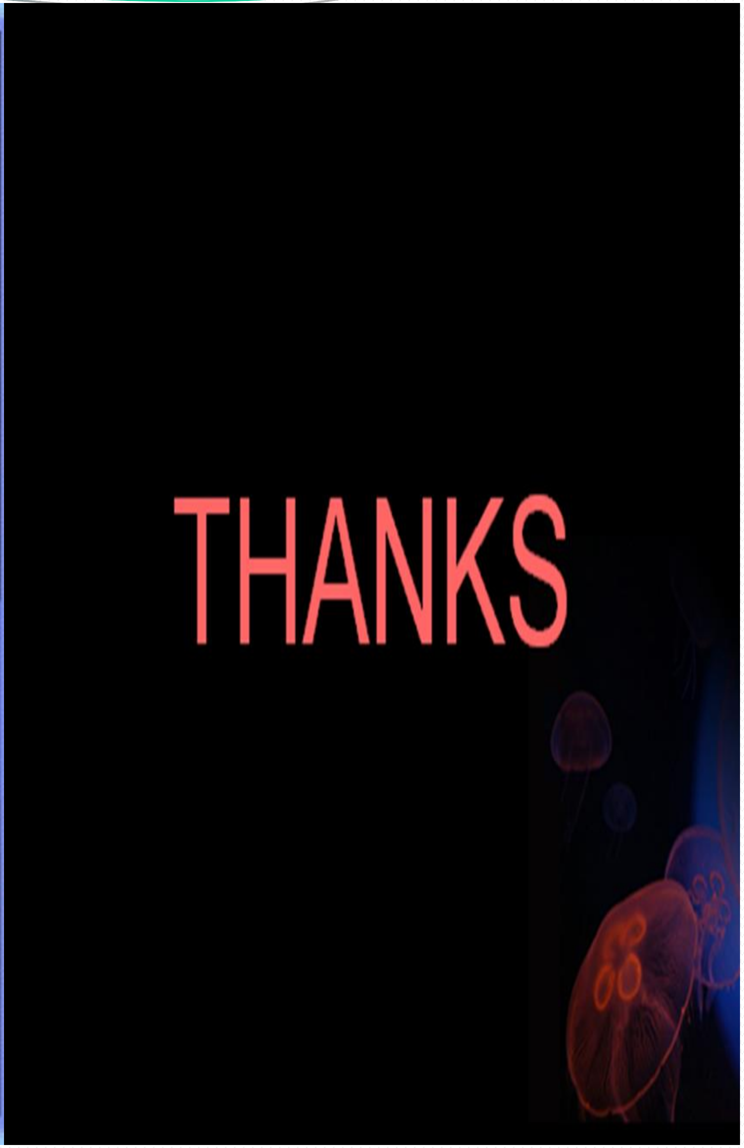
- Frothy bloat results from high protein levels in cattle
- grazing lush leguminous pasture
- feedlot cattle fed finely ground grain.
- Rumen fluid viscosity is raised, causing small bubbles to form and leading to a stable froth that cannot be eructated normally.

Signs

- Distended left flank
- Abdomen gradually becoming tense and drum-like.
- Restless
- Breathing is rapid.
- Distress, recumbency and, in extreme cases, death.

Treatment

- Affected cattle should be drenched/stomach-tubed with an anti-foaming agent such as vegetable oil or paraffin oil
- An emergency rumenotomy can be performed in extreme cases
- An orogastric tube is passed to relieve accumulated gas.
- trocar/canula


THANKS