# MARSABIT COUNTY VETERINARY HANDBOOK







### FOR PRIORITY LIVESTOCK DISEASES

Common Diagnosis, Treatment and Control Guidelines for Animal Health Service Providers

### Acknowledgements

The ASAL eXtension veterinary team led by Dr Pauline Gitonga would like to extend our sincere gratitude to the veterinary staff of Marsabit County Government. The county team played a crucial role in collecting and validating the data that contributed to the development of the veterinary handbook. We appreciate the support given by the Acting County Director of Veterinary Services Dr. Stephen G. Mutahi. Our sincere gratitude goes to the County Concern team composed of Dr. Tabitha Njeri Ndung'u, Peter Kanapal and Vincent Lekula for their technical support and their active participation in planning and supporting the handbook development process. We are indebted to the 311 livestock keepers who participated in the 41 specie specific Focus Group Discussions. Their patience and willingness to share their extensive ethno-veterinary knowledge was humbling and enlightening. We are thankful for the support provided by Dr. Wario, the Chief Officer Marsabit County, Hassan Olowprogramme director Concern worldwide, Dr. Halkano Arero Livelihoods coordinator, Jackson Mekenye- senior manager livelihoods and Hassan Abudulla Concern Marsabit Program manager who ensured successful completion of this exercise. Lastly, we recognise the generous support of the American people through the United States Agency for International Development (USAID) - Bureau for Humanitarian Assistance (BHA) that funded the I-CREATE project which supported the development of the veterinary handbook.



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### Disclaimer

THIS HANDBOOK IS INTENDED TO GUIDE ANIMAL HEALTH PRACTITIONERS WORKING IN MARSABIT COUNTY MANAGE ENDEMIC PRIORITY LIVESTOCK DISEASES. THE HANDBOOK SUMMARISES CONVENTIONAL VETERINARY KNOWLEDGE AND INTEGRATES LOCAL DISEASE NAMES AND ETHNOVETERINARY KNOWLEDGE & PRACTICE PRACTITIONERS NEED TO BE AWARE OF ALL THE LAWS AND REGULATIONS THAT APPLY TO THE PRACTICE OF VETERINARY MEDICINE IN KENYA AS THE HANDBOOK DOES NOT EXPLICITLY COVER VETERINARY JURISPRUDENCE. THIS HANDBOOK IS NOT INTENDED TO SUBSTITUTE FOR THE MANDATED PHYSICAL EXAMINATION OF ANIMALS BEFORE PRESCRIBING MEDICATION. IN ADDITION, THE DRUG PACKAGE INSERT OF INSTRUCTIONS FROM MANUFACTURERS SHOULD BE READ AND UNDERSTOOD BEFORE ANY DRUG IS ADMINISTERED OR PRESCRIBED. THE PRACTITIONERS SHOULD ALWAYS ENSURE THAT LIVESTOCK KEEPERS **UNDERSTAND THE LENGTH OF THE WITHDRAWAL PERIOD** FOR MILK, MEAT AND EGGS. THE HANDBOOK CITES VARIOUS VETERINARY MEDICINAL PRODUCTS. THEIR MENTION SHOULD NOT BE TAKEN AS AN ENDORSEMENT OF THE PRODUCT.

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### Preface

The Marsabit County Veterinary Handbook provides a simple and concise description of the priority infectious diseases and conditions known to occur in camels, cattle, goats, sheep, donkeys and chickens. The compilation of this Handbook builds on information sourced from existing veterinary medicine textbooks and peer-reviewed articles. The ethnoveterinary information was gathered from 29 Marsabit county veterinary staff and 311 livestock keeper's. The Handbook has incorporated the ethnoveterinary livestock diseases knowledge of the main livestock keeping communities of Marsabit county that is; Borana, Gabra, Samburu, Rendile, Turkana and Daasanach. The incorporation of ethnoveterinary knowledge is based on the understanding that working in pastoralists communities necessitates that animal health workers not only acquire updated technical livestock disease knowledge but should also possess an understanding of the social, economic and cultural context in which they operate.

The Handbook is written for users with formal training in animal health and production. The handbook is divided into nine chapters as detailed below:

Chapter one begins with a glossary of terminologies and gives a brief description of microbes that cause infectious diseases. Chapter two highlights guidelines on how to conduct general and physical clinical examination.

Chapter three to nine details specie specific priority diseases information that includes aetiology, epidemiology, clinical presentation, post-mortem lesions, diagnosis, treatment and control guidelines. The chapters also include local dialect names of the diseases and associated clinical signs as well as ethnoveterinary treatment and control practices.

Annex section- The handbook has 5 Annex sections: Annex 1 details the Marsabit county vaccination schedule for identified priority livestock diseases; Annex 2 summarises information of four priority Zoonotic Diseases in Kenya; Annex 3 Discusses one health approach and antimicrobial resistance; Annex 4 describes how to calculate drug dosages in livestock using weight estimates and Annex 5 is a pictorial guide of some medicinal and poisonous plants identified by livestock keepers in Marsabit County.

It is the author's sincere expectation that the handbook will prove to be a practical field companion for animal health practitioners in Marsabit county.



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### Chapter 1: Introduction to Infectious Livestock Diseases

### 1.1 Glossary of Terminologies

Abortion	Premature expulsion of the foetus and foetal membranes. The termination of pregnancy. Abortion is one of the indicators of infertility in livestock.	
Abscess	Localised lesion comprising of pus, surrounded by inflamed tissue.	
Acute	A disease characterised by sudden/rapid onset, short course (3-14 days), severe symptoms and either causes death quickly or leads to a speedy recovery.	
Aetiology / Etiology	Study of disease causative agents	
Alopecia	Loss of hair, wool or feathers	
Anaemia	A reduction in the number and/or size of the red blood cells or the haemoglobin in the blood	
Anorexia	Loss of appetite	
Anuria	Ceasation of urination	
Antibody (Ab)	A substance in the blood serum or other body fluids formed to exert a specific restrictive or destructive action on bacteria, their toxins, viruses, or any foreign proteins	
Antibiotic	Chemical compound derived from living organisms or synthesised from biological material that is capable, in small concentration, of inhibiting the life process of micro-organisms particularly bacteria	
Antigen	A toxin or other foreign substance which induces an immune response in the body especially the production of antibodies	
Ascites	Accumulation of fluid in the abdominal cavity/ peritoneal sac	
Ataxia	Incoordination of gait	
Attenuated	Term used to describe a reduction in the virulence of a micro-organism, particularly applied to those incorporated in Vaccines.	
Bacteria	One celled microscopic organism that don't require a host to survive, they are free living, they reproduce/multiply by binary fission and do not have an organized nuclei or any other membrane-bound organelles.	
Bacteraemia	Presence of non-multiplying bacteria in blood	
Carcass	Body of a dead animal or bird	
CDR	Community Disease Reporters are livestock keepers selected by the community to identify certain syndromes indicating occurrence of notifiable diseases and report these syndromes to the county veterinary staff.	
Chronic	A disease whose course is more than 4 weeks and signs are not severe	
Coma	State of unconsciousness in an animal or bird	
Colic	Abdominal pain	

Table 1: Glossary of terminologies

Congestion	Accumulation of blood in a body part due to overfilling of blood vessels.	
Contagious/ infectious	The spread of contagion or infection from animal to animal by direct or indirect contact.	
Cystitis	Inflammation of urinary bladder	
Dandruff/ Ptyriasis	Condition characterised by presence of bran like scales over the skin surface.	
Dermatitis	Inflammation of skin tissue	
Dehydration	Excessive loss of water or other fluids from the body. Typically through either vomiting and/or diarrhoea or inappropriate intake of water into the body (decreased thirst)	
Diagnosis	An art of confirming a disease with the help of case history, symptoms and laboratory tests	
Diarrhoea	Frequent passage of loose faeces	
Disease	Deviation from a normal state or function in animals that produces specific symptoms or that affects a specific body organ or system and is not directly due to physical injury.	
Dysentery	Frequent passage of loose faces mixed with blood, mucus and accompanied by straining	
Dysphagia	Difficulty is swallowing	
Dysuria	Difficulty in urination	
Ectoparasites	Live on the skin of animals and include Ticks, Mites, lice, fleas and to some extent biting flies. Some of them are visible and can be seen with the naked eye while others are microscopic.	
EDTA	Ethylene Diamine Tetra Acetic acid (EDTA) is an anticoagulant that is coated into purple top blood collection tubes. EDTA tubes are used to collect blood samples that should not clot.	
ELISA	Enzyme-linked immunosorbent assay (ELISA) is a laboratory technique used to measure the antigen-antibody concentration in a sample.	
Emaciation	Excessive loss of body condition / weight due to disease	
Endemic / Enzootic	Endemic refers to a disease that is present in a livestock population at all times while Enzootic refers to an outbreak of an endemic disease among livestock populations in a particular locality (Village or Ward)	
Epidemiology	Study of disease in populations and of factors that determine their occurrence	
Epidemic / Epizootic	A disease that spreads rapidly to affect a large population of livestock in a large geographical area (sub-county, county, country) at the same time.	
Erythema	Reddish discolouration of the skin	
Fever	Rise in body temperature associated with infection	
Fomites	Inanimate objects or materials such as clothes, utensils, and furniture which are likely to be contaminated with infectious agents	

Fungi	Fungi (plural Fungus) are single celled or multicellular organisms that are free living and include moulds and yeasts
Healthy	Describes the physical, physiological and mental wellbeing of an animal
Haematuria	Blood in urine
Haemoglobinuria	Haemoglobin in urine
Hepatitis	Inflammation of the hepatic (liver) cells
Host	Living organism that is susceptible to or harbours an infectious agent under natural conditions. host factor is an intrinsic factor (such as age, species, breed or) that influences exposure, susceptibility or response to a pathogenic agent. Reservoir host
Hyperaesthesia	Increased sensitivity / response to external stimuli like touch, noise and light
Hypersalivation	Excessive production of saliva due to disease condition
Hypothermia	Decrease of the normal body temperature associated with terminal disease state
Incoordination	In ability to move the limbs in normal sequence
Incubation period	The time that elapses between infection or exposure to a pathogenic agent and appearance of signs of a disease
Incidence (Attack rate)	Number of newly diagnosed cases of a disease. An incidence rate is the number of new cases of a disease divided by the number of animals at risk for the disease.
Infection	Invasion of body tissues by pathogenic organisms resulting in disease
Inflammation	Physiological response to injury or infection where the part of the body becomes reddened, swollen, hot and painful. Inflammatory means causing inflammation.
Immunology	Study of the body's reaction to the presence of foreign and pathogenic substances.
Immunisation	Process of artificially producing resistance to a given infection generally by means of a vaccine or an antiserum or antitoxin
Immunity	The power of the body to resist infection or the action of certain poisons.
Jaundice	Yellowish discolouration of the visible mucous membranes of the body
KEVEVAPI	The Kenya Veterinary Vaccines Production Institute (KEVEVAPI) is a government institution mandated to manufacture and supply of affordable veterinary vaccines in Kenya
Laboured breathing / Dyspnoea	Difficulty in breathing due to disease condition
Lacrimation	Excessive production of ocular discharge due to a disease condition
Lesion	Visible changes in size, shape, colour or structure of a body organ
Microbes	Microscopic organisms that cause disease. They can be bacteria, viruses, protozoa and fungi. Microbes are also called disease causative or pathogenic agents
Mode of transmission	Manner in which a pathogenic agent is transmitted from its reservoir to a susceptible host

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Morbidity	State of being symptomatic or unhealthy due to disease or condition.
Mortality	Means death. Mortality rate is a measure of the frequency of occurrence of death among a defined population during a specified time interval.
Necrosis	Death of body tissue, occurs when too little blood flows to the tissue. This can be due to physical injury, infection, radiation, or chemical exposure
Nephritis	Inflammation of kidney
Nodule	Palpable, solid swelling or aggregation of inflammatory cells that is greater than 1 cm in diameter. Nodules are usually found in the dermal or subcutaneous tissue, and the lesion may be above, level with, or below the skin surface.
Notifiable	A disease that, by law, must be reported to public health authorities upon diagnosis
Nystagmus	Involuntary constant jerky movements of the eye ball
Oedema	Abnormal accumulation of fluid in the intercellular spaces /tissues
Opisthotonus	Dramatic abnormal posture due to spastic contraction of the extensor muscles of the neck, trunk and extremities. This results in an upward deviation of the neck that causes the head to be held backwards and facing the sky.
Pathophysiology	the body function changes that accompany a particular syndrome or disease
Pandemic / Panzootic	Global spread of an epidemic disease
Parasite	Organism that benefits by nourishing itself at the expense of the host
Paralysis	Incomplete or complete loss of nervous control over any bodily function
Pallor	Unhealthy pale (white) colour of the visible mucous membranes
Pathogenic	Causing or capable of causing disease pathogenic microorganisms
Per-acute	Disease characterised by a very short course of a few hours to 48 hours with very severe symptoms
Pica	Depraved/ Abnormal appetite
Pharyngitis	Inflammation of pharyngeal mucosa
Post-mortem	Examination of a carcass
Polydipsia	Excessive thirst
Predisposing factor	Anything that makes the animal more prone to infection. Stress lowers immunity hence predisposes animals to infection
Prevalence	Number of disease cases or events among a given population. Prevalence rate is the proportion of a population that has a particular disease at a specified point in time.
Pot-belly	Pendulous abdomen that causes the lower part of the abdomen to assume a convex shape
Prognosis	Forecast regarding to outcome of the disease
Protozoa	Microscopic single celled free living parasitic organisms
Pruritus	Sensation that gives rise to the desire of scratching /itching

Purulent	Containing pus	
Pyuria	Pus in urine	
Recumbency	Abnormal state of animal lying down associated with difficulty in rising	
RT-PCR	Reverse Transcriptase Polymerase Chain Reaction (RT- PCR) is a laboratory confirmatory diagnostic test used to detect specific genetic material of antigens in samples. The test converts the antigen RNA molecule in the sample into its complementary DNA (cDNA) sequence by reverse transcriptase.	
Scab	Dried inflammatory exudate such as blood, pus and skin debris	
Septicaemia	Presence of multiplying bacteria and toxins in blood	
Shock	State in which there is generalised acute and serious reduction in the perfusion of tissue due to reduced blood circulation and pressure.	
Spasm	Involuntary contraction of group of muscles	
Sporadic	Disease that affects a few (one or two) animals in a herd and shows little or no tendency to spread within the herd	
Stomatitis	Inflammation of the oral mucosa	
Sudden death	Unexpected death of an apparently healthy animal	
Symptom	Outward clinical manifestation of disease	
Transmission	Passing of a disease causing agent from an infected individual or group to previously uninfected individual or group.	
Treatment	Curative drug and symptomatic treatments are drugs to get rid of symptoms	
Tenesmus	Straining to pass faeces	
Tetany	Tonic symmetrical muscular contractions	
Tremor	Jerky involuntary contractions of muscles	
Trismus	Lock-jaw	
Toxaemia	Presence of toxins in blood	
Vaccine	Suspension of attenuated or killed disease causing bacteria, virus or rickettsia administered for prevention of infectious diseases	
Vaccination	Protective inoculation with a vaccine to induce immunity	
Virus	Viruses are the smallest of all microbes. They are unable to exist outside the body of the host. They are sometimes referred to as microscopic particles.	
World Organisation for Animal Health (WOAH)	World Organisation for Animal Health (WOAH) formerly known as OIE, is an inter-governmental organisation responsible for improving global animal health. WOAH publishes health standards that are used by the World Trade Organisation (WTO).	
Zoonotic	Disease transmitted from animal to humans and vice versa.	

### Chapter 1: Introduction to Infectious Livestock Diseases

### 1.2 General Appearance of Heathy and Diseased Livestock

#### Table 2: Parameters of healthy and diseased livestock

Parameter	Healthy	Diseased
1. Feed and water intake	Normal	Reduced or absent
2. Rumination	Frequent	Less frequent or stops
3. Body condition and hair coat	Good body condition with smooth and glossy hair coat	Emaciated with rough and dull hair coat
4. Demeanour	Alert with head facing forward and raised	Dull with head facing downwards or facing the side if recumbent
5. Gait	Coordinated movement and ambulates with ease	Difficult slow movement or recumbency
6. Eyes	Bright, moist and normally open	Dry or have profuse lacrimation and partially or completely closed
7. Ears and tail movement	Ears are erect and both ears and tail move frequently	Dropping ears and both ears and tail move less.
8. Mouth and Muzzle	Muzzle is moist and mouth is wet with no odour	Dry muzzle, mouth with hypersalivation and has a bad odour
9. Nose	No discharge	Discharge present
10. Dung and Urine	Dung is semi-solid. Urine is slightly yellow	Hard or loose dung. Urine is dark yellow, coffee coloured or pinkish

### 1.3 Aetiology and Epidemiology of infectious livestock diseases

The success of any livestock operation is closely related to the disease level of the animals. Losses due to disease outbreaks can be classified as direct such as death, medication costs, and restriction to markets or indirect due to poor growth, low production levels, poor feed conversion rates and downgrading of livestock products. Disease is defined as any change in the state of an animal or its organs that affect the proper performance or its normal function.

#### How Infectious Diseases Are Spread

Common ways infectious diseases are introduced into and spread within herds or flocks are listed below;

- I. Introduction of diseased animals or birds.
- II. Introduction of healthy animals or birds that have recovered from disease but are still carriers and can still transmit the disease to others.
- III. Contact with inanimate object such as (fomites) that are contaminated with disease organisms. Fomites include feeds, vehicles, clothing, feeding and watering troughs.
- IV. Contact with carcasses of dead animals that have not been properly disposed.
- V. Contaminated surface water, pastures or soils.
- VI. Rodents and free-flying birds.
- VII. Arthropod vectors like Mosquitoes.
- VIII. Airborne organisms through aerosol droplets especially in heavily populated livestock areas.

## General measures for prevention of infectious diseases

I. Identification and isolation of infected

and incontact animals or birds

- II. Treatment of affected animals
- III. Slaughter of animal suffering from incurable disease
- IV. Disposal of dead animals either through burning or deep burial
- Regular disinfection of contaminated areas like feeding and watering troughs and chicken houses.
- VI. During outbreaks restrict movement of infected animals into clean areas (Quarantine and close livestock markets)
- VII. Control access of infected animals to communal pasture and watering areas. Infected animals can be watered last and troughs disinfected after use.
- VIII. Regular use of anthelmintics and acaricides to control ecto and endoparasites.
- IX. Ensure access to balanced nutrition
- X. Develop a vaccination schedule and ensure annual vaccination of animals and birds before an outbreak occurs and when animals or birds are in good body condition.

Diseases can be classified into 5 classes or groups based on;

#### A. Mode of Origin

i. Hereditary diseases- transmitted from parents to the offspring

- ii. Congenital diseases- acquired during intra-uterine life
- Acquired diseases- transmitted after birth

#### B. Causative agent

#### i. Infectious diseases

Infectious diseases are caused by pathogenic

microscopic pathogens also known as microbes that include bacteria, rickettsia, viruses, protozoa and fungi or macroscopic agents such as helminths (worms), liver or rumen flukes, lice and ticks.

#### ii. Non-infectious diseases

Non-infectious disease is caused by physical, chemical or poisonous agents, nutritional deficiency or disturbed metabolism.

#### iii. Non-specific diseases

Diseases whose cause is indeterminate or has multiple causes.

#### C. Mode of spread

#### i. Contagious disease

Transmitted by means of direct or indirect contact with diseases animal or birds. All infectious diseases may or may not be contagious but all contagious diseases are infectious.

#### ii. Non-contagious disease

Do not spread by means of contact for example tick borne infections

#### D. Duration and severity

#### i. Peracute disease

Characterised by short course (less than 24 hours) and associated with severe symptoms that are often fatal for example Anthrax.

#### ii. Acute disease

Characterised by sudden onset and a short course of 2 to 14 days with severe symptoms that may be fatal if early treatment intervention is not given for example Haemorrhagic Septicaemia

#### iii. Sub-acute disease

Characterised by symptoms that are less severe and a course of 2 to 4 weeks, for example Foot and Mouth Disease in Bos Taurus breeds or Sub-Acute Mastitis

#### iv. Chronic disease

Characterised by a course of more than 4 weeks with signs that are not severe for example Fascioliasis, Trypanosomiasis.

#### E. Geographical area of spread

#### i. Sporadic disease

Affects one or two animals in a herd or flock and has no or little tendency to spread for example Johne's disease or Rabies

ii. Enzootic/ Endemic disease Endemic refers to a disease that is present in a livestock population at all times while Enzootic refers to an outbreak of an endemic disease among livestock populations in a particular locality (village or ward). Example is Haemorrhagic Septicaemia in Camels

iii. Epizootic/ Epidemic disease Refers to a disease that affects a large population of animals in a large area at the same time and spreads rapidly. Examples, Sheep and Goat Pox, Foot and Mouth disease and New castle disease. iv. Panzootic/ Pandemic disease Wide spread epidemic disease with world-wide distribution such as bird flu.

## Detailed description of Five disease causative agents

#### I.Viruses

Viruses are the smallest of all microbes and cannot be observed with a light microscope. Viruses are unable to reproduce and function outside the body of living things hence the reason they are sometimes called microscopic particles instead of single celled organism. Due to their very small size they can easily be carried in dust, water and air. Viruses exist everywhere on earth. They are present in animals, plants, and other living organisms. Unlike bacteria which affect multiple livestock species most viruses are species specific. This explains why a virus that causes illness in sheep and goats may not affect a cow. Viruses have a core of genetic material, that is either DNA or RNA. The core is covered with a capsid, a protective coat made of protein. Around the capsid, there may be a spiky covering known as the envelope. These spikes are proteins

that enable viruses to bind to and enter host cells. Viruses hijack the host cell and commandeer it to replicate virus particles. This causes the cell to die and release viral particles that infect other cells and the multiplication process continues damaging the tissue whose cells have been infected. As they multiply viruses especially RNA have "copying errors" resulting in genetic changes known as mutations. This is a natural process that results in one virus having many antigenically distinct strains. Sometimes there is no cross-immunity among the different strains and animals that recover from infection with a specific strain are susceptible to the other strains.

Viruses come in different shapes as shown in the figure below.



#### Field based diagnosis of viral infections

Viruses are too small to be visualised by light microscopy. The most common tests used in the field to support clinical diagnosis of viral infections are rapid tests. Rapid diagnostic tests (RDTs) are easy-to-use tests as they provide quick results, usually in 20 minutes or less. Unlike most standard tests, which have to be sent to a lab, rapid tests are done and provide results at the point of care (POC). The use of rapid diagnostic tests in the field can greatly enhance disease surveillance, confirm clinical case and facilitate rapid response activities, especially in resource poor settings. The RDTs are mainly lateral flow cassettes based on immunoassay chromatography technology. These tests involve the interaction of a fixed reagent of either target antigen or antibody that is linked to some type of visible detector (Colour dye), that reacts with a patient sample. An example is the Foot and Mouth Disease simple lateral-flow assay (LFA) based on a monoclonal antibody (MAb 70-17) that detects FMD virus under non-laboratory conditions. The LFA uses epithelial suspensions from FMD lesions as the test sample.



Figure 2 FMD Lateral rapid Test

Picture source: Bovine Foot-and-Mouth Disease NSP Antibody Rapid Test http://www.lifebioscience.com.au/Life\_bio/documents/InsertsheetofAnigenRapidFMDNSPAb.pdf

#### II. Bacteria

Bacteria are bigger than viruses and unlike viruses they are free living (meaning they exist and multiply on their own outside a host). Bacteria are microscopic single-celled organisms and found everywhere on Earth and are vital to the planet's ecosystems. Bacteria are the most abundant free-living organisms on earth the animal and human body is full of bacteria. It is estimated that the body has more bacterial cells than animal/human cells. Some bacteria are harmful while others are useful. For example, the normal bacteria of the cow's rumen and human large intestine help in digestion of cellulose and fibre respectively. Bacteria in unfavourable environments produce resistant bodies called spores. Spores can remain viable for many years

in soil and cause disease outbreak as is seen in Anthrax and Black Ouarter. Some bacteria produce toxins and impair body functions by destroying tissues for example, Clostridium bacteria that causes Enterotoxaemia, Tetanus and botulism. Bacteria that enter the bloodstream are rapidly removed by white blood cells. However, if there are too many bacteria the white blood cells become outnumber and an infection develop. An infection that is widespread throughout the bloodstream is called sepsis. Bacteria are classified into five groups according to their shapes: Spherical (cocci), rod (bacilli), spiral (spirilla), comma (vibrios) or corkscrew (spirochaetes). The bacteria occur as single cells, in pairs, chains or clusters.



Leptospira

Clostridium tetani

Staphylococci

Bacteria can also be classified based on the Gram Staining properties. The two key features that lead to the differing visualization properties of Gram positive (+) and Gram negative (-) bacterial species is the thickness of the peptidoglycan layer and presence or absence of the outer lipid membrane. The bacterial wall structure affects the cell's ability to retain the crystal violet stain used in the Gram staining procedure which can then be visualized under a light microscope. Gram positive bacteria have a thick peptidoglycan layer and no outer lipid membrane while Gram negative bacteria have a thin peptidoglycan layer and have an outer lipid membrane. Gram positive bacteria have a distinctive purple appearance when observed under a light microscope. This is due to retention of the purple crystal violet stain in the thick peptidoglycan layer of the cell wall. Examples of Gram positive bacteria include Staphylococci and Streptococci species. Gram negative bacteria appear as pale reddish/pink colour when observed under a light microscope. This is because the structure of the cell wall is unable to retain the crystal violet stain and is only coloured by the safranin counterstain. Examples of Gram negative bacteria include enterococci, salmonella and pseudomonas species.



#### Figure 4 Gram Positive (left) verses Gram Negative (right) as observed under a light microscope

#### Field Based Diagnosis of Bacterial infections

Stained smears made from lesions can yield information that is inexpensive and quick to diagnose. The animal health worker only needs access to Gram stain and a light microscope. Readers should look at the reference section at the end of this chapter to access links for further reading on how to prepare and stain bacterial smears. The table below highlight some infectious diseases that can be diagnosed through stained smears.

#### Table 3: Infectious bacterial diseases diagnosed through Gram stained smears

Disease and Species affected	Specimen to collect	Pathogen	Appearance in Gram stained smears
		Staphylococcus species	Gram + cocci, often in clumps
		Streptococcus species	Gram + cocci, usually in chains
Abscesses or suppurative C	Conditions in all livestock	Trueperella pyogenes	
species Pus or exudate		Corynebacterium pseudotuberculosis	Gram + rods
		Pseudomonas aeruginosa	Gram - rods
		Pasteurella multocida	Gram - rods
		Fusobacterium necrophorum	Gram - long, slender, filaments, often staining irregularly
Strangles- Donkeys	angles- Donkeys Nasal swab with pus		Gram + cocci, often in chains
Dermatophilosis (Streptothricosis) Many animals, mainly in sheep, cattle and donkeys		Dermatophilus congolensis	Gram +, filamentous and branching with coccal zoospores arranged two or more across
Clostridial enterotoxaemia Sheep and calves mainly Clostridial intestine of recently dead animal (< 2 hours after death)		Clostridium perfringens	Gram +, fat rods. Large numbers suggestive of enterotoxaemia
Disease and Species affected	Specimen to collect	Pathogen	Appearance in Giemsa stained smears
Anthrax in cattle, sheep, goats, camels, donkeys Blood smear from ear vein		Bacillus anthracis	Purplish, square-ended rods in short chains surrounded by a reddish- mauve capsule
Dermatophilosis (Streptothricosis) Many animals, mainly in sheep, cattle and donkeys		Dermatophilus congolensis	Blue, filamentous and branching with coccal zoospores arranged two or more across



Figure 5 Gram-stained smear from the mucosa of the small intestine (lamb recently dead from enterotoxaemia): large Gram-positive rods of Clostridium perfringens. (×1000)



### Figure 6

Giemsa-stained smear from ovine scab material showing branching filaments and zoospores of Dermatophilus congolensis. (×1000)

Picture source:

Quinn, P.J., Carter, M.E., Markey, B. and Carter, G.R. (2013). Clinical Veterinary Microbiology. Wolf/Mosby, London, 1994. (Textbook)

#### III. Protozoa

Protozoa are microscopic single celled organisms that are free living and found in land and water. However, pathogenic protozoa are obligate intracellular parasites and require to be in a host cell to survive. Ruminants harbour large numbers of protozoa in their fore- stomachs to help in digestion. Most protozoa are harmless. Some species of protozoa, however, are significant as they cause disease in livestock. Most pathogenic haemoprotozoan parasites are intracellular and are found in erythrocytes (red blood cells) or white blood cells.

## Field Based Diagnosis of Haemoprotozoan infections

A routine thin blood smear useful for assessing erythrocyte abnormalities and for detecting the presence of haemoprotozoan parasites. Parasites are most likely to be detected in blood smears during acute infection when the animal has a fever. Once infections become chronic, immunologic diagnostic techniques are more sensitive. Microscopic examination of blood smears is best achieved with Giemsa staining. However, Wright's stain can also be used, it should be noted that commercial stain kits used in many veterinary practices such as Dip Quick Stain, Jorgensen Laboratories, Loveland, CO, www.jorvet.com) will also stain haemoprotozoan parasites when used as directed, but the staining will be of poorer quality.

The following procedure can be followed when Giemsa stain is being used.

#### Figure 7

Thin blood smear preparation for haemoprotozoan parasite filed based diagnosis



Picture source:

Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Text book)

To prepare a thin blood smear, place a drop of blood on one end of a microscope slide and draw the blood into a thin film as shown in Figure 7 above.

#### Giemsa Stain procedure

- 1. Air-dry the blood film, protecting it from flies and other insects if it is not to be stained
- 2. immediately.
- 3. Fix in absolute methanol for 5 minutes and air-dry.
- 4. Dilute stock Giemsa stain 1: 20 with distilled water and flood the film (or place slide in staining
- jar). Fresh stain should be prepared every 2 days.
- 5. Stain for 30 minutes.
- 6. Wash stain away gently with tap water.
- 7. Air-dry; parasite cytoplasm will stain blue, and nuclei will stain magenta.
- 8. The stained blood film can be scanned using the  $40 \times$  objective of the microscope and with the oil immersion lens for greater detail when suspected parasites are found.



Figure 8 Tear drop shaped Babesia bigemina in a Giemsa stained bovine blood smear

### Figure 9

Giemsa stained Equine blood smear showing Tetrad and small ring (Black arrow) forms of Babesia equi (left picture) and Babesia caballi joined pair form (right picture



#### Picture source:

Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell (Textbooks)





Figure 10 Giemsa stained bovine blood smear showing Theileria parva RBC forms (Piroplasms)



### Figure 11

Giemsa stained bovine lymph node aspirate smear showing Theileria parva Schizont forms (Koch's blue bodies (KBBs) in the lymphocyte cytoplasm

Figure 12 Giemsa stained bovine blood smear showing Trypanosome brucei with prominent undulating membrane (U) and sub terminally located kinetoplast (K)

Picture source:

Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)

#### IV. Fungi

The most common pathogenic fungi that cause disease in livestock are known as dermatophytes. Dermatophytes are a group of closely related, septate fungi that require and use keratin for growth. They are confined to the superficial integument including the outer stratum corneum of the skin, claws and hair of animals. They infection manifests as the classical circular lesion known as ringworm. There over 30 species of dermatophytes, however, those affecting animals are mainly from genera Microsporum or Trichophyton. The Infective arthrospores are found in soil or on infected animals they germinate within six hours of adhering to keratinized structures. Minor trauma of the skin and dampness facilitate infection. The dermatophytes are able to hydrolyse keratin thus causing damage to the epidermis, hair shafts, hair follicles and feathers. The nature of the lesions is affected by the virulence of the fungus and the immunological response of the host. Young, old and immunosuppressed animals are susceptible to severe infection. The inflammatory response of the infected animal is harmful to the fungus; this makes the dermatophyte move peripherally towards normal skin. The result is the commonly seen circular lesions (ringworm) of alopecia with healing at the centre and inflammation at the edge. The manifestations of dermatophyte infections can vary and may be summarized as:

- Subclinical or inapparent infections
- Classical round ringworm lesions

• Serious generalized lesions that may be complicated by mange mites or by secondary bacterial infection, in particular by Staphylococcus aureus or S. pseudintermedius

Field based diagnosis of pathogenic fungi

Hair should be plucked from the lesion and the edge of the lesion scraped with a blunt scalpel blade until blood begins to ooze. Plucked hair, skin scrapings (including the scalpel blade) and any scab material should be submitted for microscopic examination.

Preparation of skin scrapping sample

1. Place one to two drops of 10-20% Potassium

hydroxide (KOH) on a microscope slide and add a small amount of the specimen to the drop of KOH and mix well.

- 2. Gently pass the underside of the slide through a low flame of a Bunsen burner /cigarette lighter/match (do not boil or over-heat). This is an optional step
- 3. Place a cover slip on top of the preparation and press down gently and Allow to stand for one to two hours, or even overnight. The time required will depend on the density of the specimen.
- 4. The KOH will partially digest the proteinaceous debris.
- 5. Examine under phase contrast or under the low- and high-dry objectives of a light microscope. Microsporum and Trichophyton species appear as chains of refractile, round arthrospores on hairs.

#### V. Mites

The bodies of mites and ticks are divided into two parts: the gnathosoma, which contains the mouthparts on a false head (basis capituli), and the idiosoma, which comprises the remainder of the animal and to which the legs are attached. After hatching from the egg, mites and ticks pass through larval and nymphal stages before becoming adults. Larvae of ticks and mites have six legs; while nymphs and adults have eight legs. Parasitic mites are usually microscopic, rarely exceeding 1 mm in length. Female mites are larger than males.

Field based diagnosis of parasitic mites

Diagnosis is through deep or superficial skin scrapings. For a deep skin scraping, a dulled, rounded scalpel blade (#10) and the area of skin to be scraped are coated with mineral oil. The site selected for scraping should be at the periphery of a lesion or the predilection site of the suspected parasite. The blade should be scraped back and forth over the skin until capillary bleeding is evident.

However, sample collection for the Demodex mite which is a follicle mite, one needs to form a skin fold by squeezing the skin between the fore and thumb fingers to express the mites before the scraping is done. The debris collected on the scalpel blade is then placed on a microscope slide, the cover slip is then applied and the microscope slide with the sample is examined using the 10× microscope objective. Several slides may need to be examined before mites are found, especially in cases of Sarcoptic infestation. To recover surface mites, such as Cheyletiella, a superficial scraping that does not result in bleeding is made with a scalpel blade coated in mineral oil. Alternatively, a clear acetate tape can be used to collect material. The sticky side of the tape is pressed to the hair coat in an affected area and then placed on a microscope slide, trapping skin debris and mites against the slide and allowing microscopic examination. Brushings from the hair coat may also be examined. If sample is to be examined by a another laboratory for identification, they skin scrapings should be stored in 70% alcohol.

### Figure 13

A 40X image of a normal hair (thin arrow) and a dermatophyte infected hair (thick arrow). The infected hair has ectothrix spores on the outside of the hair shaft



Picture source:

Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)



### Figure 14

Sarcoptes mites are round bodied, the third and fourth pairs of legs are short and do not project beyond the margin of the body No.

Figure 15 Psoroptic mites have an oval body shape with longer legs that project beyond the body margins



Figure 16 Demodex mite with the 'cigar' body shape and short legs that do not project beyond the body margin

Picture source:

Zajac, Anne M.; Conboy, Gary A. (2011). Veterinary Clinical Parasitology. Hoboken, NJ: Wiley-Blackwell. (Textbook)

### Chapter 2: Clinical Examination Guidelines

### 2.1 Purpose of Clinical Examination

- Identify clinical abnormalities
- Determine risk factors that resulted in the disease
- Establish cause/aetiology of the disease
- · Deduce what organ or system is involved

### 2.2 Clinical examination steps

Clinical examination proceeds through a number of steps

- 1. Owner's complaint
- 2. Signalment (age, breed, sex, colour)
- 3. History of the animal/s affected
- 4. History of the herd
- 5. Observation of the environment
- 6. Observation of the sick animal/s at a distance
- 7. Detailed general physical examination of the animal
- 8. Further investigation- Laboratory

General Physical examination of animal using a body region approach (Thermometer and stethoscope are basic and essential diagnostic tools to have)

- Head and neck

and the location, type of lesion present,

- Understand the pathophysiologicaL processes occurring and
- · Gauge the severity of the disease
- Left thorax and abdomen
- Right thorax and abdomen
- Rear/ Tail end
- -Rectal examination,
- External genital -udder, vagina and Scrotum and Penis

Record after general physical examination;

- · Respiratory rate and temperature
- Mucous membranes colour
- Hair/skin coat condition
  - Body condition score
- Any abnormalities like lameness, lymph node enlargement, or distension of the abdomen or wounds. Note the location of abnormality (the body region affected and what side-left or right)

### 2.3 Techniques used to conduct a physical examination

Clinical examination proceeds through a number of steps

- 1. Palpation (touching)
- 2. Auscultation (listening)
- 3. Percussion (tapping)

- 4. Manipulation (moving)
- 5. Ballottement (rebound).
- 6. Visual inspection
- 7. Olfactory inspection

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### 2.4 Normal body parameters

#### **Resting Heart rate**

Specie	Range (beats per minute)
Camel	32-50
Cattle beef	40-70
Cattle dairy	48-84
Goat	70-80
Sheep	70-80
Donkey	36-52

#### **Resting Respiratory rate**

Specie	Range (breaths per minute)
Camel	5-12
Cattle beef	10-30
Cattle dairy	26-50
Goat	12-24
Sheep	16-34
Donkey	12-28

#### **Rectal Temperature**

Specie	Range (°C)
Camel*	34.0- 41.0
Cattle- Beef	36.7-39.1
Cattle-Dairy	38.0-39.3
Goat	38.5-39.7
Sheep	38.3- 39.9
Donkey	36.5-37.8

\* A fully hydrated camel has a diurnal body temperature range of 36 to 38°C. However, a dehydrated camel temperature may fluctuate to 34 to 41°C

### 2.5 Location of palpable superficial lymphnodes of ruminant livestock





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#### Reference

- 1. Clinical Examination of Farm Animals Peter G.G. Jackson, Peter D. Cockcroft Copyright © 2002 by Blackwell Science Ltd.
- 2. The MSD Veterinary Manual https://www.msdvetmanual.com/
- Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_reports/21
- 4. The clinical companion of the donkey (2021) 2nd edition Produced by The Donkey Sanctuary UK.

https://www.the donkeys anctuary.org.uk/what-we-do/for-professionals/resources/clinical-companion

### Chapter 3: Priority Livestock Diseases in Marsabit County

### 3.1 Priority Livestock Species Kept in Marsabit County

Table 4: Local dialect names for type of priority livestock species in Marsabit County

Key: S= Singular, P= Plural							
Livestock Specie Type	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach	
Camel	Gaal	Gaal	Gaal	Ntames (S) Ntamesi (P)	Ekahal (S) Ngaala (P)	Galte (S) Galsam (P)	
Cattle	Loon	Loon	Lolyo	Nkiteng (S) Ngishu (P)	Aite (S) Ngatuk (P)	Gore (S) Gorm (P)	
Sheep	Ola (S) Olich (S) Oliye (P)	Lah (S) Onoo (P)	Lah (S) Onoo (P)	Nker (S) Nkera (P)	Amesek (S) Ngamesekin (P)	Wala (P) Walach (S)	
Shoats Sheep And Goats	Ree	Ree	Adi	Ntare	-	-	
Donkey	Aree (S) Harele (P)	Aree (S) Harele (P)	Ehel	Sikiria (S) Ngiron (P)	Esikiria (S) Ngisikiria (P)	Guol (S) Olo (P)	
Chicken	Luku	Luku	Luku	Nkokon (P) Nkoko(S)	Ekukut (S) Nkukui (P)	Luge	

Key: S= Singular, P= Plural

### 3.2 Marsabit Communities Seasonal Calendar

Table 5: Local dialect name of the seasons occurring in Marsabit County

Calendar Months	Jan - March	April- June	July- September	October-
				December
Season Name	Short dry	Long rainy	Long dry	Short rainy
Rendille	Nabhai / Nabai Gaban	Guuh	Nabhai / Nabai Deer	Yeer
Samburu	Lamei Dorop	Lgergerwa	Lamei Odo	Ltumuren
Gabra	Bon Agaya	Gann	Adholes	Agaya
Borana	Bon Gaiya/	Gann	Adholes /	Hagay
	Bon Agaya /		Hadholes	Agaya
	Bon Hagaya			
Dasaanach	Margagawa	Irigutua /	Margutwa	Nyerube
	Marr Megaula	Irrgudhuna	Maar Medeera	
Turkana	Akamu Nauriye	Akiru	Akamu Nakooyan	Akiporo /
	Naachi /	Napolan /		Erupe
	Akiporo	Ngururei		
		Hait		

### 3.3 Traditional names for common clinical symptoms

Table 6: Local dialect name for common clinical signs associated with priority livestock diseases in Marsabit County

CLINICAL SIGN	BORANA	GABRA	SAMBURU	RENDILLE	TURKANA	DAASANACH
Dyspnoea Laboured Breathing	Afurat D'iib / Qabam	Afurat D'iib / Qabam	Kedia	Ahudma	Akiyang'a / Nangichan	Himichies
Enlarged Lymph Nodes	Qandichat Itaa	Qandichat Itaa	Kejei Lngarngar	Khanide Yombobta	Aboree -Ang'are	Jaar- Ikiitany
Lameness	Inokol	Inokol	Ngojini	Dokoho	Akichodo	Dhegeny
High Fever	Oww Nafaa/ Qando	Oww Nafaa / Qando	Keirewa Sesen Oleng	Sare A Kulel	Arirare / Eyala	Gonbolo
Hyper- Salivation	Ingoror	Ingoror	Koruko- Sampulal	Marakhach- Oricha	Ngakajela/ Lokajela	Abie
Coughing	Qufasu	Qufasu	Lwata	Yahar	Awala	Ufute /Gufute

CLINICAL SIGN	BORANA	GABRA	SAMBURU	RENDILLE	TURKANA	DAASANACH
Sudden Death	Qurtam	Qurtam	Lbus	Dowa Yamut	Atular	Suony
Emaciation	Uqu / Halu	Uqu / Halu	Saaso	Khocho	Erogo	Nyarog'o
Skin Wounds	Madha	Madha	Ngoldonyot	Jis	Ngajemei	Lilo
Skin Nodules	Kurso	Kurso	Ltukachi	Nyilnyilo	Abuth	Ngerero
Lacrimation	Imimaan	Imimaan	Keishir Nkonyek	Ilmo Oricha	Akigoro Ankonyen	Imiro
Nasal Discharge	Furot Funaani Yaah	Furot Funaani Yaah	Koruko Ngumechin	Sim Oricha	Akaritho	Suuno
Alopecia	Rifensat Luqaa	Rifensat Luqaa	Lputo	Time Masate	Echudanu	Dhuur Hi Gaa Bame / Baalaam Hi Gaa Okode
Foul Body Odour	Inn Ajaa	Inn Ajaa	Kongu Sesen	Naf Anuha	Ebothe	Af Mod
Foot Lesions	Madha Qencha	Madha Qencha	Kedanya Nkejek	Goffane Jisowta	Ngajemei - Angakejen	Bash Komfo / Konof
Oral Lesions	Madha Afaani	Madha Afaani	Kedanya Nkutuk	Aff Jisowa	Ngajemei - Akituk	Af Beedheny
Diarrhoea	Albati	Albati	Nkiriata	Harr	Aremor	Uu
Bloody Diarrhoea	Albati D'igaa	Albati D'igaa	Nkiriata E Lodo	Harri Dige	Aremorit Ngaakot	Uu Bura
Rough Hair Coat	Imbukanoft	Imbukanoft	Keijulus	Hawdhel	Epuriyarit	Hurdhany
Bottle Jaw	Mat'it	Mat'it	Kinyoot	Kinyoot	Lokituk	Af Dhalo
Central Nervous Signs	Sirgo	Sirgo	Sirgo	Sirgo	Ngithurko / Nkerep	Sigire / Gagabiny
Dropping Wings- Chicken	Khool Buft	Khool Buft	Keilili Nkaipupo	Barbare Lugdha	Echakunit - Ngabeben	Kuol Hi Reereme
Snoring/ Respiratory Rales- Chicken	Inkhorift	Inkhorift	Keirung	Ahuricha	Akingorare	Him Hi Chies
Twisted Neck- Chicken	Qacher Thabsit	Qacher Thabsit	Kekordo Murt	Lukhume Kholokhata	Edeke -Eutune	Luut Hi Gaa Gernete

#### Table 6: Local dialect name for common clinical signs associated with priority livestock diseases in Marsabit County

CLINICAL SIGN	BORANA	GABRA	SAMBURU	RENDILLE	TURKANA	DAASANACH
Ruffled Feathers	Inshil Khoft	Inshil Khoft	Keitachoo Nkopir	Tudube Tolosata	Epuriarit	Dhuur Hi Gaa Firay
Paralysis- Chicken	Nafat Iraat Dhue	Nafat Iraat Dhue	Keikanga	Ahanjawa	Etergege	Heljeny
Loss Of Feathers	Kolat Luqa	Kolat Luqa	Kodoyio Nkopir	Tudube Abutamte	Lochud / Echudanu	Balaam Hi Gaa Okode

#### Table 6: Local dialect name for common clinical signs associated with priority livestock diseases in Marsabit County
# 3.4 Traditional names for common ectoparasites

Table 7: Local dialect name of ecto and endoparasites found in livestock in Marsabit County

TYPE ECTOPARASITE	BORANA	GABRA	RENDILLE	SAMBURU	TURKANA	DAASANACH
Camel Nasal Bot	Siridho	Siridho/ Ramo	Sangelen	Lmarsomwa	Ekurut	Aiy Kuudienyle Soonoka
Sheep Nasal Bot	Siridho	Siridho	Sangelen	Lmarsomwa	Ekurut	Aiy Kuudienyle Soonoka
Biting Gnats/ Midges/Small Biting Flies	Bokhe	Bokhe	Bina Bargul	Nkasilei	Asipinyu / Ameribong	Kiidhiny Eeno / Kiidhiny Iriet
Leeches	Hulan Ul	Hulan Ul	Hula Hula	Lmolog	Ngidike	-
Fleas	Tafii	Tafii	Leisusu	Loisusu	Losusui	Kiidhiny
Ticks	Dhirandis / Shilmi	Shilmi / Dhirandis	Chilim	Lmanjer	Emadang	Kinne
Engorged Female	Bununi	Bununi	Turdach	Ltunturi	Emadang	Aar Dhong'ode
Nymph	Dhakho	Dhakho	Chilim Ti Nunuchul	Lmantusa	Nayeye	Kin Umo
Mites	Ch'ni	Ch'ni	Mbipiyan	Mpaipayan	Losaa	Finy
Lice	Injiran	Injiran / Injire	Injir	Lache	Elachit	Edhire
Camel Fly (Hippobosca Camelina)	Kitan	Kitan	Dakar	Ldakar	Lolingakori	Kiidhiny Galte
Horse Fly Tabanus Species (Black Fly)	Qodabe	Qodabe	Soor	Lopupoi	Lopelerengor	Nachuna
Stable Fly Stomoxys Species (Brown Fly)	Sorondis	Sorondis	Soor	Lopupoi	Echut	Suor
Tsetse Fly	D'uuga	D'uuga	Gadhu	Sanampur	Lopelerengor	Suor
Mosquito	Binni	Binni	Mune	Nkajingani	Esurut	Donine

# 3.5 Ethnoveterinary practices used to treat priority livestock diseases

Table 8: Local dialect name for plants perceived to have medicinal value to treat priority livestock diseases

Medicinal plant botanical name and disease indication	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Acacia nubica- bark is soaked in cold water or boiled then sieved. Liquid given as an oral drench for respiratory conditions.	Wanga	Wanga	Holiya	Ldepe	Eiyellel	Morte
Acacia tortilis-the bark is prepared as above to treat Gastro intestinal conditions	Dadach	Dadach	Dahar	Ltepes	Ewoi	Seechgeebe
Zanthoxylum usambarense –crushed seeds mixed with cold water, sieved and liquid given as an oral drench. Used as an antibiotic and to treat coughs	Gadda	Gadda	Gathaha	Loisuk	Eusugu	-
Commiphora species- gum and resins applied to skin wounds and ectoparasite lesions	Warra	Warra	Warra	Warra	Wuara	Kerech
Aloe scabrifolia and other Aloe species – sap drained or leaves crushed and mixed with cold water and given as an oral drench or crushed leaves make a paste and applied topically. Used for deworming, diarrhoea, wounds and ectoparasite infestation treatment	Harges	Harges	Yaharr	Sukuroi	Echuchuka	Dhelbo
Euphorbia kalisina or candelabrum- the spines/thorns are removed and the succulent leaves pounded and soaked in cold water and given as an oral drench	Adama	Adama	Lekule	Ltilemani	Emus	-
Ficus sur- fruits /seeds pounded into a paste applied to skin wounds, abscess and mange lesions (ectoparasite infestation treatment)	Odha	Odha	Odha	Lngaboli	Echoke	-
Medicinal plant botanical name and disease indication	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Azadirachta indica – Neem tree has antibiotic and anti-inflammatory properties. Leaves, bark or crushed dried seeds are soaked in cold water or boiled, sieved and given orally as a drench	Mwarubaini					
Salvadora passica (tooth brush tree)- Natural antibiotic whose crushed leaves or bark are soaked in cold water or boiled, sieved and given as an oral drench	Huda AADEE	Aadde HUDA	Hagay	Sakotei	Esekon	Nyedhe – Singular NYAA- plural
Moringa stenopetala- leaves, bark or dried seeds are soaked in water and boiled, sieved and cooled liquid given as an oral drench	Safara	Safara	Lorsanjo	Waddessa/ LORSANJO / CHIBULUKWA	Etebusoit	Hocholoch

Traditional practice	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Salty water from saline wells, springs or Lake Turkana- Topical application	Magatho	Magatho Muluthe	Magaath	Makaat/ Nkare / Nadwa / Lpolei	Amakat	Shukuta
Bleeding large ruminants from jugular vein to remove 500mls blood. For small ruminants and donkeys cutting base of ear to remove about 200 mls of blood	Idhana	Idhana	Gancho	Ng'oroto	Akugum	Fie
Chewing tobacco - crushed into powder and dissolved in water and given as an oral drench or it is made into a paste and applied topically	Tambo	Tambo	Tombo	Lkumbau	Etaba	Tambo
Hot iron or hot stone cauterisation of affected body region	Guba	Guba	Gubnan	Lminjire	Amachar Akimad	Gui
Traditional practice	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Melted fat from a sheep's tail	Dada Oila	Dada Oila	Suba Ki Iyat	Lata Ee Nker	Akuring'- Amesek	Salab Walach
Take animal to stand in hot sandy area to allow foot vesicles to burst thus speed up healing	Laf Owicho	Laf Owicho	Rorom Omhi	Ntama Ntoitoi	Ewassi Anakolong'	-
Sheep head soup. Head of sheep with hide intact is roasted over a fire. Charred remains are put in a pot, boiled to make a soup that is given orally when cooled.	Futo Mata	Futo Mata	Sar	Sar	Sar	Meeny Walach
Religious prayers- performed by specific clan members who have the spiritual gift of divine intervention	In Ebifatan	In Ebifatan	Bifnaan	Mayan	Akilip Amuronot	Waag Vimi
Cooking oil – topical to wounds	Salidhi	Salidhi	Salad	Silidh	Asilit	Salam
Honey applied topically on lesions	Dhamm	Dhamm	Malab	Naisho	Awoo	Dhaanite
Local brew- or alcohol (Vodka)	Araqe	Araqe	Changaa	Changaa	Epurot	Bersho
Pepper powder	Barbare	Barbare	Pilipili	Lpilipili	Epilipili	Barbara
Old engine oil applied topically	Olio	Olio	Oil	Oil Musana	Akimet Nangorot	Salab Muulle
Boiled Tea (Sturungi)	Qararti	Qararti	Turungi	Turungi	Eturingi	Turungi
Wood ash	Dhara	Dhara	Bembei	Nkuron	Ekuron	-

#### Table 9: Local dialect name for traditional practices used to treat priority livestock diseases in Marsabit county

# 3.6 Ethnoveterinary practices used to control priority livestock diseases

Table 10: Local dialect name of traditional practices used to prevent or control the spread of priority livestock diseases

Control strategy	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Isolation of sick animal and Avoidance of disease outbreak area	GARGAR Dowan / Kesabasan	GARGAR Dowan / Kesabasan	YEROI	OROTO / TEGEIAI	TOTIAK	AANY Baashguoko MALG RIMI
Regular Vaccination	TALALA	TALALA	HUNCHO TI HOLLAT	REMOTO EE SIOM	AKICHUM NG'IBAREN	AANY HIRICH VEERITI
Bush clearing through burning to control ticks	LAF GUBAN	LAF GUBAN	ILL GUBNAN	MPEJETO E NKOP	AKINOM Akop	TIM JIET GAAVEERITI
Hand picking of ticks	SHILMI LRACHIRAN	SHILMI LRACHIRAN	SHILIM BUTNAAN	TUPUTOL LMANJER	AKIDOT NG'IMADANG	KIN BIMI
Clearing manure	KOSI Haraan	KOSI HARAAN	LAG HAAR	TOORO LTULUGUMI	TOMUDA NG'ASIKE	DHIGIT HEEN
Use of Amulet *	ULUGO	ULOGO	ULUKH	ULUKH	AKING'OL APAK	GOOLO
Prayer /divine intervention	IN EBIFATAN	IN EBIFATAN	BIFNAAN	MAYAN	AKILIP / Amuronot	WAAG VIMI
Report outbreak to govt. vet officer	REPOT DAAN	REPOT DAAN	FARNIN EREG	AICHOYO LKILIKWAI	AKIRAM Eropot	DEE LANY
Migrate out of the area with disease outbreak	IN GOTHANAN	IN GOTHANAN	GURNAAN	AIWOT / Naidura	AIWESIT / ARAMAKIN	GURA

\*Lighting two sets of fire and animals pass between the two or ensuring camel boma fence has acacia bark or cutting into half a dead squirrel and placing at the livestock boma entrance

# Chapter 4: Cattle Diseases

Viral Cattle Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Foot And Mouth Disease (FMD)	Oyale	Oyale	Lkulub	Lkulup	Lojaa / Lokituk / Lokulup	Huluf / Guluf
Lumpy Skin Disease (LSD)	Kurtubale / Sukhi	Kurtubale / Sukhi	Nyinyilo	Nariri E Nkishu	Etune/ Edeke- Lomen / Epara	Verte
Bovine Ephemeral Fever (BEF)	Buut	Buut	Lakirkir	Lakirkir	Lokirkir	Birgach
Bacterial Cattle Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Heart Water	Qando / Barochisa / Barodha	Qando	Ririkhanyo	Lmilo	Amil/ Lotai/ Loine	Bash Chira
Listeriosis	-	-	Mboroto	Mporoto	Mporoto	-
Black Quarter (BQ)	Arka	Arka	Nengima Nengeju	Nengima Nengeju	Lokwaat	Dhalo
Haemorrhagic Septicaemia (HS)	Silisa	Silisa	Khanid / Nalgoso	Nolgoso / Nalgarngari	Elkunoit/ Lokudo	Jaaro
Contagious Bovine Pleuropneumonia (CBPP)	Sombes	Sombes	Lkibei	Lkipei Lonk-Ishu	Loukoi -Angatuk	Luoke Guo
Anaplasmosis	Dharabot	Dharabot	Ndis	Ndis	Lonyang	Bash Kinne
East Coast Fever (ECF)	-	-	Lipis	Lipis	Lipis	-
Babesiosis (Red Water)	Birte	Bira	Kati Ti Digee	Nadokula / Noonkula	Lodip /Lokul	Sin Bura
Trypanosomiasis	Awarsa / Gandhi	Awarsa / Gandhi	Omar	Saar	Ekwaakoit	Lesikonye
Parasitic Cattle Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Fascioliasis / Liver Fluke Parasite	Minni Tiru	Minni Tiru	Deyah Ti Helesi	Lkurui Lemonywa	Lomany	Nakarkarsha
Mange	Chito	Chito	Haddo	Lpepedo	Emitina Pepedo / Ekokoi	Gogosich

#### Table 11: Local dialect name of priority cattle diseases in Marsabit county

## 42 Cattle Diseases

#### Table 12: Ethnoveterinary practices used to treat priority livestock diseases in Marsabit county

Traditional practice	Target cattle diseases
Salty water from saline wells, springs or Lake Turkana-	LSD, FMD
Topical application	
Bleeding animal from jugular vein	Trypanosomiasis, Bovine Ephemeral Fever
Chewing tobacco crushed into powder and dissolved in	CBPP, Heart water, Listeriosis
water and given as an oral drench or it is made into a paste	
and applied topically	
Hot iron or hot stone cauterisation	BQ, HS, LSD, Listeriosis
Melted fat from a sheep's tail	FMD, Trypanosomiasis, Listeriosis
Take animal to stand in hot sandy area to allow foot vesicles	FMD, LSD
to burst thus speed up healing	
Sheep head soup with hide intact is roasted over a fire, the	CBPP, HS
charred remains are put in a pot with water and boiled to	
make a soup that is sieved given orally when cooled.	
Prayers for divine intervention are performed by specific	HS
clan members who have the spiritual gift of divine	
intervention.	
*Text in red highlights practices that go against animal welfare	principles

Type of Veterinary drug	Active ingredient	Purpose	Target cattle diseases
Antibiotic	Oxytetracycline	Curative and Preventive for secondary bacterial infections	CBPP, LSD, BQ, HS, Heart water, Anaplasmosis, Babesiosis
Antibiotic	Penstrep® procaine penicillin G and dihydrostreptomycin	Curative and Preventive for secondary bacterial infections	LSD, CBPP, BQ, HS, Anaplasmosis
Antibiotic	Tylosin	Curative and Preventive for secondary bacterial infections	FMD, CBPP, HS
Antiprotozoal	Diminazene Aceturate (Veriben®)	Curative	Babesiosis, Trypanosomiasis
Anthelmintic	Albendazole	Curative	Helminthiasis, Fascioliasis
Anthelmintic	Ivermectin	Curative	Trypanosomiasis, LSD, CBPP, HS, Anaplasmosis
Acaricide	Amitraz Organophosphate Synthetic Pyrethrin (Ectopor®)	Preventive and Curative	Babesia and Heart water
Antiprotozoal	Novidium® (homidium chloride)	Curative	Babesiosis and Trypanosomiasis
Metabolic support	Multivitamin	Support recovery	LSD, Helminthiasis

#### Table 13: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority cattle diseases in Marsabit county

\* The drug of choice used by livestock keepers to treat the diseases in red do not have any therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

# Cattle Diseases: Viral Diseases

# 4.1 Foot and Mouth Disease (FMD)

Disease	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Foot And Mouth	Oyale	Oyale	Lkulub	Lkulup	Lojaa / Lokituk	Huluf /Guluf
Disease (FMD)					/ Lokulup	

## Definition

- Highly contagious viral disease of cattle, swine, sheep, goats and other cloven-hoofed ruminants.
- Characterised by sudden lameness, hypersalivation and appearance of blister-like sores on the tongue, muzzle and between the hooves.

## Distribution (Epidemiology)

- The most important viral diseases of livestock in the world.
- FMD is endemic in Africa, Middle East, Asia and in a few countries in South America.
- North America and Western European countries have eradicated the disease and are currently free of FMD without vaccination.
- Cloven-hoofed wildlife, Bactrian camels and new world camelids are susceptible to FMD.
- The African buffalo is the main reservoir host for FMD virus.
- Based on WOAH estimates, FMD's morbidity rate can be as high as 100% in susceptible populations. Mortality is low in adult animals (1–5%), but higher in young calves and lambs (20%) due to myocardium lesions.
- In Marsabit county, livestock keepers in the year 2022 estimated that FMD has an apparent

annual morbidity rate of 12%, mortality rate of 2% and case fatality rate of 15%.

## Causative agent/ Aetiology

- Foot-and-mouth disease virus (FMDV) is a small, enveloped and single-stranded RNA virus. RNA viruses are prone to mutation errors during replication.
- FMDV belongs to the family Picornaviridae and genus Aphthovirus. The family includes human rhinovirus that causes common cold and poliovirus that causes polio.
- FMDV occurs as seven major distinct serotypes: A, O, C, Southern African Territories (SAT) 1, SAT 2, SAT 3, and Asia 1.
- Each serotype has multiple subtypes with varying antigenicity and degrees of virulence, especially within the A and O types.
- There is no cross-immunity between serotypes-this means that immunity to one type does not confer protection against the others.
- FMDV can persist in contaminated fodder and the environment for up to 1 month, depending on the temperature and pH conditions.
- The virus survives in milk and milk products during regular pasteurisation, but is inactivated by ultra-high-temperature (UHT)

pasteurisation.

• Quickly inactivated by conditions that have a pH of more than 6.0.

## Transmission

## Source of infection

- Virus is shed by incubating animals before clinical signs begin.
- Aerosol droplets, saliva, faeces, urine, milk and semen have high infective dose of virus.
- The virus is also found in meat and meat byproducts that have a pH of below 6.0.
- Carrier, recovered or vaccinated animals can harbour the virus in the oropharynx for more than 28 days. Carrier state in cattle varies from 15–50%.

## **Modes of Transmission**

- Direct contact between infected and susceptible animals through Inhalation of infectious aerosol droplets- FMDV is small and airborne, especially in temperate zones (virus can be carries by wind up to 60 kms overland and 300 kms by sea)
- Indirect contact with contaminated inanimate objects fomites.
- Ingestion of contaminated milk (by calves)
- Artificial insemination with contaminated semen
- Humans can harbour FMDV in their respiratory tract for 24–48 hours and can transmit it to cattle.

## Pathophysiology

- Primary site of replication of FMD virus is the mucosa of the pharynx. The virus then enters the lymphatic system and is spread throughout the body where it replicates in the epithelium of the mouth, muzzle, teats and feet.
- The replication in the epithelium causes vesicles to form that after rupture after 48 hours.
- Once the vesicles rupture the healing process begins.
- Carrier animals can harbour the virus for n more than 4 years in cattle, 9 months in sheep, and over 5 years in the African buffalo.

• The African buffalo is the main maintenance host for FMD SAT serotypes.

## Affected age group

• All age groups and all breeds of cattle are susceptible.

## **Clinical signs**

- Incubation period is variable and depends on the host, environment, route of exposure, and virus strain. The average incubation period for sheep and goats is 3–8 days and 2–14 days for cattle.
- Animals develop a high fever of 40° to 41° C.
- Animals make a smacking sound with their mouth and exhibit hypersalivation. The saliva hangs in form of rope-like strings.
- Animals careful chew the feed and or often seen grinding their teeth due to stomatitis.
- Mouth lesions appear as vesicles that are 1 to 2 cm in diameter. The vesicles are found on the buccal mucosa, dental pad and tongue.
- Vesicles are thin walled and easily rupture after 24 hours releasing fluid.
- The rupture leaves a raw painful surface that heals in 1 week.
- Vesicular feet lesions also appear in the interdigital space, heel of the claws and on the coronet band. The vesicle rupture causing severe lameness and recumbency.
- Secondary bacterial infection interferes with healing especially of foot lesions.
- Mastitis due to vesicles on the teats may also occur.
- Calves under 3 months may suffer heavy mortality due to myocardial damage. Death in calves may occur even when there are no vesicular lesions in the mouth or feet.
- Recovery is often seen after 8 to 15 days.

## **FMD** Complications

- The animal can develop serious bacterial infection of lesions especially if animal is kept in a muddy dung filled environment.
- Animals may develop hoof deformation.
- FMD caused mastitis can cause permanent impairment of milk production, abortion and

permanent loss of weight.

 An unusual symptom in cattle is linked to endocrine damage and is characterised by a chronic syndrome of dyspnoea, anaemia, overgrowth of hair, and lack of heat tolerance. Affected cattle are described as "hairy panters."

#### Post-mortem lesions

- Necrosis of heart muscle (tiger heart), usually only in young acutely infected animals.
- Ulcerative lesions on tongue, palate, gums, pillars of the rumen and feet.

#### Season of occurrence

- FMD outbreaks occur in any season due to introduction of sick animals, presence of naïve (not immune) animals. In pastoralist settings shared pasture and water are a key driver of FMD outbreaks.
- In Marsabit county, FMD is endemic and cases are reported throughout the year. However, epizootic outbreaks are common during the long rains season that falls between the months of April and June.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

## Diagnosis

#### Field based diagnosis

- FMD Point of Care Rapid Diagnosis test kit that is available in Kenya.
- Clinical signs can be used to make a field based presumptive diagnosis. Animal health workers should age lesions as this is an important aspect of outbreak investigation that determines what control intervention measures will be adopted.

#### Laboratory diagnosis

- Laboratory confirmation is essential for diagnosis of FMD and should be performed in specialized laboratories that meet WOAH requirements for Containing Group 4 pathogens.
- In Kenya, all samples are sent to the WOAH FMD reference laboratory located in Embakasi, Nairobi county.
- Samples to be taken should include- vesicular epithelium or fluid. At least 1 g of epithelium should be placed in a phosphate-buffered saline (PBS) transport medium or glycerol.
- Samples should be refrigerated or transported on ice.
- If vesicles are not present, oropharyngeal fluid can be collected via probang cup or pharyngeal swab.
- Clotted and unclotted blood should be collected with red topped and purple topped EDTA tubes respectively.

 Laboratory tests to be conducted include –RT-PCR for antigen serotype or ELISA for antibody level analysis. ELISA antibody serum tests are not useful in areas with vaccinated animals that are experiencing new FMD outbreaks.

#### Differential Diagnosis (DDx)

• Clinically indistinguishable from vesicular stomatitis

#### Other differential diagnosis:

- · Bovine viral diarrhoea and Mucosal disease
- · Infectious bovine rhinotracheitis
- Bluetongue
- Bovine mammillitis
- Malignant catarrhal fever

#### Treatment

- Viral diseases have no treatment.
- Supportive treatment with broad spectrum antibiotics to manage secondary bacterial infections observe drug label instructions for dosage and delivery route.

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM.
Phenyl butazone	Anti-inflammatory Analgesic Anti- pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

\* mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

## Prevention/control

- FMDV is quickly inactivated by pH ranges of 6.0 and 9.0. Disinfectant products that achieve this pH range include; sodium hydroxide (2%), sodium carbonate (4%), citric acid (0.2%), acetic acid (2%) and sodium hypochlorite (3%). These products should be used to disinfect contaminated areas;
- Humans exposed to herds with FMD can harbour the virus in their respiratory tract for 24–48 hours and should be prevented from coming into contact with livestock for 1 week.
- Isolate sick animals and impose movement quarantine in affected areas
- Use vaccines after receiving laboratory results of the circulating serotype. KEVEVAPI has two types of vaccines the Purified oil based FOTIVAX vaccine that requires one annual vaccination. The second vaccine called FOTIVAX requires 2 or 4 annual boosters to be effective.
- Livestock keepers should be advised not to purchase new animals during FMD outbreak (they should wait 6 months after the outbreak).

#### Zoonotic potential

- Yes
- FMD is not readily transmissible to humans and is not a public health risk. However, humans are thought to be slightly susceptible and may develop vesicles in the mouth or hands.

## Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where FMD outbreaks are occurring
- Present animals for vaccination
- Use of Amulet (sacrifice a squirrel cut it into half and put each half at either entrance of the cattle kraal)
- Request for religious blessing to safeguard the herd from disease. The blessing ritual is done by specific people who have being given the divine ability to protect livestock and property from ills like diseases.

#### **Further Reading**

- David J Paton, Simon Gubbins and Donald P King (2018). Understanding the transmission of foot-and-mouth disease virus at different scales. Current Opinion in Virology Volume 28, February 2018, Pages 85-91.
- https://www.sciencedirect.com/science/article/pii/S1879625717300962
- 2. Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. WOAH (World Organisation for Animal Health) ISBN 978-92-9044-804-4. https://rr-asia.WOAH.int/wp-content/uploads/2020/04/WOAH-atlas-of-transboundary-animal-disease\_ahs-chapter.pdf
- 3. KEVEVAPI portal https://kevevapi.or.ke/shop/
- 4. MSD Veterinary Manual https://www.msdvetmanual.com/
- WOAH (2017). Technical Disease Card https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technicaldisease-cards/#searchform-header
- Paul Freeman and herd (2012). Foot and Mouth Disease in Kenya. Online extension publication from Regional Veterinary Officer, NSW Department of Primary Industries. http://www.flockandherd.net.au/other/reader/fmd-kenya.html
- 7. The Progressive Control Pathway for Foot and Mouth Disease control (PCP-FMD) Principles. Stage Descriptions and Standards (2018). http://www.fao.org/3/CA1331EN/ca1331en.pdf



Figure 17 Profuse salivation with saliva hanging in long ropy strings



Figure 18 FMD point of care Rapid Test Kit

Picture source:

https://agritech.tnau.ac.in/expert-system/cattlebuffalo/Foot%20and%20Mouth%20Disease.html



Figure 19 A pictorial guide to estimate the age of FMD lesions

Day 1 –Intact fluid filled vesicles with overlying skin becoming blanched.

Day 2 - Vesicle rupture but the blanched epithelium is still intact with sharp edges (arrowed), and where detached, raw red underlying dermis can be seen.

Days 3-4 - Vesicular epithelium is lost, with subsequent fibrin deposition evident on the exposed dermis. Epithelium starts to re-grow at lesion borders. Day 5-10 - Epithelial regrowth is marked, with loss of fibrin infilling, and subsequent scarification Day 7 onwards - Fibrin infilling has disappeared, with new epithelium covering the dermis. Scar formation progresses.

Picture source:

http://www.fao.org/fileadmin/user-upload/eufmd/ docs/training/EnglLesionageing.pdf

# 4.2 Lumpy Skin Disease (LSD)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Kurtubale/ Sukhi	Kurtubale/ Sukhi	Nyinyilo	Nariri E Nkishu	Etune / Edeke-	Verte
				Lomen / Epara	

#### Definition

- Lumpy skin disease (LSD) is a viral vector borne disease of cattle and buffaloes that is characterised by sudden appearance of nodular skin eruptions.
- The skin nodules are also known as pox lesions.

Distribution (Epidemiology)

- LSD is endemic in Africa since 1929.
- In 2012, LSD spread to the Middle East and South Eastern countries of Europe. Vaccination has stopped further spread in Europe.
- In 2019, LSD spread to Asian countries of Bangladesh, India, China, Chinese Taipei, Vietnam, Bhutan, Hong Kong, Nepal, Sri Lanka, Myanmar and Thailand.
- Disease outbreaks are sporadic and depend on animal movements, animal immune status, wind and rainfall patterns that affect vector populations.
- Based on WOAH estimates, LSD has a varied morbidity rate of 10 to 20% and a mortality rate of 1 to 5%.
- In Marsabit county, livestock keepers in the year 2022 estimated the disease to have an apparent annual morbidity rate of 9% with a mortality rate of 2% and case fatality rate of 23%.

#### Causative agent/ Aetiology

- Lumpy skin disease virus (LSDV) is from the family Poxviridae and genus Capri poxvirus (CaPV).
- It is a DNA virus and is genetically similar to goat and sheep pox virus. LSDV is stable and is resistant to inactivation. LSDV can survive for long periods at ambient temperatures and can remain viable for up to 35 days in dried scabs.

#### Transmission

- Mechanical transmission most common mode - blood feeding vectors like biting flies, mosquitoes and ticks transmit the virus to susceptible animals. The vector species involved in transmission include; Fly species like Stomoxys, Glossina, Muscidae and Tabanidae. Mosquitoes species like Aedes and Tick species like Rhipicephalus and Amblyomma.
- Sexual transmission- the virus is found in semen. Natural mating or artificial insemination is a source of infection for adult female animals.
- Vertical transmission pregnant cows can transmit the virus to the foetus and deliver calves with characteristic nodular skin lesions.
- Direct transmission virus is also found in milk or teat skin lesions and can be directly transmitted to suckling calves.
- Animals are immune for only 3 months after natural infection.
- There is no carrier state and all animals clear the virus form their body after infection.

Affected age group

- All age groups are affected.
- Exotic breeds (Bos Taurus), calves and lactating cows are more susceptible to severe infection.

## **Clinical signs**

- LSD has an incubation period of 2 to 4 weeks.
- Animal has sudden eruption of nodules of various sizes on the skin and mucosa.
- Nodules range from a few to several hundred and are spread all over the body.
- Nodules affect the full skin thickness and involve the epidermis, dermis, subcutaneous

tissue and sometimes the underlying muscle.

- Animal develops a persistent High fever of over 40.5°C for 1 week.
- The superficial lymph nodes especially the prescapular and pre-crural become enlarged.
- Animal have serous nasal discharge and hypersalivation due to the inflammatory nodules developing in the nasal buccal (mouth) mucosa.
- Skin nodules either resolve rapidly, or indurate and persist as hard lumps ('sitfasts').

respiratory tracts and on the surface of almost all internal organ.

## Season of occurrence

- LSD is endemic in Marsabit county and cases are reported throughout the year
- However, epidemic outbreaks are reported during the long dry season that occur between the months of July to September.

## Post-mortem lesions

• At necropsy, the carcass has nodular/pox lesions throughout the entire digestive and

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

## Diagnosis

## Field based diagnosis

- There is no point of care rapid diagnosis test kit developed.
- Characteristic clinical signs are used to make a field based presumptive diagnosis.

Laboratory diagnosis

- Samples to be collected include; skin lesions and scabs, saliva, nasal swabs and clotted and unclotted blood.
- Polymerase Chain Reaction (PCR) is the main confirmatory test used to detect LSDV antigen
- ELISA is used to detect antibodies but it does not differentiate antibodies due to other Capri pox virus (sheep and goat pox virus).

## Differential Diagnosis (DDx)

Disease conditions that have superficially located nodules but have less severe signs:

- Pseudo lumpy skin disease/Bovine herpes mammillitis;
- Insect bites/stings, urticaria, and photosensitisation;

- Pseudo cowpox (Para poxvirus) (lesions occur only on the teats and udder);
- Dermatophytosis (fungus infection);
- Demodicosis (Mite infection)- lesions predominantly over withers, neck, back, and flanks, with alopecia present.
- Bovine papular stomatitis (Para poxvirus) lesions occur only in the mucous membranes of the mouth.
- Dermatophilosis (bacterial infection) lesions associated with prolonged wetting. Skin lesions are raised with matted tufts of hair and spread over the head, dorsal surfaces of the neck and body, and upper lateral surfaces of the neck and chest.

## Treatment

- Viral diseases have no treatment.
- Supportive treatment with broad spectrum antibiotics to manage secondary bacterial infections observe drug label instructions for dosage and delivery route.

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

\* mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

## **Prevention and Control**

- Movement restrictions during outbreaks.
- Annual vaccination before outbreak season. KEVEVAPI produces a vaccine called LUMPIVAX<sup>TML</sup> this is a freeze-dried, live attenuated vaccine that is available in Kenya.
- Vaccine confers immunity for 3 years but annual vaccination is encouraged due to young immune naïve animals in the herd.
- Conduct regular (quarterly) active surveillance using PDS techniques.

#### Zoonotic potential

• None, does not affect man

## Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by

entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary treatment practices

- Refer to tables 12 and 13
- Ethnoveterinary control practices (refer to table 10 for local dialect names)
- Difficult to control due to high population of arthropod vectors, some use pour-on acaricides to control biting flies.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where LSD outbreaks are occurring
- Present animals for vaccination



## Figure 20

Generalised LSD skin nodules of various sizes with enlargement of retropharyngeal lymph node (black arrow)

Picture source: Diba Denge, Saku Animal Health Assistant, Marsabit County



Figure 21 LSD nodules (N) and sit fast (S) lesions

Picture source: Tuppurainen, and Beltrán-Alcrudo 2017



Figure 22 Nodules in the internal organs, bladder (Left) and epiglottis and trachea (Right)

Picture source: Tuppurainen, and Beltrán-Alcrudo 2017

#### **Further Reading**

- 1. Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. WOAH (World Organisation for Animal Health) ISBN 978-92-9044-804-4. https://rr-asia.WOAH.int/wp-content/uploads/2020/04/WOAH-atlas-of-transboundary-animal-disease\_ahs-chapter.pdf
- Kiplagat SK, Kitala PM, Onono JO, Beard PM and Lyons NA (2020) Risk Factors for Outbreaks of Lumpy Skin Disease and the Economic Impact in Cattle Farms of Nakuru County, Kenya. Front. Vet. Sci. 7:259. https://doi.org/10.3389/fvets.2020.00259
- 3. WOAH (2017). Technical Disease Card: Lumpy Skin Disease Terrestrial Manual, Chapter 2.4.14. https://test.WOAH.int/fileadmin/Home/eng/ Health\_standards/tahm/2.04.13\_LSD.pdf
- Tuppurainen, E., Alexandrov, T. & Beltrán-Alcrudo, D. (2017). Lumpy skin disease field manual –A manual for veterinarians. FAO Animal Production and Health Manual No. 20. Rome. Food and Agriculture Organization of the United Nations (FAO). 60 pages.

http://www.fao.org/documents/card/en/c/1fcf63b0-80e9-4f8e-825f-10ea6e998479/

## 4.3 Bovine Ephemeral Fever (BEF)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Buut	Buut	Lakirkir	Lakirkir	Lokirkir	Birgach

#### Definition

- BEF is an arthropod-borne viral disease of cattle and water buffalo
- BEF is a non-contagious disease that manifests as an acute febrile condition that is usually mild in young calves. Adults manifest more severe clinical signs that include; Sudden drop in milk production, bi-phasic fever, hypersalivation, lameness, muscle stiffness and recumbency. The disease rarely causes death.
- Dairy cows can manifest a more severe disease characterised by general depression, muscle tremors, hind limb paralysis that cause prolonged sternal recumbency and death if supportive care is not instituted early. In dairy cows, BEF is also associated with reduced fertility and abortions.

#### Distribution (Epidemiology)

- BEF geographical range includes Africa, Middle East, Australia, and Asia. Inapparent infections (antibodies in serum without overt clinical signs) has been demonstrated in goats, sheep, Cape buffalo, hartebeest, waterbuck, wildebeest, deer and gazelles.
- Seasonal outbreaks, particularly following periods of high rainfall, are indicative of a vector-borne transmission. However, research has failed to identify the range of vectors with most studies implicating biting midges (Culicoides species).
- Based on WOAH, BEF in infected countries occurs throughout the year either as an enzootic or sporadic forms. During epizootic outbreaks morbidity rates are between 50 and 100%) but mortality is low at 1 to 3%.
- In Marsabit county, livestock keepers in the year 2022, estimated that BEF has an apparent annual morbidity rate of 2% and a case fatality rate of 0%.

#### Causative agent/ Aetiology

- Bovine ephemeral fever virus (BEFV) is a member of the genus Ephemerovirus in the family Rhabdoviridae. It is a single-stranded RNA virus.
- BEVF was mainly considered to exist as a single serotype but recent research have demonstrated at least 5 different antigenic strains.
- The virus is readily inactivated at pH levels below 5 and above 10 and is therefore inactivated by the meat maturation process in which the pH falls below 5. The virus is also sensitive to disinfectant chemicals and to lipid solvents.

#### Transmission

- BEFV can be transmitted from infected to susceptible cattle by IV inoculation; as little as 0.005 mL of blood collected during the febrile stage is infective.
- To date, infection by virus obtained from virus culture has not been successful. The virus has been recovered from several Culicoides species and from Anopheles and Culex mosquito species collected in the field. The identity of the whole range of vectors involved in transmission has not been proven.
- Transmission by contact or fomites does not occur.
- The virus does cause a carrier state and recovered cattle clear the virus within a week of infection.
- Infection results in long term immunity.

#### Pathophysiology

• On entry into the body, the virus causes an acute inflammatory reaction that results in neutrophils being released into circulation

within 24 hours.

• The virus also infects the endothelial cells resulting in hypocalcaemia.

## Affected age group

• Disease of adult cattle and water buffalo. All breeds of cattle are susceptible.

## **Clinical signs**

- Signs occur suddenly and vary in severity and include sudden drop in milk production, biphasic to poly phasic fever (40°- 42°C).
  Shivering, in appetence, lacrimation, serous nasal discharge, drooling saliva, pulmonary emphysema (auscultated as harsh lung sounds), increased heart rate and dyspnoea.
- Cessation of rumen movements.
- Clinical signs are mild in water buffalo.
- Affected cattle may become recumbent and paralyzed for 8 hours to more than 1 week.
- Bulls, heavy cattle, and high-lactating dairy cows are the most severely affected, but spontaneous recovery usually occurs within a few days. Hidden losses may result from decreased muscle mass and lowered fertility in bulls.

#### Post-mortem lesions

Bovine ephemeral fever is an inflammatory disease. The most common lesions include:

- Sero fibrinous poly serositis, of varying degrees, in the articular synovial membranes and in the thoracic and peritoneal cavities.
- Serous surfaces may also show signs of bleeding and oedema to varying degrees. The oedema fluid in the thoracic or abdominal cavity contains fibrin.
- In the joints, this peri articular inflammatory fluid is yellow or brown and gelatinous in appearance a Giemsa stain of the fluid reveals extravascular neutrophils.
- Other lesions include pulmonary and lymph node oedema, inflammation of the parietal and visceral pleura, pericarditis (especially at the base of the heart), necrosis at certain points of the skeletal muscles, and, sometimes, emphysematous lesions of the lungs.

#### Season of occurrence

- BEP occurs as an epizootic and is associated with high rainfall seasons that favour the arthropod vector.
- In Marsabit county, BEF also occurs an epizootic common during the long rainy season that falls between the months of April and June.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or	
					Akiporo	

#### Diagnosis

#### Field based diagnosis

- To date there is no rapid diagnostic test developed. Presumptive diagnosis is based on epidemiological factors of proliferation of arthropod vectors and clinical signs of several animals in an area experiencing sudden episode of fever accompanied by arthritis, stiffness of the limbs and lameness followed by rapid recovery.
- A Giemsa stained blood smear can be used as a field based diagnosis test- observing many

immature neutrophils (band cells) in a blood smear can be used as a field based presumptive diagnostic test.

#### Laboratory diagnosis

- Samples to be taken should include whole blood from sick and apparently healthy cattle in affected herds in heparin anticoagulant (not EDTA).
- A blood smear can be tested using Immunofluorescence that identifies the virus or its antigens using immune serum that marks the virus-specific antibodies with

fluorochrome. Confirmation can also be made by detecting the viral genome using RT-PCR or a real-time loop mediated isothermal amplification (RT-LAMP) assay.

#### Differential Diagnosis (DDx)

- BEF should be differentiated from conditions that cause fever, such as Rift Valley fever and bluetongue as they are similar due to link to with arthropod vectors (arboviruses), and both occur during the rainy season.
- BEF should also be differentiated with diseases that cause nervous and respiratory symptoms like ehrlichiosis of ruminants, intoxication by poisonous plants, hypocalcaemia and acute pneumonia that causes pulmonary oedema

Treatment

- Viral diseases have no treatment.
- Treatment includes administration of Nonsteroidal anti-inflammatory drugs (NSAIDs)

so as to pain, reduce inflammation, and bring down the high temperature. Anti-inflammatory drugs should be given early and in repeated doses for 2–3 days so as to be effective.

- Oral dosing of water or drugs should be avoided unless the swallowing reflex is functional.
- Signs of hypocalcaemia should be treated with calcium borogluconate are treated as for milk fever. Antibiotic treatment to control secondary infection and rehydration with isotonic fluids may be warranted.
- Use of antibiotics is discouraged, unless warranted. Use of tetracycline based antibiotics should not be used as it will worsen the hypocalcaemia.
- Use of NSAIDs should be accompanied by supportive care to recumbent cows.

Active ingredient	Indication	Dosage* and Route**
Calcium borogluconate	Hypocalcaemia	Slow IV or SC Cattle & Buffalo: 250 - 300 ml per day as a single dose. Monitor heart function if giving IV.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

\* mL- Milliliter, body weight-BWT, kg- kilograms, through the mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

#### Prevention/control

- Commercially available attenuated or inactivated BEFV vaccines are used in Australia but their effectiveness to induce long term immunity similar to natural infection is highly variable.
- The efficacy of vector control remains uncertain, because the insect vectors have not been fully identified.

Zoonotic potential - None. Notifiable disease - No

• Kenya does not have a BEFV vaccine

#### Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

· Burn dung at the cattle boma to reduce the number of arthropod vectors

#### Further Reading

1. Akakpo A.J. (2015). Three-day fever. Rev. Sci. Tech. Off. Int. Epiz., 2015, 34 (2), 533-538. http://boutigue.WOAH.int/extrait/19akakpo533538ang.pdf

- 2. MSD Veterinary Manual https://www.msdvetmanual.com/
- 3. Stokes, J.E., Darpel, K.E., Gubbins, S. et al. Investigation of bovine ephemeral fever virus transmission by putative dipteran vectors under experimental conditions. Parasites Vectors 13, 597 (2020). https://doi.org/10.1186/s13071-020-04485-5



A dairy cows in sternal recumbency (left) and cow in lateral recumbency with respiratory distress (dyspnoea)-Right Picture



## Figure 24

Bovine blood smear (a) shows normal neutrophils (b) shows neutrophilia with some immature neutrophils (band cells)

#### Picture source:

Albehwar, A.M., Wafaa, M., El-Neshwy, Hiam, M., Fakhry and Hemmat, S. El- Emam (2018). Molecular Characterization of Recent Isolates of BEF Virus in Egypt. Global Journal of Medical Research: Veterinary Science and Veterinary Medicine Volume 18 Issue 2 Version 1.0. https://globaljournals.org/GJMR/Volume18/E-Journal/GJMR/(G)/Vol/18/Issue/2.pdf

## Cattle Diseases: Bacterial Diseases

## 4.4 Heart Water (HW)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qando /	Qando	Ririkhanyo	Lmilo	Amil/ Lotai/	Bash Chira
Barochisa /				Loine	
Barodha					

## Definition

- Heartwater is an infectious, non-contagious, tick borne rickettsial disease of ruminants.
- HW only occurs in areas infested by ticks of the genus Amblyomma.

## Distribution (Epidemiology)

- Heartwater occurs in nearly all the sub-Saharan countries of Africa where Amblyomma ticks are present and in the surrounding islands: Madagascar, Reunion, Mauritius, Zanzibar, the Comoros Islands and Sao Tomé.
- Morbidity rate is highly variable and depends on the degree of tick infestation, previous exposure to infected ticks, and level of acaricide protection. can be as high as in susceptible populations.
- Once signs of the disease have developed, prognosis is poor for exotic breeds of cattle the mortality rate varies from 60 to 80%.
- Recovery from heart water infection usually results in complete immunity against homologous strains, although animals remain carriers of infection.
- In Marsabit county, livestock keepers in the year 2022, estimated an apparent annual morbidity and mortality rate of 4% and case fatality rate of 100%.

## Causative agent/ Aetiology

- Heartwater is caused by Ehrlichia ruminantium (formerly Cowdria ruminantium), it is a s a small Gram negative, pleomorphic coccus.
- Ehrlichia ruminantium belongs to the order Rickettsiales and the family Anaplasmataceae.
- Ehrlichia ruminantium is an obligate intracellular bacterium that does not survive outside the host for more than a few hours. It is heat labile and loses its viability and infectivity in less than 4 hours at room temperature.
- HW can only be transmitted through a tick vector, therefore, parameters associated
- with resistance to physical and chemical actions (i.e. temperature, chemical/ disinfectants, and environmental survival) are not applicable.

## Transmission and Pathophysiology

- E. ruminantium is transmitted by the bite of the Amblyomma variegatum (tropical bont tick) which is a three host tick that requires each stage (larvae, nymph, adult) to feed on a different animal.
- Tick infects the host via bacterial organisms that are found in the tick saliva or regurgitated gut contents.

- Once in the host, the organisms replicate first within the regional lymph nodes with subsequent dissemination via the bloodstream to invade the endothelial cells of blood vessels. In domestic ruminants, there is a predilection for the bacteria to infect endothelial cells of the brain.
- The tick gets infected when it feeds on infected or carrier animals and then transmits the disease to other susceptible animals
- Wild animals play a role as reservoir hosts.
- HW can be transmitted vertically and through colostrum of carrier dams

Affected age group

• All ages of domestic and wild ruminants are susceptible

#### Clinical signs

- Heart water occurs in four clinical forms that are determined by strain virulence, infective dose and host susceptibility.
- Clinical signs are dramatic in the peracute and acute forms. In peracute cases, animals may drop dead within a few hours of developing a fever (41°C or more), sometimes without any apparent clinical signs others display an exaggerated respiratory distress and/or paroxysmal convulsions.
- Cattle that develop the acute disease cases have persistent high fever (41°C) that remains high for 4 to 5 weeks and drops shortly before death. Other signs include anorexia and depression along with congested mucous membranes. Respiratory distress slowly develops along with nervous signs such as a hyperaesthesia, high-stepping stiff gait, exaggerated blinking, and chewing movements. Terminally, prostration with bouts of opisthotonus; "pedalling," "thrashing," or stiffening of the limbs and convulsions before death.
- Diarrhoea is seen mainly in cattle.
- In subacute cases, signs are less marked and CNS involvement is absent or present.
- Recovery from heart water infection usually results in complete immunity against homologous strains.
- Animals remain carriers for long periods.

#### Post-mortem lesions

- Heart water derives its name from the hydropericardium prominent lesions observed.
- Other common lesions are hydrothorax, pulmonary oedema, intestinal congestion, oedema of the mediastinal and bronchial lymph nodes, petechiae on the epicardium and endocardium. Congestion of the brain and moderate splenomegaly.

## Season of occurrence

- Heart water can occur all year round in areas infested by the tick.
- Epizootic outbreaks can occur if naïve animals are brought into a tick infested area.
- In Marsabit county, livestock keepers in the year 2022 indicated that heart water was endemic and cases occur throughout the year, with very little variation across the seasons.

#### Diagnosis

- To data there is no rapid diagnosis test kit developed.
- Clinical signs can be used to make a field based presumptive diagnosis.
- Samples to be collected include clotted blood and in dead animal's the brains tissue from well vascularised section of the brain
- Polymerase chain reaction (PCR) tests can identify E.ruminantium in tissues or in the blood of live animals.
- Heartwater can also be diagnosed by observing E. ruminantium colonies in Giemsa stained brain squash smears from the brain or the intima of large blood vessels.
- The best samples to collect from the brain are well-vascularized portions such as the cerebrum, cerebellum or hippocampus. E. ruminantium occurs as clumps of reddishpurple to blue, coccoid to pleomorphic organisms inside capillary endothelial cells. These organisms are often found close to the nucleus of the endothelia cell and may be in a ring or horseshoe shape.
- Demonstration of colonies of organisms in the cytoplasm of capillary endothelial cells is

necessary for a definitive diagnosis.

#### Differential Diagnosis (DDx)

- The peracute form of heart water can be confused with anthrax.
- Acute form may resemble rabies, tetanus, bacterial meningitis or encephalitis due to babesiosis, anaplasmosis, cerebral trypanosomiasis, or theileriosis
- HW must also be differentiated from plant poisoning

#### Treatment

- Antibiotic treatments should start early in the course of the disease for them to be effective.
- Oxytetracycline is the first choice of antibiotics as it effects good cure rates. Given that most

cases in pastoralists set up are identified late. The first oxytetracycline treatment should be given slowly IV. A minimum of three daily doses should be given regardless of temperature. If fever persists, oxytetracycline treatment should be continued for a fourth and fifth day.

- If the fever still does not decrease, after 5 days of Oxytetracycline use. Switch to a bactericidal antibiotic like potentiated sulphonamide that has been shown to be successful in clearing the fever.
- Ensure owners adheres to the withdrawal times for milk and meat after treatment with antibiotics.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting	Broad spectrum	1 mL per 10 kg BWT slow IV first dose then deep IM for subsequent
Most effective for acute cases	Bacteriostatic antibiotic	doses. Give once a day for 3 days. If fever persists, continue for 4 to 5 days.
Sulfamethoxazole Trimethoprim	Broad Spectrum	15 mg/kg/day/IM twice daily for 3 days
	Bacteriocidal antibiotic	

\* mL- Millilitre, body weight-BWT, kg- kilograms, Intramuscular (IM)

## Prevention/control

- Ticks are the main reservoir of E. ruminantium, and infection can persist in them for 15 months. Amblyomma ticks are three-host ticks whose life cycles take 5 months to 4 years to complete. In general, adult ticks prefer to feed on large ruminants (cattle) and nymphs on small ruminants (sheep and goats).
- Control of tick infestation is a useful preventive measure in some instances but may be difficult and expensive to maintain in pastoralists setting. In addition, excessive reduction of tick numbers, interferes with the maintenance of adequate immunity through

regular field challenge in endemic areas and may periodically result in heavy losses.

- It is recommended that in endemic areas a preventive dosing with long acting oxytetracycline injections be done if new and immune naïve animals are introduced into an endemic area. This will protect susceptible animals from contracting heart water while allowing them time to develop a natural immunity.
- Regular acaricide use through spraying or dipping and hand-dressing followed by inspection to ensure the absence of ticks is recommended for animals in transit to heart water free areas.

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#### Vaccination Challenge

- The infection and Treatment method" (IM) for immunization is in use in southern Africa, where infected sheep blood containing fully virulent organisms of the Ball 3 stock is used for infection, followed by monitoring of rectal temperature and antibiotic therapy after a fever develops. In certain circumstances, the "controlled" infection is followed by preventive "block treatment" without temperature recording for cattle on day 14 [susceptible B taurus breeds] or day 16 [for the more resistant B indicus breeds], sheep and Angora goats on day 11, and Boer and crossbreed goats on day 12).
- The IM vaccine method has several drawbacks such as cold chain constraints and intravenous inoculation with daily follow-up of temperature.
- Young calves (<4 weeks old), lambs, and kids (<1 week old) have an innate age-related resistance to heartwater, so if challenged by natural or induced infections within this time period, most recover spontaneously and develop a reasonable immunity.
- The IM vaccine does not protect animals from all field strains, and revaccination is risky due to the possibility of anaphylactic reactions.
- Research is ongoing to develop a better vaccine using attenuated or inactivated organisms. Field trials have revealed that antigenic diversity is important in formulating effective vaccines, and further investigations are critical for the delivery of any vaccine in the field.

#### Zoonotic potential

• None

#### Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- As it is tick borne and ticks are endemic in the area, livestock keepers if the resources are available ensure once a month spraying with acaricides.
- Some livestock keepers also hand pick the bont ticks from animals.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

#### **Further Reading**

- 1. MSD Veterinary Manual https://www.msdvetmanual.com/
- 2. World Animal Health Organisation ( WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/ animal-diseases/technical-disease-cards/#searchform-header

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Figure 25 Role of Amblyomma species tick in Heart water transmission cycle



Picture source:

 $https://za.virbac.com/home/every-health-care/page content/every-advices/heartwater-disease-in-livestock.html \label{eq:linear}$ 



Figure 26 Hydrothorax in cattle due to heartwater infection

Picture source: https://repository.up.ac.za/handle/2263/6957



Figure 27 Opisthotonus and "pedalling" of the limbs due to heart water infections in cattle

Picture source:

: https://www.msdvetmanual.com/generalized-conditions/heartwater/overview-of-heartwater



Figure 28 Ehrlichia ruminantium parasite colonies (Purplish staining) near the endothelial cell nucleus

## 4.5 Listeriosis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
-	-	MBOROTO	MPOROTO	MPOROTO	-

## Definition:

- Listeriosis is a sporadic bacterial infection that affects a wide range of animals, including humans and birds.
- The most common form of the disease in adult cattle is encephalitis specifically rhombencephalitis which is refers to inflammatory diseases affecting the hindbrain (brainstem and cerebellum).
- The disease is essentially a localized asymmetric infection of the brain stem that develops when L monocytogenes ascends the trigeminal nerve.
- Clinical signs vary according to the functional damage of neurons. Most common signs are unilateral and include depression, facial paralysis, circling, excitement, and recumbency.

## Distribution, aetiology/causative agent

- Listeriosis occurs worldwide, more frequently in temperate and colder climates.
- Main source of infection is carrier animals that harbour the bacteria in their intestines.
- The genus Listeria is composed of grampositive saprophytic bacteria widely distributed in the environment. including soil, water, sewage, decaying vegetation, feed, and human and animal faeces.
- There are 17 species identified. Listeria monocytogenes is the species usually associated with animal and human disease. Other species L. ivanovii or L. innocua have also been linked to ruminant and human disease.
- Contaminated dairy products are one of most common foods involved in human listeriosis

outbreaks. Milk pasteurisation has reduced the risk, even though cases continue resulting from post processing contamination and consumption of raw milk products.

- Among ruminants, bovine species has the highest prevalence of carrier animals that shed bacteria asymptomatically.
- The main source of L. monocytogenes infection in ruminants is poorly fermented silage but there are reports of listeriosis in grazing ruminants,
- In Marsabit County, Listeriosis infections are associated with cattle grazing decaying grass that is being inhabited by wild rats. Focus Group discussion indicated that livestock keepers believe that the urine of the rats contaminates the grass and cause the disease. The disease mainly occurs in Laisamis sub county due to presence of forested areas due to the geographical features comprising of hills and mountain ranges such as Ol Donyo Ranges and Mt. Kulal.
- Based on WOAH reports, the disease has a low morbidity and mortality rate of less than 10% and a case fatality rate of between 30 and 50%.
- In Marsabit county, livestock keepers from the Rendille, Samburu and Turkana communities in the year 2022, estimated an apparent annual morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25%.

#### Seasonal occurrence

 In Marsabit county incidences are sporadic affecting one or two animals. Most cases occur during the long dry season when cattle migrate to forested areas that have decaying grass.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

## **Clinical signs**

- Initially, affected animals are depressed, and disoriented. They may propel themselves into corners, lean against stationary objects, or circle toward the affected side. Facial paralysis with a drooping ear, deviated muzzle, flaccid lip, and lowered eyelid often develops on the affected side, as well as lack of a menace response and profuse, almost continuous, salivation; food material often becomes impacted in the cheek because of paralysis of the masticatory muscles. Terminally affected animals fall on the side and exhibit involuntary peddling movements as seen in Heartwater disease.
- Other signs in cattle include abortion, neonatal septicaemia, keratoconjunctivitis/uveitis and enteritis.
- The uterus of all domestic animals, especially ruminants, is susceptible to infection with L monocytogenes at all stages of pregnancy, which can result in placentitis, foetal infection and death, abortion, stillbirths, neonatal deaths, metritis, and possibly viable carriers. The metritis has little or no effect on subsequent reproduction; however, Listeria may be shed for 1 month or longer via the vagina discharges and milk.

## Post-mortem lesions

- Gross lesions in adult cattle are few except for congestion of meninges.
- In calves (age <3 weeks), they have focal

hepatic necrosis and marked haemorrhagic gastroenteritis.

• Aborted foetuses have marked autolysis, clear to blood-tinged fluid in serous cavities, and numerous small necrotic foci in the liver. Necrotic foci may be found in other viscera such as the lung and spleen.

## Diagnosis

- Gram-stained smears of abomasal contents in calves and foetus reveal numerous grampositive, pleomorphic coccobacilli.
- In adults, diagnosis is based on clinical signs of asymmetric brain-stem dysfunction with depression that is confirmed by means of bacterial culture or immunofluorescence assay.

## **Differential Diagnosis**

- Rabies
- Heartwater
- Ketosis
- Polioencephalomalacia (Thiamine deficiency)
- Brain abscess
- Coenurosis- present with circling, contralateral blindness, and proprioceptive deficits; however, they show no cranial nerve deficits.
- Vestibular disease is common in growing ruminants; these animals typically show nystagmus but they remain bright and alert without facial paralysis.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting Most effective for acute cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM every 24 hours for 3 to 5 days.
Penstrep Procaine penicillin G200000 IU. Dihydrostreptomycin sulphate200 mg	Broad spectrum Bacteriocidal antibiotic	1 ml per 20 kg BWT IM only for 3 to 5 days.
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL Calves: 5-10 mL IM or SC

\* mL- Milliliter, body weight-BWT, kg- kilograms, Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC)

## Zoonotic risk

- Yes,
- Samples from suspected clinical cases of listeriosis have a risk of zoonotic infection and should be handled with caution. Aborted foetuses and necropsy of septicaemic animals present the greatest hazard. Ruminants with meningoencephalitis have the organism confined to the brain and presents little risk of transmission, unless the brain is handled during necropsy.
- Humans have developed fatal meningitis, sepsis, and papular exanthema on the arms after handling aborted material.
- Pregnant women should be protected due to the risk of abortion, stillbirth, and infection of neonates.
- Although listeriosis in humans is rare (upper estimate of 12 cases per million populations per year), mortality can reach 50%. Most human cases involve older patients, pregnant women, or immunocompromised individuals. L monocytogenes can be isolated from milk of mastitic, aborting, and apparently healthy cows. Excretion in milk is usually intermittent but may persist for many months. Infected milk is a hazard, because the organism may survive certain forms of pasteurization. Listeria has also been isolated in cheeses, from sheep and goats.

## Notifiable disease

• No

## Prevention and control

- There is a vaccine available but due to sporadic nature of disease occurrence it is not commercially available in Kenya.
- Immediately remove the herd from the area with contaminated grass or withdraw contaminated silage.

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

· Avoid areas with decaying grass

#### Further Reading

 Carolina Matto, Gustavo Varela, Maria Ines Mota, Ruben Gieneechini and Rodolfo Rvero (2017). Rhombencephalitis caused by Listeria monocytogenes in a pastured bull. Journal of Veterinary Diagnostic Investigation, Volume: 29 issue: 2, page(s): 228-231.

#### https://doi.org/10.1177%2F1040638716689116

2. MSD Veterinary Manual https://www.msdvetmanual.com/



Figure 29 Listeria monocytogenes keratoconjunctivitis and uveitis in dairy cattle

A- anterior uveitis, yellowish anterior chamber fluid, opaque cornea, epiphora and hyperaemic conjunctiva. B- bluish corneal opacity and white focuses on the cornea, floccules of material attached to the endothelium of the cornea, anterior uveitis, and hyperaemic conjunctivae

Picture source: https://www.semanticscholar.org/paper/LISTERIA-MONOCYTOGENES-KERATOCONJUNCTIVITIS-AND-IN-Stari%C4%8D-Krizanec/ a36210dd2954c9562718a632c5909f710b6b9d5a



## Figure 30

Tissue smear of post-mortem brain stem samples of an animal with Listeriosis

Note the presence of large numbers of the V-shaped gram-positive bacterium Listeria monocytogenes (Gram stain, high power)

Picture source: https://www.msdvetmanual.com/generalized-conditions/listeriosis/listeriosis-in-animals



# 4.6 Black Quarter (BQ)

## Other names- Clostridial myositis; Black leg; Quarter evil

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
ARKA	ARKA	NENGIMA NENGEJU	NENGIMA NENGEJU	LOKWAAT	DHALO

## Definition

 Blackleg is an acute, febrile, highly fatal, disease of cattle and sheep caused by Clostridium chauvoei and characterized by emphysematous swelling and necrotizing myositis that commonly affects large muscles.

## Distribution (Epidemiology)

Global distribution

## Causative agent/ Aetiology

- C. chauvoei is found naturally in the intestinal tract of animals.
- It is a Gram-positive, anaerobic bacterium which produces endospores and toxins in favourable conditions.

## Mode of transmission and Pathophysiology

- Cattle and sheep are susceptible as they are grazers and come into contact with C. chauvoei spores that remain viable in the soil for years. Spores are the main source of infection.
- Outbreaks of blackleg has occurred in farms which had recent excavation activities or had flooded.
- The organisms once ingested passes through the wall of the GI tract, and, after gaining access to the bloodstream, are deposited in muscle and other tissues (spleen, liver, and GI tract) and may remain dormant or cause disease.
- In cattle, blackleg infection is endogenous. This means that lesions develop without

any history of wounds, although bruising or excessive exercise/trekking may precipitate disease in some cases.

## Affected group

- Animals that contract blackleg are mainly beef breeds that are in excellent body condition.
- Cases are sporadic and the most affected age groups are cattle 6–24 months old.
- However, there are report of calves in good body condition as young as 6 weeks and cattle as old as 10–12 years being affected.

## **Clinical signs**

- Sudden death with affected limb sticking out of the carcass.
- Sudden onset of acute, severe lameness, more commonly affecting the hind legs,
- There is a fever, but by the time clinical signs are obvious, body temperature may be normal or subnormal.
- Affected large muscle group are oedematous and swollen and when palpated make a crepitant sound. The most common muscles involved are hip, shoulder, chest, back and neck.
- At first, the swelling is hot, and painful. As the disease rapidly progresses, the swelling enlarges, there is crepitation on palpation, and the skin over the affected muscle group becomes cold and insensitive due to muscle necrosis as there is decreased blood supply to the affected muscle.

#### Post mortem lesions

- Red-brown water leaks from the swellings that has a rancid smell.
- The affected muscle when is cut open, looks like a black sponge.

#### Morbidity and Mortality Rates

- Based on WOAH reports, BQ has an estimated morbidity and mortality of local indigenous breeds of 17.9 % and 3.6 % respectively.
- In Marsabit county, livestock keepers in the year 2022 estimated the disease to have an apparent morbidity and mortality rate of 2% with 100% case fatality rate.

## Seasonality of occurrence

- BQ occurs in sporadic outbreaks affecting 1 or 2 animals in a herd. The disease can occur in any season as it is associated with events that disrupt the soil covering like excavations, drought and floods.
- In Marsabit county, livestock keepers in the year 2022, indicated that BQ occurs as a sporadic event throughout the year but most cases are observed during the long dry season that falls between the months of July and September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

#### Diagnosis

- Characteristic lesions of emphysematous swelling of the musculature.
- Standard culture techniques-anaerobic culture and biochemical identification of muscle tissue samples that are collected as soon as possible after death.
- Fluorescent antibody test for C chauvoei is rapid and reliable.
- PCR assay is available and has been reported to be reliable for clinical samples.

## Differential Diagnosis (DDx)

- Anthrax
- Trauma
- Lightning struck
- Bloat

#### Treatment

- The rapid fatal progression prevents treatment success in clinically sick animals.
- In an outbreak, all susceptible cattle should be vaccinated and treated prophylactically with administration of penicillin to prevent new cases.
- Cattle should be moved from affected pastures.
- Bury or burn the carcass to prevent the disease from spreading to other animals.

#### Prevention/control

- Calves over 2 months old should be vaccinated twice, 4 weeks apart, followed by annual boosters before the anticipated outbreak period.
- Blanthrax vaccine available at KEVEVAPI is used to control black quarter and anthrax diseases in Kenya.
- In South Africa, there is a popular management practice that has seen livestock keepers vaccinate beef cattle breeds every year until the animals are 3 years' old as they believe a lifelong immunity sets in adult animals.

## Zoonotic potential

• None, but due to toxaemia the carcass meat is susceptible to rapid decomposition and can be a source of food poisoning to humans if consumed.

## Notifiable disease

• No

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid grazing animals in past outbreak areas
- Migrate out of an area if there are reports of a BQ case
- Present animals for vaccination

#### Further Reading

- Birhanu Ayele. Worku Tigre, and Benti Deressa (2016). Epidemiology and financial loss estimation of blackleg on smallholder cattle herders in Kembata Tambaro zone, Southern Ethiopia. SpringerPlus 5:1822. https://www.ncbi.nlm.nih.gov/ pmc/articles/PMC5074928/#\_\_ffn\_sectitle
- 2. MSD Veterinary Manual https://www.msdvetmanual.com/



# Figure 32

Calf with black leg clinical symptoms of muscle swelling and lameness (white arrows) and Gram's staining of Clostridium chauvoei showing Gram positive, short, thick, straight, round ended rods (black arrows)

## Picture source:

https://www.banglajol.info/index.php/BJVM/article/view/28824

# 4.7 Haemorrhagic septicaemia (HS)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
SILISA	SILISA	KHANID /	NOLGOSO /	ELKUNOIT/	JAARO
		NALGOSO	NALGARNGARI	LOKUDO	

## Definition

- Haemorrhagic septicaemia is an acute disease of cattle and water buffalo characterised by sudden onset, fever, anorexia, depression, oedematous swelling of the throat, brisket and upper dewlap regions, dyspnoea, nasal discharge and salivation.
- WOAH defines HS as a highly fatal disease of cattle and buffaloes caused by specific serotypes of Pasteurella multocida designated B:2 (Asian serotype) and E:2 (African serotype). The WOAH Terrestrial code revised HS description in January 2021 revised HS serotypes to 6: B and 6: E. However, literature is still using B:2 and E:2 naming.
- The disease has an incubation period of 90 days and has active and latent carriers.
- Pasteurella multocida is a Gram negative bacteria.
- Animals can develop a Peracute form characterised by asymptomatic, pneumonia, endo toxaemia, severe dyspnoea and sudden death that has high mortality.
- Antimicrobial treatment very early in disease can reduce mortality.
- Definitive diagnosis requires the isolation of P multocida serotypes B:2 or E:2.

## Distribution (Epidemiology)

- HS is a major disease of cattle and buffaloes characterised by an acute, highly fatal septicaemia with high morbidity and mortality
- HS is endemic in Asia particularly India, Africa and Middle East. Sporadic outbreaks are reported in Southern Europe.

- HS outbreaks in India occur during the monsoon season due to high humidity and high temperatures. However outside Asia incidences of disease occur any month of the year.
- Natural HS occurs rarely in pigs, sheep, and goats and has been reported in camels, elephants, rhinoceros, horses, donkeys, yaks, and various species of deer and other wild ruminants.
- It is estimated that 5% of healthy cattle are colonized by small numbers of P multocida serotype B:2 or E:2 that are shed during periods of stress.
- Common stressors associated with outbreaks include high ambient temperature, humidity, concurrent infection with blood parasites or foot and mouth disease, poor nutrition, or work stress.
- Outbreaks can occur at any time but disease is most prevalent during the rainy season. Increased outbreaks associated with high rainfall are most likely due to the multiple stressors present during this time and the moist conditions, which prolong the survival time of the organism in the environment.
- Based on WOAH reports, the disease in endemic areas affects older calves and young adults with variable morbidity and mortality rates. In non-endemic areas, epizootics results in high morbidity and mortality that can reach 100%.
- In Marsabit county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 12%, mortality rate of 8% and case fatality rate of 62%.

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• Recovery stimulates acquired immunity to homologous and often heterologous strains of P multocida. Recovered animals become healthy carriers that can be a source of infection for future outbreaks.

## Causative agent/ Aetiology

- HS is caused by Pasteurella multocida serotypes B:2 and E:2. The letter denotes the capsular antigen and the number stands for the somatic antigen.
- P. multocida is, a Gram negative coccobacillus residing mostly as a commensal in the nasopharynx of animals. Pasteurella multocida has five types of capsular serotype namely; A, B, D, E and F.
- The bacteria is susceptible to mild heat (55°C) and most disinfectants.
- P. multocida does not remain viable for long periods in the environment.
- However, It can survive for hours or days in damp soil or water. Rainy conditions and high humidity facilitate transmission. Biting arthropods are not significant vectors.

## Transmission and Pathophysiology

- Infection occurs through direct contact with infected oral or nasal secretions from either healthy carrier animals or animals with clinical disease.
- Indirect contact occurs through ingestion of contaminated feed or water.
- Infection begins in the tonsil and adjacent nasopharyngeal tissues that subsequently, lead to a bacteraemia that leads to dissemination and rapid growth of bacteria in various body organs,
- The host cytokine response and release of killed bacteria lipopolysaccharides results in a rapidly progressing endo toxaemia.

## Affected age group

• All age groups and all breeds of cattle are susceptible. However older calves and juvenile adults are most affected. Buffaloes are three times more susceptible when compared to cattle.

#### **Clinical signs**

- Clinical signs appear 1 to 3 days after infection with death occurring within 8–24 hours after the first clinical signs develop.
- Peracute cases result in death within 8 to 24 hours after animals shows signs of fever, hypersalivation, nasal discharge, and laboured respiration. However, clinical signs are rarely observed before death in the peracute form.
- Acute disease lasts for 3 to 5 days and is characterized by fever of 40°-41.1°C, dyspnoea, reluctance to move, hypersalivation, lacrimation, nasal discharge that begins as serous and progresses to mucopurulent. Acute infections develop subcutaneous swelling in the pharyngeal region that extends to the ventral neck, brisket and sometimes the forelimbs. Terminally, the animals develop severe respiratory distress, cyanosis, terminal recumbency and sometimes abdominal pain with diarrhoea.

## Post-mortem lesions

- The carcass has widespread haemorrhages, oedema, and hyperaemia that is consistent with severe sepsis. Oedema fluid consists of coagulated sero-fibrinous mass with strawcoloured or blood stained fluid.
- In most cases, the carcass has swellings of the of the head, neck, and brisket.
- Petechial haemorrhages occur throughout the body.
- Thoracic and abdominal cavities contain blood-tinged fluid.
- The lungs tissue is congested and oedematous, sometimes with interstitial pneumonia.
- Gastroenteritis may also be seen.

## Season of occurrence

- HS cases are reported throughout the year but outbreaks are associated with humid and heavy rainfall season.
- In Marsabit county, livestock keepers in the year 2022 indicated that HS was endemic in the county with serious outbreaks reported common during the long rainy season that falls between the months of April and June.
| Season        | Borana | Gabra | Rendille | Samburu       | Turkana       | Daasanach |
|---------------|--------|-------|----------|---------------|---------------|-----------|
| Long Rains    | Gann   | Gann  | Guuh     | Lng'erng'erwa | Akiru Napolan | IR Gutoka |
| (April- June) |        |       |          |               | or Akiporo    |           |

## Diagnosis

## Field based diagnosis

- To date there is filed based Point of Care Rapid Diagnosis test kit developed.
- Clinical signs are used to make a field based presumptive diagnosis in endemic areas.
- There are no microscopic features that are specific for haemorrhagic septicaemia – all lesions are consistent with severe endotoxic shock and massive capillary damage.

## Laboratory diagnosis is based on culture and blood tests

- A definitive diagnosis of HS is based on isolation of P multocida serotype B:2 or E:2 (or other less common serotypes recognized by the WOAH as causing HS) from the blood and tissues of a patient with typical clinical signs. Various other P multocida serotypes can cause HS-like disease in cattle and water buffalo, which must be differentiated from classical HS.
- Pasteurella multocida is not always found in blood samples before the terminal stage of the disease, and is not consistently present in nasal secretions or body fluids of sick animals or recently dead animals.
- Samples include heparinised blood sample or swabs collected from the heart within a few hours of death. Nasal swabs or swabs of other visceral organs can also be taken.
- If a complete necropsy examination is not feasible, blood samples can be taken from the jugular vein by aspiration or incision.
- Blood samples should be placed in a standard transport medium and transported on ice packs
- In animals that have been dead for more than 12 hours, spleen and bone marrow samples provide excellent samples for the laboratory, as these are contaminated relatively late in the post-mortem process by other bacteria.
- In animals dead for a long time, a long bone (femur/humerus) should be taken from its

carcass

- In live animals, Nasal swabs and clotted and unclotted blood samples are the main samples collected.
- Definitive diagnosis of HS depends on the isolation of the causative organism, P. multocida, from the blood or bone marrow of a dead animal by cultural and biological methods, and the identification of the organism by biochemical, serological and molecular methods.
- Blood smears from affected animals can be stained with Gram, Leishman's or methylene blue stains. The organisms appear as Gramnegative, bipolar-staining short bacilli. However, no conclusive diagnosis can be made on direct microscopic examinations of blood smears alone.

## Differential Diagnosis (DDx)

- Shipping fever is often mistakenly confused for HS, but has a multifactorial aetiology that also involves Mannheimia haemolytica.
   Shipping fever does not cause septicaemia, and does not also cause multisystemic petechial haemorrhages.
- The peracute nature of the disease and the extensive oedema and haemorrhage make it difficult to differentiate HS from blackleg and anthrax.
- Other differential diagnosis includes acute salmonellosis, mycoplasmosis, lightning strike, snake bites, poisoning and pneumonic Pasteurellosis.

## Treatment

- Antimicrobials are effective against HS if administered very early in the disease. However, because HS progresses rapidly, treatment is often unsuccessful.
- During outbreaks, any patient with a fever should be treated with intravenous antimicrobials (10% Oxytetracycline and as soon as possible to quickly obtain systemic

bactericidal antimicrobial concentrations.

- Various antibiotics have been used to treat HS effectively and they include; potentiated sulphonamides, tetracyclines, penicillin, gentamicin, ceftiofur and enrofloxacin. However, plasmid- and chromosomalmediated multidrug resistance seems to be increasing for some strains of P multocida, and resistance to tetracyclines and penicillin has been reported for serotype B:2 (Asian serotype).
- Antimicrobial susceptibility testing (AST) is necessary for P. multocida.
- The table below highlights dosage of antibiotics commonly found in agrovet drug stores in Marsabit county.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline Short acting	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT given IV daily for 3 to 5 days
Penicillin G and Dihydrostreptomycin (Penstrep) The synergistic action makes the combination have a broader spectrum of activity	Penicillin G has bactericidal action against mainly Gram- positive bacteria and has narrow spectrum Dihydrostreptomycin has narrow spectrum of bactericidal action against Gram-negative bacteria	1 ml per 10 kg body weight every 48 hours three times or until fever subsides. Given IM Shake bottle well before use and do not administer more than 20 ml in cattle and 5 ml in calves per injection site
Trimethoprim and sulfamethoxazole (potentiated sulphonamide)	Antibiotic with bactericidal broad spectrum activity against Gram-positive and Gram-negative bacteria.	Twice daily 1 ml per 10 - 20 kg body weight for 3 - 5 days. Given IM
10% Gentamycin	Bactericidal against mainly Gram-negative bacteria. Has a narrow spectrum of activity	Given IM, Twice daily at a dosage rate of 1 ml per 20 - 40 kg body weight for 3 days.
Enrofloxacin 10%	Bactericidal against mainly Gram-negative bacteria. Has a narrow spectrum of activity	Given IM, Twice daily at a dosage rate of 1 ml per 20 - 40 kg body weight for 3-5 days.
Phenyl butazone	Anti-inflammatory Analgesic Anti-pyretic	Adult Cattle: 10 mL IV or IM Calf: 6-10 mL IV or IM
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC Calves: 5-10 mL IM or SC

\* mL- Milliliter, body weight-BWT, kg- kilograms, Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC) NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

#### Prevention/control

- Commercial vaccines include, killed vaccines such as bacterins that have alum-precipitated, aluminium hydroxide gel and oil-adjuvants.
- The recommended vaccination schedule is animals less than (>3) years old, an initial two doses is given 1–3 months apart, followed by booster vaccinations once or twice yearly. The oil-adjuvant vaccine provides protection for 9–12 months and is administered annually. It is most effective when administered 1 month before the outbreak monsoon or rainy season. Although it provides the strongest immunity, it is unpopular in the field because of its viscosity and difficulty of administration.
- Commercial vaccines are not readily available in Kenya. KEVEVAPI does not produce HS vaccine based on the website information accessed in May 2022.
- Animals that acquire natural infections with Pasteurella multocida serotypes 6: B and 6: E and survive develop a solid immunity.
- There is a need for a vaccine that has longer duration of immunity, easier to administer and offers cross-protection as some African countries, have both strains circulating.

#### Zoonotic potential

- · None reported to date
- However, other P. Multocida serotypes have been reported to cause human infections and precautions should be taken to avoid exposure

## Notifiable disease

- · Yes, WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals and ensure they do not share same grazing land and watering points.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- · Avoid areas where HS outbreaks are occurring.
- · Migrate out of area that has HS outbreaks.

#### **Further Reading**

- 1. MSD Veterinary Manual https://www.msdvetmanual.com/
- 2. WOAH (2021). Technical Disease Card https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
- Weerathunga M.W.D.C., Ubeyratne, J.K.H. and Nadheer M.A. (2017). An outbreak of haemorrhagic septicaemia in the Navithanveli veterinary range in Ampara district, Sri lanka. S.L.Vet.J. 64(2):7-12. http://doi.org/10.4038/slvj.v64i2.22



Figure 33 Clinical cases of HS affected buffaloes (a, depressed animal; b, congested mucous membrane; c, hypersalivation and d, oedema in submandibular, cervical region and brisket

Picture source:

Weerathunga M.W.D.C., Ubeyratne , J.K.H. and Nadheer M.A. (2017). An outbreak of haemorrhagic septicaemia in the Navithanveli veterinary range in Ampara district, Sri lanka. S.L.Vet.J. 64(2):7-12. http://doi.org/10.4038/slvj.v64i2.22

## 4.8 Contagions Bovine Pleuropneumonia (CBPP)

Other names- Lung plague

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Sombes	Sombes	Lkibei	Lkipei Lonkishu	Loukoi -Angatuk	Luoke Guo

## Definition

• Contagious bovine pleuropneumonia (CBPP) is a highly contagious disease of cattle and water buffaloes. Affected animals have difficulty in breathing due to damage to the lungs, the disease in calves occurs as a polyarthritis with painful swelling of all joints.

## Distribution (Epidemiology)

 CBPP is endemic in Africa, with minor outbreaks occurring in the Middle East. North America has been free of the disease since 1892, UK since 1898 and Australia since 1973. The last outbreak of CBPP in Europe was seen in Portugal in 1999.

- Little is known about the disease in Asia, China reports its last outbreak was in 1995 but this is not WOAH confirmed.
- CBPP is endemic in Marsabit county affecting both indigenous breeds (Bos indicus) and exotic dairy breeds (Bos taurus). Livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 6%, mortality rate of 3% and case fatality rate of 31%
- CBPP incidences in Marsabit occur throughout the year but more cases are reported during the long dry season when animals from different areas including outside the county share common grazing and watering points. The long dry season occurs in the months of July to September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

## Causative agent/ Aetiology

- Bacteria called Mycoplasma mycoides mycoides, small-colony type (MmmSC)
- The Mycoplasma bacteria lacks a cell wall it is pleomorphic and resistant to antibiotics of the betalactamine group, such as penicillin.
- Mycoplasma do not survive for long in the environment and transmission requires close contact.
- The causative agent belongs to the Mycoplasma mycoides cluster. The cluster consists of five mycoplasma species or subspecies from bovines and goats that share serological and genetic characteristics.
- A number of Mmm strain genomes have been

fully sequenced. Molecular typing showed that all European Mmm strains groups belong to a single lineage.

• Growth of mycoplasma is fastidious and requires special media rich in cholesterol (addition of horse serum).

## Mode of transmission and Pathophysiology Source of infection

- · Aerosol droplets from cough.
- Organism can also be spread through saliva, urine, foetal membranes, uterine discharges and semen.
- · Fomites are not a major source of transmission

as the organism does not survive outside the host.

• Chronically infected and asymptomatic animals play an important role in the persistence and spread of the disease. Pastoralists herds may contain chronically infected animals. Disease avoidance strategy of migrating from a focus of disease with apparently healthy animals has been known to spread the disease widely.

## Transmission and Pathophysiology

- Direct, close, repeated contacts between diseased and healthy animals in shared night bomas, water holes, dip tanks, markets, common grazing and gathering places for mass vaccination campaigns. Air-borne transmission can occur over distances of up to 200 metres.
- Transplacental infection of the unborn calf.
- Indirect transmission is rare and is not an important transmission route of CBPP as the organisms is killed rapidly in hot, dry environments.
- Outbreaks are common and extensive when groups of cattle from different herds are housed or have been transported by truck or trekked on foot.

## Affected group

• All age and breeds of cattle are susceptible. Clinical signs

- The incubation period is one to six months based on WOAH Terrestrial Code guidelines.
- Clinical diagnosis of CBPP is unreliable as initial signs may be slight or non-existent and may be indistinguishable from any severe pneumonia.
- The disease occurs in peracute, acute, subclinical and chronical forms.
- In peracute form few cattle die with no symptoms other than fever (41.5°C). This form occurs in only 10% of infected animals.
- The acute form occurs in 20% of infected animal. The first sign is fever (of up to 41.5°C); anorexia; and painful, difficult breathing. In hot periods of the day, the animal stands on its own in the shade with its head lowered

and extended, its back slightly arched, and its elbows turned out, elbows are said to be abducted. Percussion of the chest is painful; respiration is rapid, shallow, and abdominal. If the animal is forced to move quickly, the breathing becomes more distressed and a soft, moist cough results. The disease progresses rapidly and animals have a poor body condition, and breathing becomes more laboured, with a grunt at expiration. The animal then becomes recumbent and dies after 1 to 3 weeks. There may be nasal discharge, sometimes streaked with blood, and frothy saliva that accumulates around the mouth. Some animals develop swellings of the throat and dewlap. Pregnant cows and heifers may abort and diarrhoea has also been recorded.

- If animal survives the acute form they become chronically affected in which the lesions gradually resolve and the animals appears to recover.
- The subclinical form is the most common and occurs in 40 to 50% of the animals affected. The symptoms resemble those of the acute form but are less severe; fever is intermittent. Most of the animals suffering this form move to the chronic form.
- The chronic form is a natural evolution of both acute and sub-acute forms but in some animals it can develop directly. The clinical signs regress but cattle can still have intermittent fever together with loss of weight.
- Infected calves under 6 months present primarily with polyarthritis that is seen as swelling of joints and lameness. Infected calves present with poly-arthritis with swelling of carpal and tarsal joints.
- Lesions in the lungs take a long time to heal fully and inside them the causative agent can survive for up to two years. Up to 25% of affected cattle can become chronic carriers of infection. These chronic carriers are called 'lungers' and are believed to play a role in initiating new outbreaks when they are moved into susceptible herds.

## Post mortem lesions

## Lesions confined to chest cavity

- The thoracic cavity may contain 10 to 30 litres of yellow fluid that is mixed with fibrin flakes or large clots.
- The lungs are covered with thick deposits of yellow coloured fibrin and do not collapse as is the normal when the chest cavity is open. The lung tissue is firm and resembles the liver tissue.
- Lung lesions in 80 to 90% of cases are unilateral (affect only 1 lung) and in most cases only the diaphragmatic lobes are involved.
- Lung lesions adhere to the chest wall and on cut surface the cut surface of the lung tissue has a marbled appearance because of the widened interlobular septa.
- In chronic cases there may be areas of dead lung tissue that become surrounded by a capsule of fibrous connective tissue. This structure is called a sequestrum.

## Diagnosis

## Field based diagnosis

- A field based rapid latex agglutination test (LAT) that gives results in less than two minutes, using sera or whole blood, has been developed for screening purposes.
- Post mortem lesions are characteristic and slaughterhouse monitoring is a powerful tool to use in CBPP surveillance.
- Co-existence of pulmonary signs in adults and arthritis in young animals should alert the clinician to a tentative diagnosis of CBPP.
- Samples from live animals include nasal swabs and/or broncho-alveolar washings, or pleural fluid obtained by puncture. Collection of blood in anticoagulant and clot activator tubes.
- Samples taken at necropsy are lung lesions, lymph nodes, pleural fluid and synovial fluid from calves with arthritis.
- Samples should be shipped in a cool box but may be frozen if transport to the laboratory is delayed.

## Laboratory diagnosis

 Confirmation is by isolation of the mycoplasma followed by growth inhibition or immunofluorescence test using hyper immune rabbit sera against the mycoplasma, or use of molecular PCR techniques.

## Differential Diagnosis (DDx)

- Haemorrhagic septicaemia (HS).
- Bacterial or viral Broncho-pneumonia.
- East Coast Fever.
- Traumatic Reticulo-pericarditis (TRP)/ Foreign body pericarditis,
- On post mortem- Abscesses or Tuberculosis lesions.

## Treatment

- In disease free countries immediate slaughter of infected and in contact animals is practiced this is called stamping out.
- Treatment is recommended only in endemic areas because the organisms may not be eliminated, and carriers may develop.
- Drug label dosage should be followed. Three major classes of antibiotics are effective against mycoplasmas, namely tetracyclines, fluoroquinolones and macrolides. The most common antibiotic used is Tylosin at 10 mg per kilogram body weight twice a day for six consecutive days. This is a cumbersome regime for pastoralists settings but animal health providers need to encourage adherence through frequent extension messaging.
- In Marsabit county livestock keepers indicated they used the following antibiotics to treat CBPP, Penstrep® (Penicillin and Streptomycin), Tylosin and Oxytetracycline. Based on the nature of the pathogen the penicillin antibiotic is not effective and animal health providers need to discourage its use in managing CBPP.

## Prevention/control

- In endemic areas, during outbreaks cattle movement should be restricted through quarantine and ring vaccination.
- Where cattle cannot be confined as is the

case of pastoralist set ups, the spread of infection can be limited by immunization with attenuated vaccine (T1/44 strain). In Kenya, KEVEVAPI manufactures CBPP vaccine traded under the name CONTAVAXTM.

• The vaccine is effective only if herd coverage within a county is high (> 60%) of animals covered.

### Zoonotic potential

• No

## Notifiable disease

- Yes, WOAH listed disease
- Report to county and national DVS using mobile surveillance app (Kenya Animal Bio Surveillance System (KABS).

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Avoid areas where CBPP outbreaks are occurring
- · Present animals for vaccination
- Migrate out of an area when CBPP outbreak are occurring.

#### **Further Reading**

- 1. Food and Agriculture Organisation (FAO). Recognising CBPP: A field manual for recognition. http://www.fao.org/3/AC147E/AC147E00.htm
- 2. KEVEVAPI portal https://kevevapi.or.ke/shop/
- 3. WOAH (2020). Technical Disease Card https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/animal-diseases/technical-disease-cards/#searchform-header
- 4. MSD Veterinary Manual https://www.msdvetmanual.com/



Figure 34 Typical Elbow abducted stance of an animal affected by CBPP

Figure 35 Post-mortem finding of yellow turbid fluid in the thoracic cavity with thick yellow fibrin



Figure 36 Typical mottled appearance of cut surface of the lung tissue of an animal infected with CBPP Figure 37 Adherence of lung tissue to the thoracic wall due to CBPP infection

Picture source: https://vdocuments.mx/contagious-bovine-pleuropneumonia-cbpp-contagious-bovine-pleuropneumonia.html

## 4.9 Anaplasmosis

Other name: Gall sickness - due to gall bladder distention on post mortem

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dharabot	Dharabot	Ndis	Ndis	Lonyang	Bash Kinne

## Definition

- Anaplasmosis is a disease of ruminants caused by an obligate intraerythrocytic bacteria called Anaplasma.
- The Anaplasma bacteria infects red blood cells of cattle, sheep, goats, buffalo, and some wild ruminants.
- Anaplasmosis is the second most important tick borne disease in Kenya after East Coast Fever.

## Distribution (Epidemiology)

- Endemic in Marsabit county.
- Occurs in tropical and subtropical regions including South and Central America, USA, southern Europe, Africa, Asia, and Australia.

## Causative agent/ Aetiology

- Anaplasma bacteria is from the order Rickettsiales, family Anaplasmataceae, genus Anaplasma.
- Bovine Anaplasmosis is caused by Anaplasma marginale.
- A marginale has a strain that has an appendage, formerly known as A caudatum before genome analysis.
- Cattle are also infected with A centrale, which causes mild disease.
- The Anaplasma genus also includes species that were previously known as Ehrlichia like A. bovis (formerly E bovis), and A. platys (formerly E platys). These can invade erythrocytes of other mammalian hosts and therefore are potentially zoonotic.
- In small ruminants, anaplasmosis is caused by Anaplasma Ovis.

# Mode of transmission and Pathophysiology Source of infection

- In Kenya infections are due to the bite of the blue tick, Boophilus decoloratus and possibly also biting flies. Boophilus ticks are one-host ticks and only the engorged adult females drop off the host to lay eggs in the environment.
- Infections can also be due iatrogenic transmission through re-use of hypodermic needles or surgical instruments that have not been cleaned between animals for example during castration or dehorning procedures.

## Transmission lifecycle and Pathophysiology

- Close to 17 tick species can transmit
   A. marginale including Dermacentor,
   Rhipicephalus, Ixodes, Hyalomma, and Argas.
- Transplacental transmission has been reported and is usually associated with acute infection of the dam in the second or third trimester of gestation.
- There is a strong correlation between age of cattle and severity of disease. Calves are more resistant to disease (although not infection) than older cattle. This resistance is not due to colostrum antibody from immune dams but it is from the fact that calves born in endemic areas become infected with A. marginale early in life and gain carrier status without manifesting any signs. This situation is called enzootic stability.
- Studies have shown that cattle are capable of developing certain natural immunity to ticks and tick-borne diseases when exposed to them.
- Animals that are infected with the parasite remain lifelong carriers and serve as sources of future infections. Serious losses occur when mature cattle with no previous exposure are moved into endemic areas.

## Affected group

• All age groups and cattle breeds are susceptible to infection especially if they are not from endemic areas. However, calves and indigenous cattle breeds (Bos indicus) are less susceptible to severe clinical diseases.

#### Clinical signs

- Steadily increasing temperatures but does not exceed 41°C like in ECF.
- Anaemia, weakness and respiratory distress particularly after exercise.
- Jaundice and marked loss of body condition.
- Urine is often brown due to the presence of bilirubin.
- Constipation, faeces are dark and firm and sometimes covered with mucus.
- Severely affected animals may die.
- The urine brown colour in Anaplasmosis contrasts with the bloody red urine seen in Babesiosis infections. This is because the infected red blood cells in Anaplasmosis are being destroyed in the spleen while in Babesiosis, the red blood cells rupture/ haemolyse while in circulation hence the bloody urine.
- Anaplasmosis frequently occurs at the same time with ECF. The reason is that ECF causes a depression of the animal's immune responses. This then allows an Anaplasma infection which was already present in the carrier state in the animal to flare up as clinical Anaplasmosis.
- Animals with mixed ECF and Anaplasma infection are very sick with poor prognosis. Research at KALRO Muguga in Kenya indicates that even low Anaplasma parasitaemia (2%) can cause severe symptoms if there is co-infection with ECF.
- Animals with high Anaplasmosis parasitaemia of between 10% and 70% may show milder disease than those with co-infection.

## Post mortem lesions

Lesions are typical of those found in animals with anaemia due to erythrophagocytosis.

- Blood is thin and watery.
- Entire carcass is generally markedly anaemic and jaundiced.
- Liver is enlarged with a yellow-orange discolouration.
- Gall bladder is distended and contains thick brown or green bile.
- There are fluid (serous) effusions in the body cavities.
- Petechiae haemorrhages occur in the epi and endocardium.
- Spleen is enlarged with reddish-brown pulp and enlarged splenic follicles.

## Morbidity and Mortality Rates Estimates

- A recent molecular study in Kenya, estimated the prevalence of Anaplasma in dairy cows in Nairobi county and its environs to be for A. platys (44.8%), A. marginale (31%) and A. bovis (13.8%). Another study found the seroprevalence of Babesiosis, Anaplasmosis and ECF to be 0.42%, 4.64% and 4.92% and 1.45%, 32.11% and 39.05% in the rural and peri-urban areas of Western Kenya respectively.
- Molecular based Anaplasma ovis prevalence studies conducted in 2018 and 2019 found a 40.8% prevalence in sheep and goats from central and western Kenya, 19.5% prevalence in small ruminants from North-eastern Uganda, a 60.1% prevalence in sheep and goats from Sudan and in Tunisia the prevalence was higher in sheep at 93.8% when compared to goats at 65.3%.
- Marsabit county livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 3%, mortality rate of 2 % and case fatality rate of 60% in cattle. In small ruminants they estimated an apparent morbidity rate of 6%, mortality rate of 3% and case fatality rate of 50%.

#### Seasonality of occurrence

- The presence of active and infected tick vectors determines season of occurrence. reports indicate it can occur in all seasons.
- In Marsabit county, Anaplasmosis occurrence

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

in both cattle and small ruminants is common during the long dry season during the month of July to September.

## Diagnosis

## Field based diagnosis

- Microscopic examination is used as a rapid and confirmatory test.
- Samples to be collected include unclotted blood and lymphnodes smear if the nodes are enlarged so as to rule out mixed infections with ECF.
- The best blood smear should be taken from the tip of either the ear or the tail of the sick animal (peripheral blood smear).
- Thin blood smears stained in Giemsa show Anaplasma species appearing as dense, homogeneously staining blue-purple inclusions in the red blood cells that are located towards the margin of the infected erythrocyte.
- At necropsy, thin blood films of liver, kidney, spleen, lungs, and peripheral blood should be prepared for microscopic examination

## Differential Diagnosis (DDx)

- Babesiosis
- Leptospirosis
- Bacillary haemoglobinuria
- Eperythrozoonosis

## Treatment

- Always check if animal has a mixed infection with ECF before deciding on course of treatment. ECF treatment with Parvaquone or Buparvaquone causes Theileria piroplasms to become round and dark-staining thus resembling Anaplasma parasites.
- The carrier state may be eliminated by administration of a long-acting oxytetracycline preparation (20 mg/kg, IM, at least two injections with a 1-week interval). Injection into the neck muscle rather than the rump is preferred.
- Imidocarb dipropionate is also highly efficacious against A marginale as a single injection at 3 mg/kg. Elimination of the carrier state requires the use of higher repeated doses

Active ingredient	Indication	Dosage* and Route**
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT given IV daily for 3 to 5 days
1 mL per 10 kg BWT deep IM hind legs or neck muscles q48 hours PRD IM	Antiprotozoal	6.6 mg/kg BWT (2.5mL per 100kg BWT) Repeat dose in two (2) weeks, for a total of two (2) treatments.
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

\* Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeatq and mL- Milliliter, body weight-BWT, kg- kilograms\*\*By Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC) of imidocarb (5 mg/kg, IM or SC, two weeks apart). Imidocarb is a suspected carcinogen with long withholding periods and is not approved for use in the USA or Europe.

#### Prevention/control

- Vaccine is not available in Kenya.
- Vaccine is available in South Africa, Australia, Israel, and South America using a live vaccine derived from Anaplasma centrale (originating from South Africa). There is a killed vaccine in USA used as an experimental vaccine. It requires two doses (28 days apart) and booster doses every 1 to 2 years depending on herd history
- Attempts to use live or attenuated A. marginale have results in fatal disease.
- Sustained and stringent tick control using acaricides is the most common strategy, however it is not practical in pastoralist settings and a balance needs to be made to ensure young animals acquire endemic stability this become immune to infections when they are adults.

### Zoonotic potential

 No for A. Marginale. Yes, for Anaplasma species that are adapted to infect more than one mammalian host like A. bovis and A. platys.

## Notifiable disease

• No

Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Hand pick ticks from animal,

#### **Further Reading**

- Mbogo S.K., Kariuki D.P., McHardy N., Payne R., Ndungu S.G., Wesonga F.D., Olum M.O. and Maichomo M.W. (2016). Training manual for veterinary staff on immunisation against East Coast Fever. A Kenya Agricultural and Livestock Research Organisation (KARO) publication supported by GALVmed. Bill & Melinda Gates Foundation and UK aid. https://assets.publishing.service.gov.uk/ media/5aa8e9a040f0b66b625e2bfa/66\_Training\_Manual\_for\_Veterinary\_Staff\_on\_Immunisation\_against\_ECF.pdf
- 2. MSD Veterinary Manual https://www.msdvetmanual.com/
- Okuthe OS, Buyu GE. Prevalence and incidence of tick-borne diseases in smallholder farming systems in the western-Kenya highlands. Vet Parasitol. 2006 Nov 5:141(3-4):307-12. https://doi.org/10.1016/j.vetpar.2006.05.016
- Peter, S.G., Aboge, G.O., Kariuki, H.W. et al. Molecular prevalence of emerging Anaplasma and Ehrlichia pathogens in apparently healthy dairy cattle in peri-urban Nairobi, Kenya. BMC Vet Res 16, 364 (2020). https://doi.org/10.1186/s12917-020-02584-0



Figure 38 Jaundiced liver with distended gall bladder as seen in anaplasmosis in a sheep



Figure 39 Post mortem Anaplasmosis lesions: Enlarged and jaundices liver (right) and spleen (left) in a beef animal



Picture source: http://www.ndvsu.org/images/StudyMaterials/Parasitology/Anaplasmosis.pdf

## Cattle Diseases: Protozoal diseases

## 4.10 East Coast Fever (ECF)

## Other names: Corridor disease, Fortuna disease, January disease, and Theileriosis.

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
-	-	Lipis	Lipis	Lipis	-

## Definition:

- ECF is an acute disease of cattle, characterized by high fever, swelling of the superficial lymph nodes, dyspnoea and high mortality.
- It is estimated that annually, ECF causes the death of 100,000 cattle of all ages in Kenya. This makes ECF the most important disease of cattle in the country.

## Distribution (Epidemiology)

- ECF occurs in 13 countries in Sub-Saharan Africa.
- The disease is a new incursion in Marsabit county with few sporadic cases being reported in Laisamis sub county.

Causative agent/ Aetiology

- ECF is cause by a protozoan parasite called Theileria parva.
- Theileria parva is an obligate intracellular protozoan parasite that has many strains that vary in virulence. Most mild strains do not result in the animal developing the disease.

## Mode of transmission and Pathophysiology Source of infection

• The bite of an infected tick called Rhipicepalus appendiculatus, the brown ear tick. The saliva

of the tick carries the infective form of the parasite called Sporozoites.

- The brown ear tick is a three-host tick. This means that the three stages in its life cycle, the larva, nymph and adult attach to different hosts.
- Eggs of the brown ear tick are not infected with Theileria parasites, so the larvae which hatch from them cannot transmit the disease. Only nymphs and adults are infective.
- About 2-10% of ticks in an ECF endemic area are infected with Theileria parasites. This means that the presence of brown ear ticks on a susceptible animal does not necessarily mean that the animal will develop ECF. However, only 1 tick is needed to transmit the disease to a susceptible animal.
- Animals that recover are immune to subsequent challenge with the same strains but may be susceptible to heterologous strains. However, most recovered or immunized animals remain carriers.

## Affected group

- All age groups and all breeds of cattle are affected.
- · Calves rarely develop fatal disease like adults.
- Exotic breeds (Bos Taurus) are more

susceptible when compared to indigenous Bos indicus breeds.

## Clinical signs

- ECF incubation period is 8 to 12 days.
- Fever occurs and continues throughout the course of infection and may be as high as 41 to 42 °C.
- Marked enlargement of all palpable and superficial lymph nodes, particularly, the ones in front of the shoulder (pre-scapulars) and below the ears (parotids) and the on the flanks (pre-crurals).
- Lacrimation, increased nasal discharge and coughing also occurs.
- Terminally there is marked dyspnoea. In the advanced stages of ECF the lungs become full of fluid, breathing becomes difficult and finally froth fills the airways and the animal dies.
- Just before death, there is a sharp decrease in body temperature that is accompanied by recumbency (at this point treatment should not be attempted).
- After death pulmonary exudate pours out of the nostrils.
- Death occurs in 18–24 days after infection.

## Other important clinical signs

- Corneal opacity due to infiltration of the cornea by infected lymphoblast cells. This may resolve with treatment but sometimes recovered animals can have permanent loss of eyesight.
- Diarrhoea.
- Infected cells sometimes block capillaries of the central nervous system and result in neurological signs called the 'turning syndrome'.
- Petechiae and ecchymosis haemorrhages may be found on the conjunctiva, oral, lingual (underside of the tongue) and vaginal mucous membranes.
- Mixed infection with other tick-borne parasites due to destruction of lymphoid immune. Cells. Animals may also be infected with Babesiosis (red water) and Anaplasmosis (gall sickness). In endemic tick borne areas, mixed infection

with the three parasites is common. This makes correct diagnosis and treatment difficult and may be the reason for failure of some ECF treatments strategies.

• Other diseases, particularly bacterial infections, may also become more apparent in cattle suffering from ECF.

## Post mortem lesions

- Marked external and internal lymph node enlargement.
- Extensive pulmonary oedema and hyperaemia, the lung tissue does not collapse and remain distended with fluid after death.
- Haemorrhages are common on the serosa and mucosal surfaces of many organs, sometimes together with obvious areas of necrosis in the lymph nodes and thymus. Anaemia is not a major diagnostic sign (as it is in Babesiosis) because there is minimal division of the parasites in RBCs and thus no massive destruction of them.
- The mucosa of the digestive tract has lesions that resemble "cigarette burn" these are ulcers in the rumen. In addition, the colon, may show "zebra striping" due to destruction of the cells of the gut wall.
- Surfaces of the kidneys may show white spots (lymphoid infarcts), which extend down into the kidney tissue.

## Morbidity and Mortality Rates

- The fatality rate for untreated ECF is 100% in cattle from non-endemic areas especially exotic breeds (Bos Taurus).
- In endemic areas, the morbidity rate is high at 100% even amongst indigenous cattle breeds. However, the mortality for untreated ECF in indigenous breeds is lower and is estimated to range from 20% to 50%.
- Cross breed cattle show an intermediate degree of susceptibility.
- In Marsabit county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25%.

## Seasonality of occurrence

- Occurrence of disease is dependent on availability of an infected tick, susceptible animals and effectiveness of tick control strategies. Disease incidence can therefore occur all year round.
- In Marsabit county, ECF is common during the long rainy season

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June					or Akiporo	

### Diagnosis

- Presumptive diagnosis is based on clinical signs.
- Laboratory samples include lymph node smear, peripheral blood smear, unclotted blood in EDTA and clotted blood in Red-top tubes with clot activator.
- In resource constrained settings like the county laboratories, the main confirmatory tests done are Giemsa staining of lymph node and blood smears to identify Macroschizont (Koch's blue bodies, KBBs) in lymphoblast and piroplasms in red blood cells.

## Differential Diagnosis (DDx)

- Haemorrhagic septicaemia
- Babesiosis
- Malignant Catarrhal Fever (MCF)
- Trypanosomiasis
- Heart water
- Bovine leucosis

### Treatment

 Tetracycline though widely used by some livestock keepers in Marsabit County has limited value in treating clinical ECF cases.
 Tetracycline is mainly used during ECF vaccination to suppress development of overt disease.

Active ingredient	Indication	Dosage* and Route**
Parvaquone or Buparvaquone	Antiprotozoal drug	1mL per 20kg bodyweight. (2.5mg / kg BWT) IM into the neck muscles q48-72 hours in severe or advanced cases
Tripelennamine hydrochloride	Anti-inflammatory (anti-histamine)	0.5 – 1.1mg per kg BWT (2.5 – 5ml / 100kg)
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

\* Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q mL- Milliliter, body weight-BWT, kg- kilograms

\*\*By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

## Prevention/control

- Ticks can survive for up to 2 years on the ground without feeding on cattle. This means it can take more than two years to eliminate ECF infection. This fact makes tick control in pastoralists rangelands a daunting task.
- Chemical control of ticks with acaricides is still the most practical and widely used method for the control of ECF. However, acaricide resistance, high cost of acaricides and improper acaricide use hinder the effectiveness of acaricides.
- Tick control strategy aims at reducing the tick burden but not to eliminate the ticks as cat-tle acquire resistance to most tick borne disease if they are exposed to a low burden of tick infestation when young.
- ECF vaccination is done through the infection and treatment Method (ITM) in which cattle are given a subcutaneous dose of tick-derived Sporozoites and a simultaneous treatment with a long-acting tetracycline formulation. This treatment results in a mild or inapparent ECF reaction followed by recovery.

- Recovered animals demonstrate a robust immunity that lasts for the lifetime of the animal.
- The most widely used and studied ITM vaccine is the Muguga cocktail, developed in Kenya and comprising of 3 stocks (T. parva Muguga and T. parva Kiambu 5 and buffaloderived T. parva Serengeti transformed).

## Zoonotic potential

• No

### Notifiable disease

• No

#### Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices

• Refer to table 10

#### **Further Reading**

- Kipronoh K A, Gathuma J M, Kitala P M and Kiara H K (2011). Pastoralists' perception of the impact of East Coast fever on cattle production under extensive management in Northern Rift Valley. Kenya. Livestock Research for Rural Development 23 (6). http://www.lrrd.org/ lrrd23/6/cont2306.htm
- Mbogo S.K., Kariuki D.P., McHardy N., Payne R., Ndungu S.G., Wesonga F.D., Olum M.O. and Maichomo M.W. (2016). Training manual for veterinary staff on immunisation against East Coast Fever. A Kenya Agricultural and Livestock Research Organisation (KARO) publication supported by GALVmed, Bill & Melinda Gates Foundation and UK aid. https://assets.publishing.service.gov.uk/ media/5aa8e9a040f0b66b625e2bfa/66\_Training\_Manual\_for\_Veterinary\_Staff\_on\_Immunisation\_against\_ECF.pdf
- 3. MSD Veterinary Manual https://www.msdvetmanual.com/



Figure 41 Picture of a cow with heavy tick infestation in the ear

> Picture source: courtesy of Diba Denge, Animal Health Assistant in Saku, Marsabit County



Picture source: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf



- Black arrow showing enlarged parotid and prescapular lymphnodes of a cow with ECF
- Picture source: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf



Figure 44 Black arrow showing enlarged parotid and prescapular lymphnodes of a cow with ECF

Picture source:

https://assets.publishing.service.gov.uk/media/5aa8e9a040f0b66b625e2bfa/66-Training-Manual-for-Veterinary-Staff-on-Immunisation-against-ECF.pdf



Picture source: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf

## 4.11 Babesiosis

Other names: Red water, Piroplasmosis, Tick fever, Texas Fever

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Birte	Bira	Kati Ti Digee	Nadokula /	Lodip /Lokul	Sin Bura
			Noonkula		

## Definition

- An acute disease characterised by, fever, intravascular haemolysis leading to progressive anaemia, haemoglobinuria, and jaundice.
- Diagnosis is principally by light microscopic evaluation of blood smears. The most common treatments are imidocarb dipropionate and diminazene aceturate.
- Babesiosis is caused by an intraerythrocytic protozoan parasites of the genus Babesia.
- It is a tick borne infection and affects a wide range of domestic and wild animals and occasionally people.

## Aetiology and Epidemiology

- Babesiosis occurs where the arthropod vector is distributed, especially tropical and subtropical climates.
- The main Babesia species that cause disease in cattle are Babesia bovis and B. bigemina in Africa, Asia, Australia, and Central and South America. Babesia divergens occurs in some parts of Europe and northern Africa.
- In small ruminants, Babesia species that cause the disease include; B ovis, B motasi and B. crassa. The disease is more severe in sheep than in goats. Ovine babesiosis Infection are important in the Middle East, southern Europe, and some African and Asian countries.
- A 2018 meta-analysis study estimated the global bovine babesiosis prevalence to be 29% with B. bigemina having a prevalence rate of 22% and B. divergens prevalence of 12 %. An Ethiopian study in 2018 found a prevalence of 11.7% babesiosis and 6.1% anaplasmosis.

# Mode of transmission and Pathophysiology Source of infection in cattle

- Transmitted by tick vectors Rhipicephalus microplus (formerly Boophilus microplus) and Rhipicephalus annulatus (formerly Boophilus annulatus) where transmission occurs transovarially.
- Intrauterine infection has also been reported but it is rare.

Source of infection in small ruminants

• Babesia ovis and B. motasi. They are transmitted vertically by ticks of the genera Rhipicephalus, Dermatocenter, Ixodes and Haemophysalis.

## Transmission and Pathophysiology In endemic areas, two features are important in determining the risk of clinical disease:

- Calves, lambs and kids have a degree of immunity (related both to colostrum-derived antibodies and to age-specific factors that persists for 6 months to 2 years) animals that recover from Babesia infections are immune for their commercial life (4 years),
- Susceptibility of breeds to ticks and Babesia infections varies; Bos indicus cattle are more resistant to ticks and the effects of B bovis and B bigemina infection than Bos taurus breeds. In small ruminants' indigenous breeds like the black head Persian and Galla goats are less susceptible when compared to exotic breeds like Dorper and Toggenburg.

## **Clinical signs**

- Incubation period is between 2 to 3 weeks or longer after tick infestation.
- B bovis is a much more virulent organism than B bigemina in cattle while B. ovis is more virulent in small ruminants especially in sheep.
- B bovis, causes hypotensive shock syndrome, combined with generalized nonspecific inflammation, coagulation disturbances, and erythrocyte stasis in capillaries.

## Babesia bovis clinical signs

- High fever (41°C) that persists throughout the clinical course of the disease.
- There is muscle tremors, anaemia, jaundice, and weight loss.
- Haemoglobinemia and haemoglobinuria occur in the final terminal stages of the disease.
- CNS signs may be seen due to adhesion of parasitized erythrocytes in brain capillaries can occur with B bovis infections.
- Either constipation or diarrhoea may be present.
- Late-term pregnant cows may abort, and temporary infertility due to transient fever may be seen in bulls.
- Animals that recover from the acute disease remain infected for a number of years with B bovis and for a few months in the case of B bigemina. No clinical signs are apparent during this carrier state.
- Haemoglobinuria is the production of dark red or brown-coloured urine

## B. ovis infections

 acute form of the disease is characterized by fever, progressive anaemia and icterus. Haemoglobinuria is rare but may occur in the late stage of the disease. Pregnant animals may abort. Susceptible flocks may suffer a high rate of mortality. Recovered animals that are latently infected are usually immune for a certain period. There is no cross-immunity between the parasites.

## Post mortem lesions

- intravascular haemolytic condition -Pale or icteric mucous membranes; blood may appear thin and watery
- Subcutaneous tissues, abdominal fat and omentum appear icteric
- Swollen liver with an orange-brown or paler coloration; enlarged gall bladder containing thick, granular bile
- Enlarged, dark, friable spleen
- Kidneys appear darker than normal with possible petechial haemorrhages
- Bladder may contain dark red or brown-coloured urine
- · Possible oedema of lungs
- Petechiae or ecchymosis on surface of heart and brain

## Morbidity and Mortality Rates

- Morbidity and mortality vary greatly and are influenced by several factors, such as tick control, previous exposure to a species/strain of parasite, age and breed.
- In endemic areas, cattle become infected at a young age and develop a long-term immunity. However, outbreaks can occur in these endemic areas if exposure to ticks by young animals is interrupted or immuno-naïve cattle are introduced. The introduction of Babesia infected ticks into previously tick-free areas may also lead to outbreaks of disease.
- In Marsabit county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 5%, mortality rate 3% and case fatality rate of 64% in cattle while in sheep and goats they estimated an apparent morbidity rate of 4%, mortality rate of 1% and case fatality rate of 25%.

## Seasonality of occurrence

- Disease occurs throughout the year as it depends on tick vector presence and availability of susceptible host.
- In Marsabit county, the disease is endemic and occurs throughout the year. Most cases are however, reported during the long dry season in cattle and long rainy season in sheep and goats.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry (July- September)	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu Nakooyan	Mar Me Mederka

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

## Diagnosis

## Field based diagnosis

- Microscopic examination of Giemsa-stained blood or organ smears. From the live animal, thick and thin blood smears should be prepared, preferably from capillaries from the ear or tail tip. Smears of heart muscle, kidney, liver, lung, brain, and from a blood vessel in an extremity (lower leg) should be taken at necropsy.
- Babesia are found within RBCs, and all divisional stages – ring (annular) stages, pearshaped (pyriform) also known as trophozoites are found as single or in pairs.

## Confirmatory test

• Serologic tests such as indirect fluorescent antibody test and ELISA for detection of antibodies to Babesia especially in animals with low parasitaemia or carriers. PCR and real-time PCR assays are also done to detect extremely low parasitaemia, as occurs in carrier animals.

## Differential Diagnosis (DDx)

- Anaplasmosis
- Trypanosomiasis
- Theileriosis
- · Bacillary haemoglobinuria
- Leptospirosis
- Eperythrozoonosis
- · Rapeseed poisoning
- · Chronic copper poisoning
- · Rabies- Central nervous system signs

## Treatment

Early treatment before animals become severely anaemic gives a better prognosis. In both cattle

and small ruminants, the following drugs should be used. Always use lower range in very sick animals. Supportive treatment with multivitamin should also be done.

- Diminazene aceturate at 3.5 mg/kg, IM, once.
- Imidocarb dipropionate at 1.2 mg/kg, SC, once. At a dosage of 3 mg/kg, imidocarb provides protection from babesiosis for approximately 4 weeks and may also eliminate B bovis and B bigemina from carrier animals.

## Prevention/control

- Vaccination using live, attenuated strains of the parasites has been used successfully in Argentina, Australia, Brazil, Israel, South Africa, and Uruguay. The vaccine is provided in either as chilled or frozen form. One vaccination produces adequate immunity for the commercial life of the animal (4 years).
- Vaccine breakdowns have been reported.
- Controlling or complete eradication of the tick vector can break the transmission cycle, this approach is rarely feasible in the long term and can lead to large, susceptible populations in endemic areas with consequent risk of outbreaks of disease in naive animals.

## Zoonotic potential

- Yes, Human babesiosis has been reported with the rodent parasite B microti and the cattle parasite B divergens in North America and Europe, respectively.
- Human Babesia infections are acquired via bites from infected ticks or through contaminated blood from an infected transfusion donor. Disease can be fatal in splenectomised or immunocompromised individuals.

## Notifiable disease

 Yes, Bovine Babesiosis is listed by WOAH and should be reported to county and national director of veterinary services through the mobile app Kenya Animal Biosurveillance System(KABS).

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

#### Ethnoveterinary control practices

• Refer to table 10.

## Further Reading

Further reading

- Esmaeilnejad, B., Tavassoli, M., Asri-Rezaei, S., Dalir-Naghadeh, B., Mardani, K., Golabi, M., Arjmand, J., Kazemnia, A., & Jalilzadeh, G. (2015). Determination of Prevalence and Risk Factors of Infection with Babesia ovis in Small Ruminants from West Azerbaijan Province, Iran by Polymerase Chain Reaction. Journal of arthropod-borne diseases, 9(2), 246–252. https://www.ncbi.nlm.nih.gov/pmc/
- Keneth Iceland Kasozi, Susan Christina Welburn, Gaber El-Saber Batiha, Najat Marraiki, David Paul Nalumenya, Monica Namayanja, Kevin Matama, Kelly Katenta Zalwango, Wycliff Matovu, Gerald Zirintunda, Justine Ekou, Stellamaris Kembabazi, Claire Mack Mugasa, Annah Kitibwa, Dickson Stuart Tayebwa, Simon Peter Musinguzi, Michael Mahero, Ibrahim Ssengendo, Anne Nanteza, Enock Matovu, Ewan Thomas MacLeod.Molecular epidemiology of anaplasmosis in small ruminants along a human-livestock-wildlife interface in Uganda, Heliyon.Volume 7, Issue 1,2021.. https://doi.org/10.1016/j.heliyon.2020.e05688
- Mosqueda, J., Olvera-Ramirez, A., Aguilar-Tipacamu, G., & Canto, G. J. (2012). Current advances in detection and treatment of babesiosis. Current medicinal chemistry, 19(10), 1504–1518. https://doi.org/10.2174/092986712799828355
- 4. MSD Veterinary Manual https://www.msdvetmanual.com/



**Figure 46** Clinical signs of bovine babesiosis in naturally infected cow showing icteric vaginal mucous membrane (A) and dark red to brown urine (B)

Picture source: http://www.ndvsu.org/images/StudyMaterials/Parasitology/theileria.pdf



Blood smear showing Babesia Bovis (Right) and Babesia bigemina (Left)

Picture source:

Fernandez P. and White R.W. (2010). Atlas of Transboundary Animal Diseases. WOAH (World Organisation for Animal Health) ISBN 978-92-9044-804-4.



infected with Babesia motasi

Figure 48 shows various B. motasi intracellular stages. a-c Trophozoites in the periphery of infected RBCs (arrowheads). b-d Different

schizonts due to multiplication of Babesia motasi merozoites inside RBCs (arrows).

Picture source:

Hussein, Nermean & Mohammed, Eman & Hassan, Amal & El-Dakhly, Khaled. (2017). Distribution Pattern of Babesia and Theileria Species in Sheep in Qena Province, Upper Egypt. Archives of Parasitology



## Figure 49

Giemsa stained blood smear showing mixed infection of Anaplasma spp. and Babesia ovis

## Picture source:

Picture source: Pieragostini, Elisa & Ciani, Elena & Rubino, Giuseppe & Petazzi, Ferruccio. (2011). Tolerance to Tick-Borne Diseases in Sheep: Highlights of a Twenty-Year Experience in a Mediterranean Environment. 10.13140/2.1.2385.6968.



Urine before treatment (i) and progressive improvement (ii to iv) to normal (v) after prompt treatment.

Figure 50 Serial collection of urine from a dairy cow affected by Babesiosis

Picture source: https://www.dairyknowledge.in/article/ babesiosis

## 4.12 Trypanosomiasis

Other Names: Surra, Nagana, Tsetse Fly Disease, African Animal Trypanosomosis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Awarsa / Gandhi	Awarsa/ Gandhi	Omar	Saar	Ekwaakoit	Lesikonye
<b>Definition</b> <ul> <li>Protozoan disease of animals and humans</li> </ul>			which are for tissues and	ound in blood plass fluids. The disease	ma, various body causes serious

Animals Mainly Affected Major Geographic Distribution Trypanosoma spp South and eastern Africa T congolense Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals T vivax Africa, Central and South Cattle, sheep, goats, camels, horses, various wild animals America, West Indiesa T brucei brucei All domestic and various wild South and eastern Africa animals; most severe in dogs, horses, cats T brucei rhodesiense Cattle, wild hooved stock, people South and eastern Africa

## Causative agent/ Aetiology

• The major veterinary species are T congolense, T vivax, T brucei brucei, and T simiae. T brucei rhodesiense and T brucei gambiense are zoonotic, with people as the predominant host. Cattle, sheep, and goats are infected, in order of importance, by T congolense, T vivax, and T brucei brucei. T vivax is found outside tsetse-infested areas of sub-Saharan Africa, carried mechanically by biting flies. There are three variants of T.congolense, called the savannah, forest and kilifi (or Kenya Coast) types.

caused by parasites of the genus Trypanosoma,

• Trypanosomes do not survive for long periods outside the host.

## Mode of transmission and Pathophysiology Source of infection

• Tsetse flies (Glossina spp.) are biological vectors for the trypanosomes that cause African animal trypanosomiasis and transmit these organisms in their saliva. Trypanosomes must develop for one to a few weeks in the fly before they reach the infective stage. T. vivax

has the shortest cycle.

economic losses in livestock.

- Mechanical transmission is significant for T. vivax, which is transmitted primarily by this route through biting flies like Stomoxys, Tabanus and Chrysops species.
- Animals infected with trypanosomes can become chronic carriers, and inapparent infections can be reactivated if the animal is stressed.
- Transplacental and venereal transmission has been reported.

## Transmission and Pathophysiology

- Infected tsetse inoculates metacyclic trypanosomes into the skin of animals, where 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 fission.
- 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

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tissue damage in several organs.

• The immune response is vigorous, and immune complexes cause inflammation, which contributes to fever and other signs and lesions of the disease. Antibodies against the surfacecoat glycoproteins kill the trypanosomes. However, trypanosomes have a large family of genes that code for variable surface-coat glycoproteins that are switched in response to the antibody response, thus enabling them to evade animal's immunity. This antigenic variation results in persistence of the organism. Antigenic variation has prevented development of a protective vaccine and permits reinfections when animals are exposed to a new antigenic type.

## Affected group and season of occurrence

- All breeds affected but indigenous breeds less susceptible and seem to resist clinical disease if they are in good nutritional status.
- All ages but studies have shown that cattle less than 2 years are not affected by the disease. This may be because older cattle graze in tsetse fly infested areas whereas younger animals graze close to homesteads, hence lowering their exposure to tsetse bites.
- In Marsabit county, livestock keepers in the year 2022 indicated that the disease occurs mostly during the short dry season in the months of January to March.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Short dry season	Bon Agaya	Bon Agaya	Nabhai Gaban	Lamei-dorop	Akamu	Mar
(January- March)	Bon				Nauriyen	Megaauka
	Hagaya					

## Morbidity and Mortality rates

- Study estimate a morbidity rate of between 30 and 50% and mortality rate of 6 to 30%.
- In Marsabit county livestock keepers in the year 2022, estimated an apparent annual morbidity rate of 1% with no mortality rate as they are aware of clinical signs and they treat the animals early.

## **Clinical signs**

• The incubation period is usually 1 to 4 weeks. The first sign is a localized swelling (chancre) at the site of the fly bite, but this usually remains unnoticed.

## Acute form

• An acute haemorrhagic syndrome has been seen sporadically in cattle infected with some isolates of T. vivax, mostly in East Africa. This condition might be mediated by antibodies directed against red blood cells. Affected animals have enlarged lymph nodes and signs of severe anaemia, and they develop widespread visceral and mucosal haemorrhages, particularly in the gastrointestinal tract. In one outbreak, the main haemorrhagic sign was bleeding from the ears. This form can be rapidly fatal.

## Chronic form

- The primary clinical signs are an intermittent fever, signs of anaemia (e.g., pale mucous membranes, lethargy), lymphadenopathy and weight loss.
- Progressive emaciation leads to concurrent signs such as decreases in milk yield, decreased appetite, and may develop neurological signs, dependent oedema (including
- submandibular oedema), cardiac lesions, diarrhoea and keratitis/ corneal opacity.
- There may also be abortions, premature births, perinatal losses and damage to the male reproductive organs (e.g., orchitis, epididymitis), with reduced semen quality.
- Trypanosomes can cause immunosuppression, and concurrent infections may complicate this disease. Sudden deaths have been reported
- Animals that recover clinically may relapse when stressed.

## Post mortem lesions

• Necropsy findings vary and are nonspecific. In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen. In chronic cases, swollen lymph nodes, serous atrophy of fat, and anaemia are seen.

## Filed based diagnosis

- A presumptive diagnosis is based on finding an anaemic animal in poor condition in an endemic area.
- Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. The most sensitive rapid method is to examine a wet mount of the buffy coat area of a PCV tube after centrifugation, looking for motile parasites. This is called the Buffy coat concentration technique
- Rapid agglutination tests to detect circulating trypanosome species-specific antigens in peripheral blood are available for both individual and herd diagnosis, although their reliability remains varied.

## Differential Diagnosis (DDx)

• Infections that cause anaemia and weight loss, such as babesiosis, anaplasmosis, theileriosis, should be excluded by examining a stained blood smear.

## Treatment

- Most have a narrow therapeutic index, which makes administration of the correct dose essential. Drug resistance occurs and should be considered in refractory cases
- Drugs are normally sold in powder form or compressed tablets, and are stable over long periods when kept dry. Some become unstable when dissolved and must be used within a short period. The manufacturer's instructions should always be followed and it is a good practice to use solutions only on the day they are made. Some are a sterile solution diluent while some ask you to dilute in hot/warm or cold clean boiled water. (allow hot water preparations to cool before administering)

Drug	Effective against Trypanosoma	Main Action	Dosage and route
Diminazene aceturate (7%)	vivax, congolense, brucei	Curative	Deep IM or SC in the neck muscles 5-10 ml/100 kg BWT
Homidium chloride or bromide (2.5%)	vivax, congolense, brucei	Curative and some Prophylactic	Deep IM of neck muscles only 1 mg/kg BWT (1 ml/25 kg)
Isometamidium chloride (1% or 2%)	vivax, congolense	Curative and Prophylactic For preventive repeat every 10 to 12 weeks	Deep IM of neck muscles only 50 kg- 1.25 to 2.5 ML 100kg-2.5 – 5 ML 150kg-3.75-7.5 ML 200kg-5-10ML

Body weight-BWT, kg- kilograms; Intramuscular (IM), and Subcutaneous (SC)

## Sourced:

https://www.msdvetmanual.com/circulatory-system/blood-parasites/trypanosomiasis#v3258185

## Prevention/control

- Control is ideally achieved by combining methods to reduce the tsetse/biting fly challenge and by enhancing host resistance with prophylactic drugs.
- · Control can be exercised at several levels, including eradication of tsetse flies and use of prophylactic drugs. Tsetse flies can be partially controlled by frequent spraying and dipping of animals, aerial and ground spraying of insecticides on fly-breeding areas, use of insecticide-impregnated screens and targets, bush clearing, and other habitat removal methods. The Sterile Insect Technique (SIT) has been used with success in Zanzibar and may be used in other area-wide control operations after suppression of tsetse populations by insecticides. There is renewed international interest in large-scale tsetse eradication through the Pan African Tsetse and Trypanosomiasis Eradication Campaign (PATTEC) supported by the African Union. Animals can be given drugs prophylactically in areas with a high population of trypanosomeinfected tsetse. Drug resistance must be

carefully monitored by frequent blood examinations for trypanosomes in treated animals.

• Several breeds of cattle and water buffalo have been identified that have innate resistance to trypanosomiasis and could play a valuable role in reducing the impact of the disease in these areas. However, resistance may be lost because of poor nutrition or heavy tsetse challenge.

## Zoonotic potential

- No
- Humans are normally not susceptible to the trypanosomes that cause African animal Trypanosomiasis.

## Notifiable disease- No

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Avoid areas with Tsetse flies.

#### Further Reading

- Garoma DesaSamson LetaSamson LetaShibiru Debeda Sisay GetachewSisay Getachew (2016). Prevalence of Bovine Trypanosomosis in Gari Settlement Area of Eastern Wollega Zone, Ethiopia. Global Veterinaria 17(2):169-174. https://www.researchgate.net/ publication/306066825\_Prevalence\_of\_Bovine\_Trypanosomosis\_in\_Gari\_Settlement\_Area\_of\_Eastern\_Wollega\_Zone\_Ethiopia/references
- 2. MSD Veterinary Manual https://www.msdvetmanual.com/
- Simwango, M., Ngonyoka, A., Nnko, H. et al. Molecular prevalence of trypanosome infections in cattle and tsetse flies in the Maasai Steppe, northern Tanzania. Parasites Vectors 10, 507 (2017). https://doi.org/10.1186/s13071-017-2411-2



Figure 51 Trypanosome evansi in cattle blood

Picture source: https://www.hindawi.com/journals/ bmri/2013/194176/



Figure 52 Trypanosome vivax in bovine blood

Picture source: https://www.msdvetmanual.com/ circulatory-system/blood-parasites/ trypanosomiasis#v3258185

## Cattle Diseases: Parasitic Diseases

## 4.13 Fascioliasis

## Other name: Common liver fluke

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Minni Tiru	Minni Tiru	Deyah Ti Helesi	Lkurui	Lomany	Nakarkarsha
			Lemonywa		

## Definition

• Common parasitic liver disease of both cattle and sheep caused by Fasciola hepatica

## Distribution (Epidemiology)

Global distribution

## Aetiology

- Fasciola hepatica is a leaf shaped parasite from the helminth class Trematode.
- The parasite has as a broad host range, including people.

## Pathophysiology

- Eggs are passed in the faeces where miracidia develop. Hatching only occurs in water, and miracidia are short-lived (approximately 3 hours). Miracidia infect pond snail (Galba truncatula, previously known as Lymnaea truncatula).
- Once in the snail miracidia undergoes asexual development and multiplication through the stages of sporocysts, rediae, daughter rediae, and cercariae. After 6–7 weeks or longer if temperatures are low), cercariae emerge from snails,

- The cercariae encyst on aquatic vegetation, and become metacercariae. Metacercariae remain viable for many months unless they become desiccated.
- After ingestion by the host, usually with herbage, the metacercariae excyst in the duodenum, penetrate the intestinal wall, and enter the peritoneal cavity, where they migrate to the liver. The time required for this transit can vary and affects the efficacy of some treatments because many are only effective against adult flukes.
- The young flukes penetrate the liver capsule and tunnel through the parenchyma, for 6–8 weeks they grow and destroy tissue. They then enter small bile ducts and migrate to the larger ducts and, occasionally, the gallbladder, where they mature and begin to produce eggs.
- The prepatent period is usually 2–3 months. The minimal period for the completion of one entire life cycle is 17 weeks. Adult flukes may live in the bile ducts of sheep for years but in cattle they are eliminated from the liver after 5 to 6 months.

## **Clinical signs**

- Fascioliasis symptoms in cattle is determined by the number of metacercariae ingested. Acute disease occurs 2 to 6 weeks after the ingestion of large numbers of metacercariae (usually >2,000) in a short period. Acute disease manifests as distended, painful abdomen; anaemia; and sudden death.
- In subacute disease, cattle ingest moderate numbers (500 to 1,500) metacercariae over a long period of time. The cattle can survive for 7 to 10 weeks even in cases of significant hepatic damage, but deaths occur due to haemorrhage and anaemia.
- Chronic fascioliasis occurs as a result of ingestion of low numbers (200 to 500) metacercariae over a long period of time. Signs include progressive emaciation, anaemia, unthriftiness, submandibular oedema (bottle jaw), and reduced milk production.
- It should be noted that heavily infected cattle may show no clinical signs other than progressive emaciation despite access to good pasture. The immunity to other pathogens may also be reduced.
- In cattle, a partial acquired resistance develops after 5 to 6 months following infection.

## Post mortem lesions

- Severity depends on the number of metacercariae ingested, the phase of development in the liver, and the species of host involved.
- During the first phase, immature, wandering flukes destroy liver tissue and cause haemorrhage. The main lesions seen are enlarged liver that is friable with fibrinous deposits on the capsule. Migratory tracts can be seen, and the liver surface has an uneven appearance.
- In the second phase when flukes enter the bile ducts, ingest blood and damage the mucosa with their cuticular spines. The lesions are consistent with chronic liver damage such as cirrhosis where the damaged bile ducts become enlarged, or even cystic, and have thickened, fibrosed walls. In cattle but not sheep, the duct walls become greatly thickened

and often calcified. Aberrant migrations occur more commonly in cattle, and encapsulated flukes may be found in the lungs. Mixed infections with Fasciola gigantica can also be seen in cattle.

• Tissue destruction by wandering flukes may create a microenvironment favourable for activation of Clostridial spores and animal can succumb to Clostridial infection.

## Morbidity and Mortality Rates

- Morbidity rates are between 40 and 60% in most areas where animals have access to contaminated pasture
- Mortality rates in cattle is generally low between 0 and 13%. Most cases of infection are diagnosed at slaughter for most animals. However, in weaned or yearling calves it may be high at 30% due to ingestion of large numbers of mertcercariae cysts over a short period of time.
- In Marsabit county, livestock keepers in the year 2022, estimated an apparent annual morbidity rate of 12%, mortality rate of 1 % and case fatality rate of 8%.

## Age group affected and Seasonality of occurrence

- Affects all ages of cattle that have access to swampy pasture with intermediate snail host. However, beef breeds have higher incidence.
- Can occur throughout the year if animals are grazed in swampy areas.
- In Marsabit county, occurrence is throughout the year with no distinct season of occurrence.

## Diagnosis

## Field based Diagnosis

- Composite faecal egg counts where dung samples are collected from 10 animals and a single (composite) sample is submitted to the laboratory for faecal egg count.
- Faecal microscopic identification of the oval, operculated, golden brown eggs that must be distinguished from those of paramphistomes (rumen flukes), which are larger and clear. Eggs of F hepatica cannot be demonstrated in

faeces during acute fascioliasis. In subacute or chronic disease in cattle, the number varies from day to day, and repeated faecal microscopic analysis may be required.

## Laboratory diagnosis

- Confirmatory Diagnosis can be aided through ELISA analysis (commercially available in Europe) that enables early detection (2 to 3 weeks) of antibodies in serum or milk after infection.
- Blood serum analysis of plasma concentrations of y-glutamyltransferase, which is increased with bile duct damage, is also helpful during

the late maturation period when flukes are in the bile ducts.

• At necropsy, the nature of the liver damage is diagnostic. Adult flukes are readily seen in the bile ducts, and immature stages may be squeezed from the liver cut surface.

## Differential Diagnosis (DDx)

- Helminthiasis
- Paratuberculosis
- Pyelonephritis,
- Chronic mastitis,
- Chronic suppurative pneumonia.

Active ingredient	Indication	Dosage* and Route**
10% Albendazole	Anthelmintic and Flukicide Effective for mature (more than 90 days old) flukes only.	10 mg/kg BWT that is (1 mL per 10 kg BWT PO) Repeat after 2 to 3 weeks to target the immature flukes that have become adults
10% Triclabendazole	Anthelmintic and Flukicide Effective against all stages all stages of Fasciola hepatica from 2 day old immature to adult flukes	3 mL/25 kg BWT PO (2 mg /kg BWT) One dose
Levamisole HCl 30 mg Oxyclozanide 60 mg	Anthelmintic and Flukicide Oxyclozanide only active against the adult liver fluke	10 mL per 40 kg BWT PO Repeat after 2 to 3 weeks to target the immature flukes that have become adults
34% Nitroxyl	Anthelmintic and Flukicide Effective against immature and mature liver flukes	1.5 mL per 50 kg BWT SC only (10mg/ kg)
Multivitamin	Boost immunity	Adult Cattle: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

\* Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q and mL- Milliliter, body weight-BWT, kg- kilograms

\*\*By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SQ or SC)

NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

## Prevention/control

- Animals should always drink from safe, uncontaminated water sources. If this is not possible, measures should be taken to control the snail with copper sulphate, but this is short-term and not very effective.
- Examination of the livers at slaughter houses will give an indication of the degree of

infestation and thus used to advise livestock keepers when to deworm animals.

## Zoonotic potential

- Yes, important parasitic zoonosis
- WHO estimated in 19195 that approximately half of the 2.39 million infected individuals globally were living in three Latin American

countries (Bolivia, Ecuador and Peru). The Andean highlands represent the most endemic areas of the world, with high levels of prevalence of infection among indigenous communities.

1. MSD Veterinary Manual https://www.msdvetmanual.com/

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

· Avoid grazing animals in swampy areas

## Notifiable disease - No

**Further Reading** 

#### Migration of Adult flukes reside juveniles takes Host ingests cysts, out 6-8 weeks to in the bile ducts, juvenile flukes excyst reach the bile and lay their eggs and migrate through Fluke eggs are ets. PPP is 10-12 the liver of the host weeks from passed in ingestion of the faeces cysts until eggs are ieen in the faeces Eggs cars hatch in 9-10 days under ideal conditions. (22-25°C) Cercariae leave the snail when the temperature and moisture levels are Miracidium hatch then suitable, and encyst have 3 hours to find a as metacercariae on snail intermediate host pasture Mud snail - Galba Minecidium to metacercartae takes about 6 -7 weeks truncutala.

## Figure 53

Lifecycle of the Liver Fluke in cattle

Picture source: https://www.farmhealthonline.com/US/disease-management/cattle-diseases/liver-fluke/



Liver flukes lesions seen as thickening and calcification of bile ducts of cattle liver (Pipe Stem Liver)



Figure 55 Fluke eggs under the microscope (Left hatched) and right a fluke egg and miracidia

Fluke eggs are thin-shelled, oval, operculated and have a brown-colour. Picture source:

https://projectblue.blob.core.windows.net/media/Default/Beef%20&%20Lamb/CHAWG/From%20 2013/Liver-fluke-in-cattle-costs-and-control.pdf



Adult Fasciola hepatica (1) and Fasciola gigantica (2) left picture and adult paramphistomes on the surface of rumen

Picture source: https://www.sciencedirect.com/topics/agricultural-and-biological-sciences/liver-fluke

## 4.14 Mange in Cattle

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Chito	Chito	Haddo	Lpepedo	Emitina Pepedo / Ekokoi	Gogosich

## Epidemiology

- Cattle are affected by 5 types of mite species.
- Clinical signs depend on the type of mite biology and ecology, for example, whether the mite burrows into the skin or whether it inhabits the hair follicles or sebaceous glands.
- Sarcoptic mange, or scabies is caused by Sarcoptes scabiei var. bovis, a skin-burrowing mite. The mite is highly contagious and zoonotic. It causes intense pruritus and papules. If left untreated the skin thickens, forming large folds, and the entire outer body surface can be affected in a few weeks.
- Psoroptes ovis is a non-burrowing mite causing psoroptic mange. The mite pierces the skin to imbibe the fluids emanating from the wound. Infection is characterised by the formation of thick crust, exudative dermatitis, alopecia, and intense pruritus.
- Chorioptes bovis, the chorioptic mange mite, typically inhabits the skin surface of the tail and lower legs and therefore the condition may be called tail, foot, or leg mange. Although C bovis can survive off the host for up to 3 weeks, chorioptic mange is relatively less pathogenic to cattle than sarcoptic or psoroptic mange.
- Demodex bovis is the most common of 3 Demodex species that can infest the hair follicles and sebaceous glands of cattle where they feed on sebum, oozing plasma, and epidermal debris. Skin lesions are susceptible to secondary bacterial infection. Young cattle appear to be more susceptible to heavy infestations, which can also result in hide damage.
- The cattle itch mite, Psorobia bos, can cause psorergatic mange of limited pathogenicity.

Cases involve slight skin thickening and some scaling where low-grade pruritus can occur.

• Cattle are also susceptible to infestation by the ear mites Raillietia auris and Raillietia flechtmanni. In addition to the subclinical otitis, ear mite infestation also result in accumulation of wax.

## Diagnosis

## Field based diagnosis

- Skin scraping as described in chapter one
- Clinical signs can be used to make a field based presumptive diagnosis.

## Treatment and control

- Treatment of mange is achieved efficiently with macrocyclic lactones (Ivermectin at a dose rate of 1 mL per 50 kg bodyweight repeated 2 weeks apart) and use of acaricides like Amitraz or synthetic pyrethroids for topical application for chorioptic and sarcoptic mange.
- Control is achieved by isolating infested animals and treating them immediately the skin lesions are seen.

## Zoonotic potential

• Yes, for Sarcoptic mange

#### Notifiable disease

- Yes, for Sarcoptic and Psoroptic mange as they are WOAH listed disease
- Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.


Figure 57 Bovine sarcoptic mange (scabies)

Picture source: Dr. Raffaele Roncalli in MSD veterinary manual Figure 58 Demodex bovis, skin lesions



Figure 59 Skin lesion location for mites in cattle

## Ethnoveterinary treatment practices

• Refer to tables 12 and 13

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

Isolate sick

#### Further Reading

- 1. MSD Veterinary Manual https://www.msdvetmanual.com/
- 2. WOAH (2020). Technical Disease Card https://www.WOAH.int/ en/what-we-do/animal-health-and-welfare/animal-diseases/ technical-disease-cards/#searchform-header

# Chapter 5: Camel Diseases

Camel Protozoal Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Trypanosomiasis*	Dukan	Gandhi	Omar	Saar	Ekwakoit / Lokipi	Lesikonye		
Camel Viral Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Contagious Ecthyma (ORF)	Umburur	Abthara	Afturo	Afturo	Ngiboruok/ Lokutuk	Dicho		
Camel Pox	Finno / Bagaa	Finno/ Bagaa/ Abdara Raba	Nyinyilo	Nariri Ee Ntamesi	Ekolemeri/ Etune	Vert - Galte		
Camel Cough/Flu	Qufaa	Qufaa	Yahar	Lchama	Lowala	Furs-Galte		
Camel Bacterial Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Haemorrhagic Septicaemia (HS)	Qandich	Qandich/ Qando	Khanid	Nalngaringari	Elkunoit / Longauroi	Jaaro		
Caseous Lymphadenitis (CLA)	Qarfat	Qarfat	Orokajire	Lngarngar Nkimek	Abuus Ngabulon	Dhuorich		
Contagious Skin Necrosis (CSN)	Dulla	Qarfat	Kharfat	Lmonkoi	Akitherit Eparaa	Alkine		
Camel Parasitic Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Mange	Chito	Chito	Haddo	Lpepedo	Emitina Pepedo / Ekokoi	Gogosich		
Ring Worm	Robbi	Robbi	Robo /	Ngamunyani	Akiserit	Finy		
			Nabhar					
Camel Non- Specific Disease	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Wry Neck	Ch'ach'apsa	Ch'ach'apsa	Dhahasi	Lajingani	Lokonyo	Luut Gaa Nyurua		
* Nervous form of Try	* Nervous form of Trypanosomiasis in camels is called - Shimpir or Shimpiro in Gabra and Borana community.							

Botanical name for medicinal plant	Target camel disease
Acacia Nubica	Trypanosomiasis, Camel Flu, Pneumoniae, HS, CSN
Acacia tortilis	Trypanosomiasis, Camel Flu, Pneumoniae, HS,CSN
Azadirachta indica – Neem tree	Trypanosomiasis, Camel Flu, Pneumoniae, HS, CSN
Zanthoxylum usambarense (Prickly ash)	HS, Trypanosomiasis, Camel flu
Ficus sur (Bush fig)	CSN
Salvadora passica- tooth brush tree	Improves camel immunity to diseases Immune booster
Commiphora species	CLA, CSN
NB: Refer to table 8 above for details on most	commonly used plant parts in the preparation of remedies

## Table 14: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority cattle diseases in Marsabit county

Table 15: Traditional pr	ractices used to treat	priority camel diseases	in Marsabit county
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Traditional practice	Target camel diseases
Salty water from saline wells, springs or Lake Turkana- Topical application	Orf, Camel pox, CSN
Bleeding animal from jugular vein	TrypanosomiasisBrucellosis
Chewing tobacco crushed into powder and dissolved in water and given as an oral drench or it is made into a paste and applied topically	HS, Camel flu
Hot iron or hot stone cauterisation	Orf, Wry neck, HS, Camel flu, Brucellosis, Camel pox, Pneumonia, CSN
Melted fat from a sheep's tail	Orf, Wry neck, Mange, Trypanosomiasis, Ring worm
Sheep head soup	HS
Fumigation/Smudge SmokingBurn leaves of medicinal plants or livestock dung	Wry neck and to chase away biting flies that transmit Trypanosomiasis, CSN
Prayers for divine intervention	HS
NB: Text in red highlights practices that go ago	ainst animal welfare principles

### Table 16: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority Camel diseases in Marsabit county

Type of VMP	Active ingredient	Purpose	Target camel diseases
Antibiotic	Oxytetracycline	Curative and Preventive for secondary bacterial infections	Trypanosomiasis, Orf, Wry neck, CLA, HS, Camel flu, Camel pox, Pneumonia, Anaplasmosis
Antibiotic	Penstrep® procaine penicillin G and dihydrostreptomycin	Curative and Preventive for secondary bacterial infections	CLA, Camel flu, HS, Brucellosis, CSN, Camel pox, Pneumonia.
Antiprotozoal	Triquin® (Quinapyramine sulphate)	Curative	Trypanosomiasis, HS, Orf, Camel flu
Anthelmintic	Ivermectin	Curative	Mange, Ringworm, <mark>Camel pox,</mark> Anaplasmosis
Acaricide	Amitraz Organophosphate Synthetic Pyrethrin (Ectopor®)	Curative and Preventive	Mange and Ring worm
Metabolic support	Multivitamin	Supportive treatment	HS, Trypanosomiasis, Orf, Wry neck, CLA, Mange, Ringworm

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

## Camel Diseases: Protozoal diseases

## 5.1 Trypanosomiasis

## **Other names: Surra**

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dukan	Gandhi	Omar	Saar	Ekwakoit / Lokipi	Lesikonye

## Definition

- Camel trypanosomosis (Surra) is caused by a single celled (unicellular) protozoan parasite that is transmitted by biting flies.
- Trypanosomiasis is the most important disease in camel productions systems in the world.

## Distribution (Epidemiology)

- Most camels live outside Africa's tsetse belt meaning that Tsetse flies (Glossina species) are not involved in transmission of the disease. Other haematophagus flies like Tabanus, Stomoxys, Haematopota, Chrysops and Lyperosia transmit the disease through mechanical transmissions.
- Due to the wide geographic range of the haematophagus flies the disease is endemic in Africa, Asia, South and central America.

## Causative agent/ Aetiology

- Trypanosome evansi, types A and B,
- T. evansi is morphologically related to T. equiperdum—a derivative of T. brucei.
- During T. evansi evolution, it lost the mitochondrial (kinetoplast) DNA making it unable to infect invertebrate vector. This means it cannot establish life-stages in the Glossina fly (Tsetse fly) like other Trypanosome species.
- T. evansi has a wide host range. Surra affects

mainly camels and horses but buffaloes and cattle are also affected. Other species that develop severe disease include donkeys, mules, deer, llamas, dogs, cats, cattle and buffalo. Sheep, goats, pigs and elephants may occasional develop mild or chronic disease.

• In Pakistan, it has been found to be the most prevalent trypanosome species in donkeys.

## Mode of transmission and Pathophysiology

- Trypanosoma evansi is transmitted mechanically by haematophagus biting flies.
- No developmental stage in a vector has been demonstrated which differentiates the parasite from T. brucei.
- Tabanids/Tabanus (horsefly) play the major role in transmission. The flies establish a new infection through trypanosome-contaminated mouthparts. Trypanosomes remains infective on the proboscis for a short period only.
- Cattle, sheep, goats and antelopes often carry the parasite subclinically, acting as asymptomatic reservoirs.
- Transmission can also be transplacental resulting in disease of the foetus.
- Vampire bats in South and Central America are hosts, reservoirs and vectors of T. evansi; they transmit T. evansi mechanically in their saliva, and may develop high parasitaemia which may kill the bat. Recovered bats serve as

carriers.

- Carnivores may become infected after ingesting infected meat.
- Transmission in milk and during coitus has been documented.
- Numerous environmental and host factors influence the course of the disease, such as presence of other infections, nutritional status, age, pregnancy, previous exposure or immunosuppression and stress.
- The presence of the parasite in circulation results in chemical and morphological changes in the surface of RBC resulting in increased clearance of RBC by the body that exceeds the replenishment of RBC by the bone marrow hence the severe anaemia clinical sign observed.
- Infected animals' mucous membranes are pale and the packed cell volume (PCV) drops to below 25% (v/v) and can be as low as 10% (v/v).
- The herders may notice a characteristic odour of the camel's urine and identify infected animals by this sign alone. The odour of the urine may be due to ketone bodies, which are often elevated.
- in trypanosome-infected camels, abortion in all stages of pregnancy is common. If the foetus is full term it may be born alive but weak with parasitaemia and will die within two weeks of birth.
- In endemic areas, camel herds eventually reach an endemic stable state where some animals carry the trypanosome parasite for years while others never do. Within a herd there are all forms and stages of surra from new infections to subclinical and chronic conditions. The course of the disease varies widely. In Kenya, a small percentage of animals die within 2 to 5 months of contracting the disease. Some live for up to four years with subclinical infections and some eventually self-cure.

## Affected group

• Camels of any age can become infected. However, high incidence of infections is seen in young camels shortly after weaning.

## **Clinical signs**

- In the initial phase of the disease there is fever, lacrimation, shivering, reduced appetite and mild diarrhoea.
- The animal then develops progressive anaemia and has fluctuating body temperature with initial peaks of fever of up to 41 °C and after a few weeks as the animal enters chronic phase the temperature returns to normal.
- The animal's appetite is minimally affected but the animal begins to waste despite feeding well. Wasting manifests as a drooping hump.
- Animal is unable to walk long distances and has a rough hair coat.
- Most of the clinical signs are variable but typically include weight loss, decreased stamina, oedema (base of the neck, ventrum and eyelids), anaemia, intermittent fever, poor milk production, and late term abortion or calving of premature calves.

## Post mortem lesions

- Carcass is very thin (emaciated) and tissues are often pale (anaemic).
- Subcutaneous oedema of the feet, brisket, underbelly and eyelids has been reported.
- There is enlargement of the lymph nodes and spleen and pericardial effusion.

## Morbidity and Mortality Rates

- Morbidity rates in camel rearing areas varies from 20–70%. Case fatality rate (CFR) in untreated camels can be as high as 100%. Surra in cattle and buffalo tends to be chronic with a much lower CFR.
- Animals subjected to stress, like malnutrition, pregnancy, and physical labour, are more susceptible to disease causing mortality of 3%.
- In Marsabit county. Livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 7%, mortality rate of 2% and CF of 17%.

## Seasonality of occurrence

- In some countries incidence of surra increases significantly during the rainy season when biting fly populations is high
- Studies have reported varying seasonality of occurrence with some reporting outbreaks during the end of the rainy season while others reporting higher incidences during the dry season.
- In Marsabit county, Camel Trypanosomiasis is endemic and occurs throughout the year with most cases reported during the long dry season that runs from July to September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

#### Diagnosis Field based di

## Field based diagnosis

- Direct examination of the blood by light microscopy is a rapid diagnostic test that can be easily applied in the field. The basic technique, involves examination of fresh or stained buffy coat blood smears. Buffy coat smears are made after centrifugation of unclotted blood in a haematocrit tubehaematocrit centrifuge technique (HCT).
- The direct methods of trypanosome detection, utilise a wet blood film, Giemsa stained thick drop of blood and thin blood smears. The wet blood film is used to detect live parasite through observing parasite movement while the stained smears identify the parasite morphology and allows for parasitaemia calculation. The drawback of this method is that the animal should be in the acute phase of the disease when there is fever due to parasitaemia.
- Concentration techniques HCT is used to determine PCV and the buffy coat is Giemsa stained and is examined for the parasite. This can also be done in the field with batteryoperated mini centrifuge.
- Card agglutination and Latex agglutination test are useful antibody testing that can be used in the field.

## Laboratory diagnosis

• Confirmatory diagnosis is through ELISA

for chronic cases that detects antibodies and is useful for large-scale surveys. Polymerase chain reaction (PCR) has also proven useful in confirming trypanosomes in infected blood or tissues.

## **Differential Diagnosis**

- Other tsetse-transmitted trypanosomosis,
- · Chronic parasitism- helminthiasis

## Treatment

- Melarsomine dihydrochloride (Cymelarsan) is the latest Trypanocidal drug to be developed. It was first available for commercial use in 1992. It is used to control surra in camels via deep intramuscular injection at a dose rate of 0.25mg/kg body weight.
- The most effective drug used globally to treat Surra in camels is Quinapyramine sulphate and quinapyramine chloride (Triquin®). Triquin® is used as a curative as well as a preventive drug against T. evansi in camels. It is administered by subcutaneous injection at a dose of 8mg per kilogram body weight. Local tolerance at the injection site is low. However, the drug is quite efficient and the chemo prophylactic effect can last up to 4months. Triquin® is available in Kenya.
- Most of the drugs used to treat cattle trypanosomiasis are either not curative (homidium bromide, or Homidium Chloride=Novidium®) or are toxic at high

doses for camels (diminazene aceturate = Berenil®).

• Supportive treatment with multivitamin and improving nutrition will give the animals a better chance of recovery and survival.

## Prevention/control

- No vaccines are available and none are likely in the future as the trypanosome parasite is able to rapidly change its surface glycoproteins to avoid the host's immune response.
- Control measures include detection and treatment of infected animals, prophylactic treatment of susceptible animals, and protection of animals from biting flies through use of insect repellents like pour on especially during the rainy season when the biting flies' population is high.

## Zoonotic potential

• None for T. evansi. Despite having a wide range of host the protozoa can only infect nonhuman hosts as it is susceptible to cytolysis by the trypanolytic factor that is found in normal human serum.

### Notifiable disease

• No, but it is recognised by WOAH as a camel disease of economic importance.

### Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Avoid grazing animals in known areas with biting flies that transmit the parasite.

#### Further Reading

- 1. D. Röttcher, D. schillinger and E. zweygarth (1987). Trypanosomiasis in the camel
- (Camelus dromedarius). Rev. sci. tech. Off. int. Epiz., 1987, 6 (2), 463-470. https://www.WOAH.int/doc/ged/D8495.PDF
- 2. World Animal Health Organisation ( WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/ animal-diseases/technical-disease-cards/#searchform-header
- Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_reports/21



Figure 60 Emaciated camels showing lack of muscle mass, drooping hump, prominent and visible ribs, spine and hip bones (red arrows)



Figure 61 A good blood smear has a head, body and tail



Figure 62 Giemsa-stained blood smears from camel showing Trypanosoma evansi



## Figure 63 Ventral oedema in a camel due to Trypanosomiasis (Surra) infection and Subcutaneous oedema (expansion of the subcutis by clear gelatinous fluid



Figure 64 Pale camel lung due to anaemia and Normal pink-coloured lung

## Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib. dr.iastate.edu/vdpam\_reports/21

## <sub>Camel Diseases:</sub> Viral Diseases

## 5.2 Camel Contagious Ecthyma (CCE) -Orf

Other name: Scabby mouth, Sore mouth, Contagious Pustular dermatitis or Auzdik

## disease

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Umburur	Abthara	Afturo	Afturo	Ngiboruok / Lokutuk	Dicho

## Definition:

• Highly contagious viral dermatitis of camels characterised by pustule lesions on the lips, especially in young animals.

## Aetiology, Distribution/epidemiology:

- CCE is caused by Parapoxvirus (PPV) which is a large DNA virus of the family Poxviridae. Other members of the genus PPV are Orf virus (ORFV) that affects sheep and goats, Pseudo cow pox virus (PCPV), Bovine papular stomatitis virus (BPSV) and PPV of red deer in New Zealand (PVNZ).
- The disease has a worldwide distribution
- The disease is endemic in most camel keeping countries with variations in intensity of infection, morbidity and mortality rates.
- Orf outbreaks are most common during the rainy season and affects mostly young animals. The age group at risk are those less than one year of age. Cases have been reported in one month-old camel calves.
- The major factors associated with increased likelihood of CCE occurrence are season of the year (rains), camel age (calves more susceptible), camel movements and their

association with thorny trees.

• In most cases, the disease causes no mortality, but when camel calves are severely affected the pox-lesions interfere with the calves' ability to suckle or graze. Lesions can extend to eyelids leading to blindness and death.

## Zoonotic potential

- Yes, transmission is through direct contact with infected animals or indirect through fomites.
- Lesions in man appear as solitary or several skin lesions that appear commonly on the fingers, hands, or forearms. The lesions start as small, firm, papules, that proliferate and progress to a severe ulcerative, pustular dermatitis. The clinical manifestation of the disease and a personal history of contact with an infected animal are sufficient to diagnose orf virus in man.

## Morbidity and Mortality rate

- Morbidity rate is about 60-100% while the mortality rate is 5-20%.
- The camel calves die due to an inability to graze and suckle their dam.

• In Marsabit county livestock keepers in the year 2022 estimated the annual apparent morbidity rate of 6%, mortality rate of 2% and case fatality rate of 35%.

## Seasonal Occurrence

• In Marsabit county, CCE is endemic and occurs throughout the year, outbreaks are common during the long rainy season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan/	IR Gutoka
(April- June)					Akiporo	

## **Clinical signs**

- Pox-lesions first appear on the lips of affected animals as small papules that progressively develops into scabs on the lips, muzzle, nares and eyelids that later coalesce into fissured crusts on the lips. Swelling of the head and sometimes the neck is also reported.
- In young camels, lesions sometimes have a tendency to generalize. In this case, lesions occur all over the body, with prevalence on the distal area of the leg, inner thighs, and the perineum. Superficial lymph nodes of the head region such as parotid, submaxillary, and cervical lymph nodes, become enlarged.

## Post mortem lesions

- The papular lesions and scabs are found around mouth, nostrils and on eyelids.
- Intense pruritus results in haemorrhages and extensive skin excoriation.
- There is enlargement of superficial parotid, submaxillary and cervical lymph nodes.

## Diagnosis

- Clinical signs of papular lesions present around the mouth and nostrils in young camels.
- The typical parapoxvirus particles are diagnosed in scabs of camel skin using electron microscopy.
- Different serological tests are available with variable results.

## Differential diagnosis

- Camel pox
- Mange
- Fungal infections

## Treatment

- No treatment since it is viral.
- Local application of antiseptic solution such as povidone iodine or iodine ointment on the affected areas is recommended.
- Use of Gentian violet spray with oxytetracycline antibiotic also assists in drying lesions and prevents local secondary bacterial infection
- Antibiotic therapy is useful if the lesions become generalised and secondary infection has set in. Long acting oxytetracycline (20 to 30%) at dosage rate 1mL per 10 kilograms can be administered.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.

## Prevention and control:

- Affected animals must be segregated to stop the spread of the disease in the herd.
- Any sheep or goats kept with camels should be vaccinated against ORF. A vaccine is available for sheep and goats and can be used safely in camels.
- A recombinant vaccine is under research as a bivalent vaccine for both contagious ecthyma and camel pox.

## Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

· Avoid grazing animals in known areas with

biting flies that transmit the parasite.

- Isolate sick animals by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

#### Further Reading

 Khalafalla, A.I., El-Sabagh, I.M., Al-Busada, K.A. et al. Phylogenetic analysis of eight sudanese camel contagious ecthyma viruses based on B2L gene sequence. Virol J 12, 124 (2015). https://doi.org/10.1186/s12985-015-0348-7



Figure 65 Camel orf oral lesions (red oval) and enlarged sub-mandibular lymph node (red arrow)

Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib. dr.iastate.edu/vdpam\_reports/21



Camel orf lesions that have coalesced into scabs, photos courtesy of Morris Ogom and Diba Denge Animal Health Staff, Marsabit County

# 5.3 Camel Pox

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
FINNO /	FINNO/	NYINYILO	NARIRI EE	EKOLEMERI /	VERT - GALTE
BAGAA	BAGAA /		NTAMESI	ETUNE	
	ABDARA /				
	RABA				

## Definition:

• Camel pox is a highly contagious viral skin disease of camels characterised by generalised pustule formation. Camel pox is an WOAH notifiable skin disease.

## Causative agent

- Camel pox virus (CMLV)- CMLV genus Orthopoxvirus (OPV), is genetically similar to variola virus (VARV), the causative agent of smallpox in humans.
- Orthopox viruses are exclusively specie specific, CMLV infects only camels and variola only humans.

## Zoonotic potential

• Yes, CMLV infection has been documented in camel handlers.

## Distribution (Epidemiology)

- Disease occurs frequently during the rainy season due to increased population of suspected arthropod vectors biting flies.
- Disease occurrence is restricted to camels and is enzootic in almost every region, where camels are reared except in Australia.
- Mortality in adult animal's ranges from 10 to 28 % and in young animals (< 4 years of age) from 25–100 %. Mortality is also influenced by presence of other immune suppressing diseases like trypanosomiasis and helminthiasis.
- In Marsabit County, livestock keepers in the year 2022 estimate the apparent morbidity rate to be 3%, mortality rate to be 1% and case fatality rate of 33%. The disease is common in the months of October to December during the short rains season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Short Rains	Agaya	Agaya	Yeer	Ltumuren	Nyerube	Erupe/
(October-						Ngirupei
December)						

## Transmission-

- CMLV is transmitted by either direct or indirect contact. Direct transmission is either by inhalation of infected nasal or oral secretion and through skin abrasion or mechanical transmission by arthropods like the camel tick Hyalomma dromedarii.
- Indirect transmission is through contact with contaminated fomites. Affected camels contaminate the environment with shed scab

materials and secretions saliva, ocular and nasal discharges.

- Predisposing factors- Presence of chronically infected animals in close proximity with immune naïve animals is an important predisposing factor.
- Dried scabs shed from pox lesions contain live virus for at least 4 months. Poor nutrition and concurrent parasitic and bacterial infections often worsen the clinical disease. Animal

congregating in markets, watering and grazing areas are important sources of infections.

## Clinical signs

- Camel pox commonly occurs in naïve populations (herds that have not experienced an outbreak). The disease is more severe in younger animals (< 4 years).
- Fever and enlarged lymph nodes.
- Skin lesions that include vesicles or pustules with crusts that start at the head region before spreading to the neck, limbs, genitalia, mammary glands and perineum.
- Excessive lacrimation, muco-purulent nasal discharge and abortion in pregnant camels.
- Animals may also have diarrhoea.
- Skin lesions may take up to 4–6 weeks to heal. In the generalised systemic form of the disease, pox lesions can be found in the mucous membranes of the mouth and serosal surfaces of internal organs.

## **Differential Diagnosis**

- Contagious ecthyma (ORF)- Camel parapox virus (CPPV), Papillomatosis (wart) -Camelus dromedary papilloma virus (CdPV) and Urticaria-skin reaction to insect bites.
- Difference between camel orf and camel pox is the type distribution of lesions, in orf lesions coalesce and form crusts and are mainly located on the lips and nostrils and often cause oedema and enlargement of regional lymph nodes due to secondary bacterial infection. Camel pox lesions are mainly discrete pustular lesions that don't coalesce and occur all over the body of the animal, they are generalised. Orf is also more common in calves while camel pox occurs in all age groups. [
- Warts are small, cauliflower-like lesions also occurring on the lips but can be generalised in some animals. The wart lesions are not covered by surface crusts, do not coalesce and are not associated with oedema or regional lymph node enlargement.

## Post mortem lesions

• In the generalised form, the animal's external

skin and internal organs have pox lesions.

## Diagnosis

• Clinical and post-mortem lesions pf pox like lesions are used to make a tentative field based diagnosis.

## Sample collection and Laboratory diagnosis

- During the viraemic stage of the disease (within the first week of the occurrence of clinical signs) camel pox virus can be isolated in cell culture from heparinised blood samples, or viral DNA can be detected by the polymerase chain reaction (PCR) from blood in EDTA (ethylene diamine tetra-acetic acid). EDTA blood samples should be collected in a sterile manner by venepuncture and stored at 4°C for up to 2 days prior to processing, but should not be frozen or kept at ambient temperatures.
- Unclotted blood obtained for serum samples should be collected in plain tubes with no anticoagulant. The sample in plain tubes should be left to stand at room temperature for 4–8 hours until the clot begins to contract, after which the tube is centrifuged at 1000 g or rpm for 10–15 minutes. Separated serum can then be collected with a pipette and held at 4°C for a short period of time or stored at –20°C.
- Skin biopsy or organ tissues should be a minimum weight of 2 grams for virus isolation and histopathology. Tissue samples collected for virus isolation should be placed in a virus transport medium, such as Tris-buffered tryptose broth or minimal essential medium (MEM) without foetal calf serum (FCS) and kept at 4°C for transportation and stored at -80°C until processed.
- Tissue for histology should be placed immediately after collection into 10% formalin. The formalin should be ten times the sample volume.
- Confirmatory diagnostic tests include antibody detection using ELISA and virus identification through egg or cell culture, molecular techniques (PCR) and virus agent visualisation

using a Transmission electron microscopy (TEM) that differentiated orf and camel pox virus. Use of TEM is a rapid confirmatory test.

## **Treatment and Control**

- Viral disease there is no specific treatment
- Animals that develop secondary infection characterised by a fever or unthriftiness after 1 week should be treated with one dose of long acting oxytetracycline at dose rate of 10 mg/kg body weight IM. Multivitamin therapy can also boost the animal's immunity.
- Quarantine herds.
- Life-long immunity follows after natural infection hence there may be no need to vaccinate affected herds.
- There is commercial cell attenuated camel pox vaccine using isolates from UAE (strain CaPV298-2). The vaccine confreres immunity of up to 6 years. However, a booster vaccination is recommended in young animals vaccinated below 6 months as there will be maternal antibody interference.

## Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Avoid grazing animals in known areas with biting flies that transmit the parasite.
- Isolate sick herds by ensuring herds with lesions do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff

#### Further Reading

- Vinayagamurthy Balamurugan, Gnanavel Venkatesan, Veerakyathappa Bhanuprakash and Raj Kumar Singh (2013). Camel pox, an emerging orthopox viral disease. Indian J. Virol. 24(3):295–305. https://www.ncbi.nlm.nih.gov/pubmed/24426291
- World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animalhealth-and-welfare/animal-diseases/technical-diseasecards/#searchform-header
- Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https:// lib.dr.iastate.edu/vdpam\_reports/21



Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib. dr.iastate.edu/vdpam\_reports/21





## Figure 69

Post mortem lesions of systemic form of camel pox in naturally infected animal

A. Multiple erosive, ulcerative and vesicular lesions (arrow) in the mucosa of tongue. B. Multiple pox lesions with haemorrhagic (white arrow) and ulcerated (black arrow) areas on lung surface

C. Multiple raised pox lesions (arrow) on lung surface.

D. Liver showing minute pox lesions (arrow) and pale areas of necrosis.E. Abomasum showing thickened congested mucosa (white arrow) with ulcerations and pox lesions (black arrow).



Picture source: Thomas Lenyakopiro, CDR, Samburu County

## 5.4 Acute Respiratory Disease Syndrome (ARDS)

Other names: Camel parainfluenza, Camel Cough, Camel Flu

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qufaa	Qufaa	Yahar	Lchama	Lowala	Furs-Galte

## Definition

- ARDS is described as a severe respiratory infection of camels characterised by fever, cough, initial serous nasal discharge that becomes mucopurulent, laboured breathing and terminal progression to sternal recumbency with extension of the neck before death.
- The disease affects all ages of camels but weaner calves between the ages of 1 to 2 years are the most severely affected.

### Distribution (Epidemiology)

- The disease has been described in all camel keeping countries globally.
- In Kenya, the most recent outbreak occurred from June 2020 and was reported in most camel keeping counties that includes Mandera, Wajir, Marsabit, Isiolo, Samburu, Turkana and Garissa.
- During the recent 2020 outbreak, camel owners reported that they had experienced a similar disease in the past which they called 'Camel flu' that presents with milder symptoms. However, the current outbreak was presenting with more severe symptoms and had a longer course duration compared to the seasonal camel flu known to many of them.
- ARDS begun during the long rainy season (April -June) with only a few cases reported. However, the long dry season (July – September) resulted in explosive outbreaks with daily reports of camel deaths in all Northern pastoralist counties of Kenya.
- There were no reports of animals having been moved to new area prior to the outbreak.

### Causative agent/ Aetiology

- The definitive causative agent for acute respiratory diseases has not been conclusively determined. It is hypothesised that the disease starts as a parainfluenza 3 infection that becomes complicated by secondary bacterial infection.
- A variety of viral, fungal, bacterial and parasitic microorganisms have been associated with outbreaks of respiratory disease among camels.
- The causative agents identified are a normal flora in the upper respiratory tract of camels. The disease requires predisposing circumstances that cause the normal flora to invade respiratory tissues and cause serious disease.
- A recent 2020 metagenomic sequencing analysis of nasopharyngeal swabs sampled from 108 MERS CoV-positive dromedary camels in UAE identified other co-infecting viruses from 10 viral families including Coronaviridae, Nairoviridae, Paramyxoviridae, Parvoviridae, Polyomaviridae, Papillomaviridae, Astroviridae, Picornaviridae, Poxviridae, and Genomoviridae.
- A similar study in Sudan that analysed lung tissue identified parainfluenza virus 3 (PIV3), influenza viruses A, B and D, adenovirus, respiratory syncytial virus (RSV), and infectious bovine rhinotracheitis (IBR).
- The most common pathogenic bacteria isolated during outbreaks include;
  Staphylococcus aureus, Corynebacterium pyogenes, Streptococcus pyogenes, Escherichia coli, Klebsiella pneumonia, Pseudomonas aeruginosa, Trueperella pyogenes (formerly

Arcanobacterium pyogenes), Mannheimia haemolytica and Pasteurella multocida.

# Mode of transmission and pathophysiology and affected group

- Direct transmission through inhalation of respiratory droplets especially due to the cough and indirect transmission through contaminated fomites.
- Studies have shown that the most commonly reported risk factors for viral or bacterial pneumonia are age and season. The highest incidence of pneumonia is reported during the cold season in all camel ages. Proliferative bronchopneumonia and pleuropneumonia are more frequent in older camels (about 10 years of age) while interstitial pneumonia and lung abscesses are more frequently seen in younger camels (6 months to 4 years of age).

## **Clinical signs**

- The first clinical symptom observed is fever which is characterised by the animal isolating itself from the rest of the herd.
- After 2 days the animal develops a cough and serous nasal discharge that lasts for a week

after which the nasal discharge becomes mucopurulent. In the second week following onset of clinical symptoms the affected animals develop a more severe cough, difficulty in breathing that forces them to extend the neck and become recumbent. If there is no treatment in the first 2 to 3 days after onset of symptoms the animals dies within 7 days after onset of symptoms especially in camels that are under 2 years.

- Recovery for those that are older than 3 years of age occurs with or without treatment and it takes 2-3 weeks from onset of clinical symptoms to recovery.
- The Kenya 2020 outbreaks estimated an apparent morbidity rate of 35%, mortality rate of 1% and case fatality of 2%.
- In Marsabit county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 8%, mortality rate of 5% and a case fatality of 64%.
- The disease in Marsabit county occurs throughout the year but epizootic outbreaks occur during the long dry season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

### Post mortem lesions

- The gross lesions observed during the Kenyan 2020 outbreak included hydro-peritoneum with about 60 mls of fluid. Hydrothorax with about 30mls of thoracic fluid.
- Fibrinous adhesion of the lung to the thorax. The right lung was severely congested with fibrinous bronchopneumonia appearing as red and grey hepatisation. The lung was marbled in appearance and the cut section oozed froth and purulent exudates with the lower section showing varying atelectasis.
- Thick mucoid exudates were present in the trachea and bronchial lumen. There were no gross lesions on the liver, kidney and spleen.

## Diagnosis

- Samples that yield bacterial isolates are nasal, nasopharyngeal, tracheal swabs and lung tissues.
- Samples that yielded viral agents are nasal swabs and lung tissues.
- Bacterial isolation using routine culture methods with different media such as nutrient agar, blood agar, brain heart infusion, mannitol salt agar, MacConkey agar and brilliant green agar followed by identification using morphological and biochemical characteristics of the isolated strains such as colony morphology, Gram staining, spore forming ability and acid-fast staining. ELISA or PCR can be used for virus identification.

During the 2020 outbreak, 99 nasal swabs from camels in Marsabit county submitted for bacterial culture and sensitivity yielded eight (8) genera of bacterial species that were identified on the basis of their phenotypic and biochemical properties. The isolated bacteria included Pseudomonas aeruginosa 20.2% (20/99), Klebsiella pneumoniae 19.2% (19/99); Staphylococcus aureus 15.2% (15/99); Bacillus and Escherichia coli each with a 13.1% (13/99); Streptococcus 8% (8/99); Proteus mirabilis 6.1% (6/99) and Serratia 5.1% (5/99).

## Treatment

- Antimicrobial susceptibility testing (AST) is necessary for
- All antibiotic drugs use in camels is extra-label meaning that the drug manufacturer has not indicated for camel use.
- Antibiotic treatment needs to be given in

the early phase of the disease for them to be effective. Ideally before the laboured breathing sets in. Treatment is futile if given when animal has become recumbent.

- In severe cases dexamethasone can be used to reduce inflammatory accumulation of fluid in the respiratory tract. Unlike in cattle where pregnancy is supported by placenta hence can result in abortion when dexamethasone is used. Camels pregnancy is supported by the ovarian located Corpus luteum (CL). CL is not affected by dexamethasone administration.
- The 2020 Marsabit outbreak investigation revealed that bacterial isolates were sensitive to most antibiotics available in the market such as oxytetracycline, Penicillin and Streptomycin (Penstrep®), Sulphamethoxazole and Trimethoprim (Intertrim®), and Amoxycillin (Betamox®). The dosage regimes are given in the table below.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting in severe cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM for 3 to 5 days.
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM neck muscles q48 hours PRD
Amoxycillin 150 mg Long acting	Broad-spectrum antibiotic with bactericidal action	1 ml per 10 kg BWT deep IM neck muscles q 48 hours.
Sulfamethoxazole 200 mg Trimethoprim40 mg	Broad-spectrum antibiotic with bactericidal action	Twice daily 1 ml per 10 - 20 kg BWT for 3 - 5 days. IM neck muscles
Enrofloxacin 100 mg	Broad-spectrum antibiotic with bactericidal action	Single-Dose by SC only 7.5-12.5 mg/kg of BWT (10 to 20 ML) SC
Multivitamin	Boost immunity	Adult Camel: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

\* Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q and mL- Milliliter, body weight-BWT, kg- kilograms, mg- milligram. \*\*By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC). NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

#### Prevention/control

- There are no vaccines available in Kenya to prevent camel respiratory conditions.
- Vaccination against Pasteurellosis is routinely practiced in the Middle East. There are 3 vaccine preparations in the market; Dense bacterins combined with either alum adjuvant or oil adjuvant, and Formalin-Inactivated bacterins. The oil adjuvant bacterin provides protection for up to one year and the alum bacterin for 4–6 months.
- Maternal antibodies interfere with vaccine efficacy in calves, calves should therefore be vaccinated after 6 months.
- Ensure animals have a good plane of nutrition and internal and external parasites have been controlled. Camels in good body condition and are generally healthy are more resistant to pneumonia incidences due to their strong immunity.

## Zoonotic potential - No Notifiable disease- No

## Ethnoveterinary treatment practices Medicinal plants

## Acacia Nubica, A.tortilis, A.bussei- bark soaked in cold water for a few hours and given orally.

• Zanthoxylum usambarense - Fruits/Seeds pounded and mixed with cold water

## Traditional practice

• Tobacco snuff – animal force to inhale the powder.

### Ethnoveterinary control practices

- Isolate sick herds by ensuring they do not share same grazing land and watering points
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff
- Use of Amulet the camel boma live fence should include Acacia tree barks to wade of disease.
- Request for religious blessing to safeguard the herd from disease. The blessing ritual is done by specific people who have being given the divine ability to protect livestock and property from ills like diseases.

#### **Further Reading**

- Li Y, Khalafalla AI, Paden CR, Yusof MF, Eltahir YM, Al Hammadi ZM, et al. (2017) Identification of diverse viruses in upper respiratory samples in dromedary camels from United Arab Emirates. PLoS ONE 12(9): e0184718. https://doiorg/10.1371/journal.pone.0184718
- M. S. Abubakar, M. Y. Fatihu, N. D. G. Ibrahim, S. B. Oladele and M. B. Abubakar (2010). Camel pneumonia in Nigeria: Epidemiology and bacterial flora in normal and diseased lung. African Journal of Microbiology Research Vol. 4(23), pp. 2479-2483.
- Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_reports/21
- Zuhair Bani Ismail (2010). Pneumonia in dromedary camels (camelus dromedarius): A review of clinico-pathological and etiological characteristics. Journal of Camel Practice and Research. https://www.indianjournals.com/ijor. aspx?target=ijor.jcpr&volume=24&issue=1&article=007



## Figure 71

Sternal Recumbency and nasal discharge in a camel affected by ARDS in Samburu County, April 2020.

> Picture source: Emmanuel Lesiantam, Animal Health Assistant, South Horr, Samburu County





Figure 73 Hydrothorax and lung adhesions to the rib cage (Left) and exudate of mucopurulent discharge from cut lung surface (Right) in a camel affected by ARDS.

Picture source: Emmanuel Lesiantam, Animal Health Assistant, South Horr, Samburu County

## Camel Diseases: Bacterial Diseases

## 5.5 Haemorrhagic Septicaemia (HS)

## Other names: Pasteurellosis, Pneumonic Head

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qandich	Qandich / Qando	Khanid	Nalngaringari	Elkunoit / Longauroi	Jaaro

## Definition

- Pasteurellosis / Haemorrhagic septicaemia is an acute fatal respiratory bacterial disease of camels caused by Pasteurella multocida and Mannheimia haemolytica.
- Disease is characterised by fever, oedema of the throat region, dyspnoea, and sudden death.
- HS has a complex pathogenesis linked to predisposing factors that causes the two bacteria that are normal flora of the upper respiratory tract to become pathogenic organisms.

## Distribution (Epidemiology)

- Pasteurellosis occurs in camels throughout Sub-Saharan Africa, Egypt, Saudi Arabia, India, Iran, Iraq, and Russia.
- Outbreaks of HS like disease that caused large mortalities in camels (Camelus dromedarius) in Asia and in Africa have been reported since 1890.

### Causative agent/ Aetiology

 HS is caused by two gram-negative coccobacillus bacteria namely Pasteurella multocida and Mannheimia haemolytica. The two bacteria have several serotypes that cause disease in camels

- HS has a complex actiology and recent PCR studies have revealed the serotypes involved are:
  - P. multocida type A, B and E (capsular and non-capsular strains)
  - Mannheimia haemolytica
  - M. granulomatis (new species identified)

### Mode of transmission and Pathophysiology

- The disease occurs when the resistance of the body is lowered by environmental influences such as trekking or transportation over long distances, deficiencies of dietary vitamins and minerals, heavy parasitic infestation (Trypanosomiasis) and sudden changes in weather.
- The role of Parainfluenza-3 virus is not clear, however, coinfection of HS and PI-3 virus has been reported in camels.
- P. multocida is transmitted by direct contact with infected animals and indirectly through contact with contaminated fomites
- Incubation period varies from 2 to 5 days depending upon the virulence of the organism and the degree of immunosuppression of the camel. The organism enters into the blood stream through tonsils and proliferates rapidly.

It causes a septicaemia in animals within 10-24 hours, leading to high temperature, swelling of the throat region, pulmonary oedema, fibrinous pneumonia, diarrhoea, and prostration. Affected camels rarely recover, and death usually occurs within 2-3 days.

• Biting arthropods are not significant vectors for transmission.

Affected group

• All age groups are equally affected

## **Clinical signs**

- Clinical signs include increased rectal temperature (40°C), pulse, and respiration rate, dyspnoea, dullness, depression, and abdominal pain associated with haemorrhagic enteritis. There is subcutaneous swelling of the neck and submandibular space.
- Mandibular and cervical lymph nodes are enlarged and painful.
- Severe dyspnoea is characterised by camels showing dilated nostrils and open-mouthed breathing.
- Some cases, have tar coloured faeces (melena), abdominal pain, and coffee coloured urine.
- Prognosis is guarded and death occurs within 2-3 days. Both recovered and sick animals discharge the organism through excretions and secretions of the body.

## Post mortem lesions

- Camels that succumb to infections have gross lesions indicating of septicaemia
- All organs are congested and serosa surfaces have petechial haemorrhages throughout the body.

- There is excess serosanguineous fluid present in the thoracic (hydrothorax) and abdominal (hydroperitoneum) cavities
- The trachea is filled with froth and there is occlusion of nasal passage with thick sticky mucous
- HS bacteria, causes bronchopneumonia and the lung cut surface oozes exudate. The lung tissue does not collapse when cavity is open

## Morbidity and Mortality Rates

- Morbidity is low, but mortality can be as high as 80%. Morbidity depends on immunity and environmental conditions, including both weather and husbandry practices; morbidity is higher when animals are herded closely, are in poor condition, or exposed to wet conditions.
- Mortality is nearly 100% unless the animal is treated very early on in course of the disease
- In Marsabit county, livestock keepers in the year 2022 estimated an annual apparent morbidity rate of 9%, mortality rate of 4% and case fatality rate is 44%.

### Seasonality of occurrence

- HS is most common during the rainy season or long dry season when animals are in nutritional stress
- In Marsabit county, livestock keepers indicated that HS occurred throughout the year.
  However, explosive outbreaks were common during the long and short rainy seasons. The local dialect names for these two seasons are shown in the table below.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains (April- June)	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan or Akiporo	IR Gutoka

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Short Rains	Agaya	Agaya	Yeer	Ltumuren	Nyerube	Erupe/
(October- December)						Ngirupei

### Diagnosis

- Tentative field based diagnosis using clinical signs and post-mortem lesions.
- Confirmatory diagnosis is through demonstrate the organisms-in live animals the organism may be difficult to culture and identify from venous puncture blood samples in addition, nasal swabs of sick animals also yield inconsistent culture results.
- Samples for isolation should be collected immediately after death (less than 4 hours) and include; blood from the heart, nasal and tracheal swabs and tissue impression smears of the spleen.
- In animals that died a long time ago (> 2 days) which is often the case in pastoralist setting. The femur and ulna long bones can be collected for culture of the bone marrow.
- Diagnosis is through bacterial culture and identification of the organism through biochemical sugar tests.
- ELISA and PCR molecular tests can also be used on blood, nasal swabs and tissue samples.

## Treatment

- Antimicrobial susceptibility testing (AST) is necessary for P. multocida for which resistance to commonly used antimicrobial agents has occurred.
- All antibiotic drugs use in camels is extra-label meaning that the drug manufacturer has not indicated for camel use.
- Antibiotic treatment needs to be given in the early phase of the disease for them to be effective. Before laboured breathing and recumbency sets in.
- In severe cases dexamethasone can be used to reduce inflammatory accumulation of fluid in the respiratory tract. Unlike in cattle where pregnancy is supported by placenta hence can result in abortion when dexamethasone is used. Camels pregnancy is supported by the ovarian located Corpus luteum (CL). CL is not affected by dexamethasone administration.

# Differential Diagnosis (DDx)Other Bacterial and Viral Pneumonias

• Anthrax or Clostridial infections in peracute cases that causes sudden death.

Active ingredient	Indication	Dosage* and Route**
10% Oxytetracycline short acting in severe cases	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT slow IV or IM for 3 to 5 days.
Oxytetracycline Long acting 20- 30%	Broad spectrum bacteriostatic antibiotic	1 mL per 10 kg BWT deep IM neck muscles q48 hours PRD
Amoxycillin 150 mg Long acting	Broad-spectrum antibiotic with bactericidal action	1 ml per 10 kg BWT deep IM neck muscles q 48 hours.
Sulfamethoxazole 200 mg Trimethoprim40 mg	Broad-spectrum antibiotic with bactericidal action	Twice daily 1 ml per 10 - 20 kg BWT for 3 - 5 days. IM neck muscles
Enrofloxacin 100 mg	Broad-spectrum antibiotic with bactericidal action	Single-Dose by SC only 7.5-12.5 mg/kg of BWT (10 to 20 ML) SC
Multivitamin	Boost immunity	Adult Camel: 20-30 mL IM or SC PRD Calves: 5-10 mL IM or SC PRD

\* Once daily- SID, Twice daily- BID, Three times daily- TID, Four times daily- QID, As needed – PRD Repeat- q and mL- Milliliter, body weight-BWT, kg- kilograms, mg- milligram. \*\*By mouth (PO), Intramuscular (IM), Intravenous (IV) and Subcutaneous (SC). NB: Treatment suggestion is a guideline always read drug manufacturer's insert and label dosage

## Prevention/control

- During outbreaks mass medication of affected herd.
- HS vaccine is commercially available but it is not marketed in Kenya. Vaccination should be done in camels using the alum precipitated or oil adjuvant Pasteurella multocida type B vaccine, before rainy season or during early phase of an outbreak. Vaccines provide protection for 6–12 months.
- There is also, a vaccine against Mannheimia haemolytica that offers protection against pneumonic Pasteurellosis.

**Zoonotic potential** – Yes, Pasteurella multocida can infect humans through skin wounds and immunocompromised individuals through aerosol droplets.

## Notifiable disease- No

## Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick herds by ensuring herds showing signs do not share same grazing land and watering points.
- Report outbreaks to the CDRs, chief, ward administrators or county veterinary staff.
- Avoid taking animals to areas with HS outbreaks.
- Use of Amulet the camel boma live fence should include Acacia tree barks to wade of disease.
- Request for religious blessing to safeguard the herd from disease. The blessing ritual is done by specific people who have being given the divine ability to protect livestock and property from ills like diseases.

#### Further Reading

- Gluecks, I.V., Bethe, A., Younan, M. et al. Molecular study on Pasteurella multocida and Mannheimia granulomatis from Kenyan Camels (Camelus dromedarius). BMC Vet Res 13, 265 (2017). https://doi.org/10.1186/s12917-017-1189-y
- Yaeger, Michael; Coatney, John W.; Dioli, M.; and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_reports/21

## Figure 74

Camel affected with HS showing fibrinous nasal discharge and open mouth breathing (Left) and spontaneous haemorrhages and swelling of the head and submandibular regions (Right)





## Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_ reports/21



## Figure 76

Pulmonary oedema demonstrated by failure of lung tissue to collapse and lung tissue hepatisation photo courtesy of Dr. Bernard Chege, Marsabit County

# 5.6 Caseous Lymphadenitis (CLA)

## **Other name: Pseudotuberculosis**

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qarfat	Qarfat	Orokajire	Lngarngar / Nkimek	Abuus/ Ngabulon	Dhuorich

## Definition

- Caseous lymphadenitis (CLA) is a chronic bacterial disease of camelids, wild and domestic small ruminants, pigs and man.
- CLA lesions usually appear clinically as abscesses in the superficial lymph nodes.
- Economic losses are associated with progressive weight loss, carcass trimmings and skin condemnation at abattoirs.

## Distribution

- The infection is spread by inhalation, ingestion or directly through wounds.
- CLA has been reported in all camelids that include the old world camels: Camelus dromedarius and Camelus bactrianus, including the Australian feral dromedary

population and the new world camels: Llamas and Alpacas

- CLA has a high morbidity rate of 90% and mortality rate of 28%.
- Both, young and adult camels are affected by the disease.
- In Marsabit county. Livestock owners in the year 2022 estimated an annual apparent morbidity rate of 2% with no mortality reported.
- The disease in Marsabit county occurs during the short and long rainy seasons.
- The local dialect names for these two seasons are shown in the table below.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Short Rains	Agaya	Agaya	Yeer	Ltumuren	Nyerube	Erupe/
(October- December)						Ngirupei

## Causative agent and pathogenesis

- Corynebacterium pseudotuberculosis is a short, irregular ovoid Gram-positive rod almost resembling a coccus. Smears from abscesses show marked pleomorphism.
- Cultured colonies are small, white and dry and can be surrounded by a narrow zone of haemolysis when sheep or cattle blood is used.

The plates should be incubated at 37°C for at least 48 hours.

• Corynebacterium pseudotuberculosis is a Gram-positive rod-shaped, intracellular, facultative aerobic bacterium with worldwide distribution. The facultative, intracellular bacterium multiplies in infected phagocytes and gets disseminated via lymph or blood to secondary sites where it causes abscesses of different size.

- Bacteria has two biotypes known: ovine/ caprine and equine/bovine. Recently it has been shown, that strains isolated from dromedaries in the United Arab Emirates and Kenya belong to both serotypes, serotype I (biotype ovis) and serotype II (biotype bovis) using the nitrate reduction test.
- CLA is spread primarily through direct contact of bacteria excreted from the abscesses caused by Corynebacterium pseudotuberculosis and indirectly through contaminated fomites.
- The bacterium can survive several months within the environment and the soil, making the containment of the disease difficult to control.

## **Clinical signs**

- Development of superficial abscesses localized mainly in the cephalic, prescapular and prefemoral lymphnodes is pathognomonic of the disease in camels. The lymphnodes swellings are cold, closed and painless. They swollen external lymphnodes are the size of a lemon or orange.
- In camels, the characteristic laminated (onion ring) pattern seen in goats has not been described.
- In camelids three different types of pyogranulomatous lesions have been observed: Single abscess, when ripe and opened, the abscess extrudes thick, white-cream like pus; Large central abscess with multiple small abscesses in the peripheral connective tissue capsule and Multiple abscesses and no central abscess.
- A few cases have been seen in dromedaries whereby the abscess breaks through the ribs and the organisms enter the lung producing severe bronchopneumonia with pulmonary caverns

## Post-mortem lesions

• Camels rarely die of the disease and in most cases CLA is an incidental finding during slaughter. Most common lesions are pyogranulomatous abscess of the internal organs and lymphnodes.

## Diagnosis

- Filed based diagnosis can be made through Gram stained impression smears from abscesses. CLA agent appears as a short, ovoid Gram-positive rods almost resembling a coccus.
- Bacterial culture of lymph node swabs or lymphnodes tissues reveals mucoid, opaque, greyish-white colonies the bacteria cultured can be confirmed using API biochemical tests or molecular PCR tests. bacilli arranged as Chinese letters or Vshape.

## **Treatment and Control**

- Antimicrobial treatment is often not rewarding and other treatment methods should be considered. Since erythromycin is able to penetrate tissues. It is proposed a combination of penicillin and erythromycin to treat pseudotuberculosis in camels.
- In cases with multiple abscesses, surgical and antibiotic treatments are recommended Subcutaneous ripe abscesses can be lanced and cleaned out on a daily basis with iodine solution. It is of paramount importance to provide strict aseptic methods,
- Globally, there are no commercial vaccines for camels. However, commercial ones exist for goats and sheep.
- Cull from the herd infected animals.
- Purchase of animals should only be allowed from herds with no history of abscessation

## Zoonotic potential

• Yes, can infect man skin wound, always wear gloves when treating abscesses.

## Notifiable disease – No

Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

• None

#### Further Reading

 U Wernery and J Kinne. Caseous Lymphadenitis (Pseudotuberculosis) in Camelids: A Review. Austin J Vet Sci & Anim Husb. 2016; 3(1): 1022. ISSN: 2472-3371 https://austinpublishinggroup. com/veterinary-science-research/fulltext/ avsah-v3-id1022.php

## Figure 77

Cold, closed painless abscesses up to the size of a lemon or orange in a dromedary camel with bilateral inferior cervical lymph node abscesses







## Figure 78

Incised mandibular lymph node lesion showing greenish cheesy pus (Left) and Incised inferior cervical lymph node lesion showing enlargement and congestion without abscess formation (Right)

### Picture source:

https://www.bibliomed.org/mnsfulltext/31/31-1509535551.pdf?1630646409



## Figure 79

Grams stain of culture showing Gram positive cocco-bacilli arranged in a Chinese pattern (black arrows)

### Picture source:

Singh, R. (2018). Pathology of Caseous Lymphadenitis in Slaughtered Goats Associated Infection with Corynebacterium Pseudotuberculosis. Journal of Animal Research, 8.

# 5.7 Contagious Skin Necrosis (CSN)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qandich	Qandich / Qando	Khanid	Nalngaringari	Elkunoit / Longauroi	Jaaro

## Aetiology/ causative agent

- Contagious skin necrosis (CSN) is a chronic inflammation of the skin primarily caused by the bacteria Staphylococcus aureus.
- CSN lesions are characterized by necrosis, suppuration, and sinus formation in the skin, and enlargement of regional lymph nodes.
- Lesions predominantly occur on the skin of the back, hump, shoulders, base of the neck, and inguinal region, but may also occur on any other part of the body.
- CSN primarily affects young camels of both sexes and is more prevalent in the hot months of the year.
- Different microbial agents have been isolated from CSN lesions; the most predominant being Staphylococcus aureus.
- Other organisms isolated include; Streptococcus species, Corynebacterium pyogenes, Pseudomonas aeruginosa, Escherichia coli, Bacillus subtilis, Erysipalothrix species, Actinomyces species, Lactobacilli as well as a variety of fungal species.
- Diagnosis of CSN is usually based on clinical signs and distribution of the lesions while the causative agent (s) can be determined using standard microbiological method

## Transmission and Predisposing factors

- Presence of arthropod biting flies is associated with spread of disease in a herd.
- Disease is prevalent during hot months of the year.
- Prevalence of CSN is common in young animals (<5 years). One study in Egypt found that disease prevalence increased as the age of animal increased till 5.5 years and thereafter decreased gradually by further increase in the age.

## Occurrence

- CSN occurs in all regions of the world where camels are raised.
- In Marsabit county, livestock keepers in the year 2022 indicated that CSN was endemic and occurs throughout the year with no seasonal preference. The livestock keepers also estimated the annual apparent morbidity rate to be 7% with a 1% mortality rate and 20% case fatality rate.

## Main clinical signs

- Both male and female animals are affected equally
- The lesions start as circular sores that a few centimetres in diameter the sore area then begin to discharge fluid. A thick layer of dead skin/scab covers the lesion and after 7 to 14

days the thick scab falls off, leaving a circular ulcer that may extend deep into the skin dermal layer and does not heal

- The lesions cause irritation to the animals, camel try to rub and bite the affected skin area.
- The draining lymph nodes may become enlarged
- Rarely, does the camel develop septicaemia (blood poisoning)
- When the sore heals, it leaves a star shaped scar

## **Differential Diagnosis**

- Abscess due to other bacteria like Corynebacterium that causes Caseous lymphadenitis
- Infected saddle wounds

## Diagnosis

- Clinical diagnosis of painful skin sores, on which the hair falls out. The sores are usually found on the neck, shoulders, withers, sides and quarters, and rarely on the lower legs.
- Collect sterile swab smear and gram stain if there is a laboratory for bacterial culture and antibiotic sensitivity. Sterile swabs should be collected from the centre of the ulcer lesion once the necrotic scab is removed.
- The swab should be put in transport media like Stuart's transport medium. Where possible use swabs already pre-packed in media.

## Post mortem lesions

 Animals rarely die of CSN, if they do signs will be similar to those due to septicaemia with haemorrhages seen in mucosa and serosa surfaces.

## **Treatment and Control**

- Local treatment of sores involves washing with warm water, removal of the dead tissue, then clean with diluted lugol's iodine. Spray the sore with antibiotic spray that has gentian violet, oxytetracycline or gentamycin antibiotics. The gentian violet prevents flies from laying eggs resulting in myiasis.
- Inject a broad-spectrum antibiotic such as long-acting oxytetracycline or penicillinstreptomycin at a dosage rate of 1 mL for each 10-kilogram body weight by intramuscular route. It should be noted that organism has been shown to have high levels of antibiotic resistance and practitioners are encouraged to take bacterial swabs for culture and sensitivity especially in herds reporting recurrent occurrence.

### . Zoonotic potential

• No

#### Notifiable disease

• No

## Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals from herd
- Hand pick ticks
- Regular acaricide use especially pour on that work well as fly repellents.

#### Further Reading

- World Animal Health Organisation (WOAH) technical disease cards https://www.WOAH.int/en/what-we-do/animal-health-and-welfare/ animal-diseases/technical-disease-cards/#searchform-header
- 2. A field manual of camel diseases: Traditional and modern health care for the dromedary. Compiled by Ilse Köhler-Rollefson, Paul Mundy and Evelyn Mathias. http://vetbooks.ir/a-field-manual-of-camel-diseases-traditional-and-modern-healthcare-for-the-dromedary/
- Studies on contagious skin necrosis and trypanosomosis in camels. Maha I. Hamed, Mahmoud R. Abd Ellah, Infectious Diseases, Clinical Laboratory Diagnosis. Department of Animal Medicine, Faculty of Veterinary Medicine, Assiut University 71526, Egypt. https://pdfs. semanticscholar.org/79fa/a0bc28058cf4778b783f3ce20a2a5a37fcf9.pdf



Figure 80 Contagious skin necrosis (CSN) on the hind leg of a one-year-old camel calf. Photo: Abdul Raziq

> Picture source: https://doi.org/10.1186/1746-4269-6-16



Figure 81 CSN ulcer lesion once crust is removed, ulcer usually are filled with white pus material

> Picture source: https://pdfs.semanticscholar.org/79fa/ a0bc28058cf4778b783f3ce20a2a5a37fcf9.pdf



Figure 82 Cutaneous Skin Necrosis lesion on the neck of a camel, source: Maurizio Dioli

## Camel Diseases: Parasitic Diseases

## 5.8 Camel Mange

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Chito	Chito	Haddo	Lpepedo	Emitina Pepedo / Ekokoi	Gogosich

- Aetiology: Mange is caused by the mite (Sarcoptes scabiei var cameli).
- **Clinical signs:** The mite burrows through the skin producing irritation and intense pruritus, causing the camel to scratch against objects. Affected areas may appear reddened (inflamed) and the hair will have been rubbed off. Infection leads to loss of body condition, due to decreased grazing time.
- Transmission and age group affected: The disease is highly contagious and is more common in the young. It can spread directly from animal to animal, particularly when it is cold and animals huddle together. It can also be spread by fomites (saddles, harnesses) or in areas of common use (bedding, tree trunks,

dust baths). Young calves that are in poor body condition are more susceptible.

- Morbidity and mortality rate: In young animals' morbidity rates can be as high as 100% while mortality rate is lower at 30%. In Marsabit county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 4% with 1% mortality rate and a case fatality rate of 25%.
- Seasonal occurrence: Occurs all year round. In Marsabit county, mange is common throughout the year but most cases are seen when the animals are in poor nutritional state during the long dry season that occurs between the months of July and September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry (July- September)	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu Nakooyan	Mar Me Mederka

- **Diagnosis** Mange is easy to diagnose clinically from the pruritus, alopecia and encrusted plaques. Clinically affected animals are recognised by large areas devoid of hair, with thickened skin and folds affecting the hind limbs and neck. Chapter 1 describes the process of collecting a skin scrapping for field based confirmation of the parasite.
- **Differential diagnosis** other skin diseases that cause thickening of the skin include tick infestation, mineral deficiencies, recovered pox lesions and ringworm infections.
- Zoonotic potential Yes man can be an accidental host. Close contact with infected camels may result in the spread of disease to caretakers.
- Notifiable disease- No.
- **Treatment** Mange is treated effectively with acaricides like organochlorine (Amitraz®, Tix fix®), organophosphates like Malathion. These are applied as a spray, although hand application with a cloth is used for areas that are least accessible.

- Treatment should be repeated after an interval of 8-15 days.
- Ivermectin is a macrocyclic lactone is effective against mange two subcutaneous injections, 2 weeks apart combined with acaricide wash results in clinical resolution of signs.
- Supportive multivitamin therapy may be required at a dosage rate of 5 to 10 MLs for adult animals and 3 to 5 mL for calves via the subcutaneous or intramuscular route.
- Prevention- From the prophylactic standpoint, it would be desirable to treat the entire herd but this may be impracticable. Priority should be given to young camels and breeding males.

## Ethnoveterinary treatment practices

• Refer to tables 14 and 15

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals in the herd and create a separate night kraal/boma.
- Migrate out of the area or Relocate the camel boma
- Regular acaricide use or hand pick ticks.

#### Further Reading

- 1. Richard D. (1987). Camel mange. Rev. sci. tech. Off. int. Epiz., 1987, 6 (2), 475-477. https://www.WOAH.int/doc/ged/D8497.PDF
- Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate.edu/vdpam\_reports/21



## Figure 83

Mange skin lesions in a camel calf red arrows show alopecia, encrusted plaques and hyperkeratosis of the skin

### Picture source:

Yaeger, Michael, Coatney, John W, Dioli Maurizio and Plummer, Paul, "Camel Digital Necropsy Guide" (2016). Veterinary Diagnostic and Production Animal Medicine Reports. 21. https://lib.dr.iastate. edu/vdpam\_reports/21

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## 5.9 Dermatophytosis

## Other name: Ring worm

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Robbi	Robbi	Robo / Nabhar	Ngamunyani	Akiserit	Finy

## Definition and Epidemiology

- Camel dermatophytosis also known as ringworm is a highly contagious fungal dermatitis that characterised by circumscribed crusty hairless lesion, that are 1 to 2 cm in diameter that are distributed over the head, neck, shoulder, limbs and flanks. The fungal lesions are described as having a 'moth-eaten' appearance of the camel calf's wool.
- The disease is mostly found in young calves less than 3 years of age
- Trichophyton verrucosum is the most common fungi affecting young camels while older animals are affected by T. mentagrophytes.
- The morbidity rate in young calves is estimated to be 43.5% while the overall herd incidence rate of all age groups is 11.2%. In Marsabit county, livestock keepers in the year

2022 estimated that camel dermatophytosis has an annual apparent morbidity rate of 5%, mortality rate of 1% and case fatality rate of 20%.

• The disease is more prevalent during the rainy and cold seasons of the year when calves hurdle together due to the wet conditions. The persistent wet woolly skin of calves weakens the natural skin immunity barrier providing ideal conditions for infection. In Marsabit, camel ringworm infections occur throughout the year, however, more cases are reported during the long dry season as the calves are in nutritional stress as dams have less milk or they are nurses less due to the dry season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry (July- September)	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu Nakooyan	Mar Me Mederka

### Aetiology and Transmission

- The disease is caused by a specialised group of zoonotic fungi called dermatophytes.
  Dermatophytes are filamentous fungi which invade keratinized tissues of humans and animals, causing mild to severe, localized and/ or diffuse infections.
- Transmission of dermatophytes is usually by direct contact with infected host (animals or humans) or asymptomatic carriers and/or indirect contact with infected exfoliated skin

or hairs in the environment.

#### **Clinical signs:**

- The incubation period of the disease is 8-30 days. Skin lesions are extremely variable in distribution from multi focal to generalized lesions. The typical skin lesions are circular alopecic areas with peripheral expansion and that have the centre covered by grey powdery crust.
- The periphery spreading lesions may become
confluent and produce a moth- eaten appearance of the wool.

- Lesions are found in the head, neck, shoulders with extension to the flanks and legs.
- Generalised skin infections lead to emaciation.
- Mixed infection of dermatophytosis and Sarcoptic scabiei (Mange), other fungal infections like Microsporum gypseum and bacterial infection with Dermatophilus congolensis especially in rainy season have been reported.

#### Diagnosis

- Field based diagnosis can be done through Direct examination of skin scrapings digested with 20% potassium hydroxide (KOH) and examined under low and high power light microscope. Procedure for skin scraping is described in detail in chapter 1.
- Routine 'gold standard' culture of samples takes 5 weeks to grow on Sabouraud's dextrose agar (SDA). Molecular diagnostic tests can shorten the confirmatory diagnostic period.

#### Differential diagnosis

- Mange
- Other Dermatomycosis species
- Dermatophilosis due to Dermatophilus congolensis bacteria.

Zoonotic potential – Yes, Notifiable disease- No

#### Treatment

- Optimal therapy of dermatophytosis requires a combination of topical antifungal therapy,
- concurrent systemic antifungal therapy and environmental decontamination.
- In pastoralists setting systemic therapy and environmental decontamination may be impractical. The following treatment regime has been used; Wear gloves, Shave the affected woolly areas and remove scabs, Topical application of 2% solution of tincture iodine or 10% iodine solution once daily for three weeks (duration is difficult to adhere to in pastoralists settings). Systemic treatment can be done with Griseofulvin10 mg/kg body

weight for 7 days in mild infections; in severe cases 2 to 3 weeks (impractical in pastoralists settings)

 Dermatophytes can remain viable in infected soil for many years. Environmental decontamination can be done with 10% hypochlorite solution (Jik<sup>™</sup>).

#### Prevention

- Isolate infected animals from herd by ensuring they sleep in a different boma/kraal.
- Treat infected animals immediately with iodine based solutions.
- Regular ectoparasite control.
- Ensure good nutrition of calves.
- There is a vaccine commercially available. However, it is not marketed in Kenya. The vaccine is a live attenuated vaccine against T. verrucosum and T. mentagrophytes and is administered every five years.

#### Ethnoveterinary treatment practices

• Refer to tables 14 and 15

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals in the herd and create a separate night kraal/boma.
- Migrate out of the area or Relocate the camel boma
- Regular acaricide use or hand pick ticks.

#### Further Reading

 Abdulaziz M. Almuzaini, Salama A. Osman & Elhassan M.A. Saeed (2016) An outbreak of dermatophytosis in camels (Camelus dromedaríus) at Qassim Region, Central of Saudi Arabia, Journal of Applied Animal Research, 44:1, 126-129, DOI: 10.1080/09712119.2015.1021806: http://dx.doi.org/10.1080/097121 19.2015.1021806 (a)





### Figure 84

Cutaneous lesions(Top), Colonies on SDA (b) and Distorted hyphae with chlamydospores in chains of T. verrucosum (c) on microscopy.

#### Picture source:

Abdulaziz M. Almuzaini, Salama A. Osman & Elhassan M.A. Saeed (2016) An outbreak of dermatophytosis in camels (Camelus dromedaríus) at Qassim Region, Central of Saudi Arabia, Journal of Applied Animal Research, 44:1, 126-129. http://dx.doi.org/10.1080/09712119.2015. 1021806



### Figure 85

Skin scrapping KOH preparation observed under a light microscope X400 showing hair surrounded with chain of large ectothrix spores (Left) and Colony of T. verrucosum on SDA fungal media culture

#### Picture source:

Abdalla; SAJRM, 4(3): 1-10, 2019; Article no.SAJRM.50776. https://www.journalsajrm.com/index.php/SAJRM/article/download/30109/56501

## Camel Diseases: Non-specific Disease Cause

## 5.10 Wry neck syndrome

#### Other names: Kinked or Twisted neck, Bent neck, Torticollis, Cold struck

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Ch'ach'apsa	Ch'ach'apsa	Dhahasi	Lajingani	Lokonyo	Luut Gaa
					Nyurua

#### Case definition

- Wry-neck is a common syndrome of camels that occurs sporadically.
- The disease is characterised by a dramatic clinical sign characterized by an irreversible S-shaped twisting of the neck.
- The syndrome can affect camels of various age groups and occurs in all camel keeping countries pastoralists in East Africa: Kenya and Sudan

#### Aetiology:

- Unknown aetiology (idiopathic) but hypothesis from livestock keepers and animal health practioners is the syndrome is caused by consumption of poisonous plants or due to mineral deficiency and brucellosis infection.
- The condition has also been reported after trauma to the neck or head with consequent damage to the underlying nervous system, muscles or bones.

## Epidemiology, Transmission and Age group affected

- Syndrome occurs randomly.
- · Common in both old (Dromedary and

Bactrian) and new world (Guanaco, Alpaca, Llama and Vicuna) and in the domestic goat.

- Camelids are not the only animals affected by wry neck other "long necked" species like the Giraffe and goats can also be affected.
- Due to idiopathic aetiology there is no known route of transmission identified.
- All ages of animals are expected.

#### Clinical signs:

- Main clinical symptom of the affected camels is a permanent S-shaped bend of the neck
- The affected neck has mild to extreme decreased range of motion

#### Morbidity and mortality rate:

- In Marsabit county, livestock keepers in the year 2022 estimated an apparent annual morbidity rate of 7% with 6% mortality rate and a case fatality rate of 89%.
- Global estimate of wry neck occurrence is not available with most literature focusing on individual case documentation.

#### Seasonal occurrence:

· Based on livestock owners account, Wry

neck occurs any season of the year but most cases are reported during the long dry season that occurs between the months of July and September.

#### Further Reading

 Maurizio Dioli (2010). Clinical observations on the "wry-neck" syndrome or torticollis in the camel (Camelus dromedarius) population of Kenya and Sudan. The international camel symposium "linking camel science and development for sustainable livelihoods" from 7th to 10th June 2010 in Garissa, Kenya.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

#### Diagnosis

- Due to idiopathic nature of disease, clinical sign of the dramatic kinking of the neck is used as a definitive diagnosis of wry neck.
- Laboratory analysis of sick camels has not revealed a diagnostic reliable pattern.

#### Differential diagnosis

- Trauma of the neck muscles or bonesresulting in fractures or subluxation of cervical bones
- · Plant poisoning- Capparis tomentosa
- Osteoarthritis Brucella bacteria

#### Zoonotic potential – None recorded Notifiable disease- No Treatment and Prevention

- Wry neck has no effective treatment documented
- Livestock keepers and animal health service

#### Figure 86

Camel with a severe case of wry-neck with traditional cauterization at the side of the neck.

Picture source: Schwartz & Dioli 1992 providers have attempted to administer multivitamins (Vitamin B complex) with varying results but not proven efficacy.

• There is need for research to identify wry neck causative agent, so as to develop treatment and preventive strategies.

#### Ethnoveterinary treatment practices

• Refer to tables 14 and 15

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

• None mentioned as cause is unknown.



## Chapter 6: Sheep Diseases

#### Table 17: Local dialect name of priority sheep diseases in Marsabit county

Viral Sheep Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Peste Des Petits Ruminants (PPR)	Thadi Ree	Thadi Ree	Siim / Ilmokhor	Lodua / Nagumoo E Ntare	Lomaa / Lonoo/ Lokiyo	Mokodo / Lokotokonyan
Sheep Pox	Kurtubale	Kurtubale	Nyinyilo	Nariri / Tune	Etune,/ Tune	Verte
Foot And Mouth Disease (FMD)	Oyale	Oyale	Lkulub	Lkulup	Lojaa / Lokituk	Huluf / Guluf
Progressive Interstitial Pneumonia (PIP)	Qufaa Ola	Qufaa Ola / Suutes	Chur	Chur	Lowala / Lotai	Lejuo
Contagious Ecthyma (Orf)	Umburur	Abdhara	Afturo	Apturo	Ngiboruok	Dicho
Bacterial Sheep Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Enterotoxaemia	Dukub Tiru / Qurtam / Marchaqas	Dukub Tiru	Lbus	Lbus / Nadomanyita	Lobus	Suony
Foot Rot	Bargao	Bargao	Dokoho	Ngojini	Ebaibai	Aiy Komfo / Konof
Parasitic Sheep diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Mange	D'oq	D'oq	Haddo	Lpepedo	Ekoikoi / Pepedo	Gogosich
Helminthiasis	Minii	Minni	Deyah	Ntumwa	Ngipelei	Chade
Coenurosis	Sirgo	Sirgo	Sirgo	Sirgo	Nkerep	Sigire
Protozoal Sheep Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Babesiosis	Birte	Bira	Kati Ti Digee	Nkula	Lodip	Sin Bura

#### Table 18: Plants identified by livestock keepers as having medicinal value in treating sheep diseases in Marsabit county

Medicinal plants used	Target Sheep diseases
Acacia Nubica	PPR, Sheep pox, PIP
Euphorbia species	PPR, Sheep pox, PIP
Zanthoxylum chalybeum (knob wood)	FMD, PPR, PIP
Ficus sur (Bush fig)	PIP
Salvadora passica	PPR, Sheep pox

NB: Refer to table 8 above for details on most commonly used plant parts in the preparation of remedies

#### Table 19: Traditional practices mentioned by livestock keepers as effective in treating sheep diseases in Marsabit county

Traditional Practices	Target Sheep diseases
Salty water from saline wells, springs or Lake Turkana- Topical application	PPR, Sheep pox, Mange, Haemonchosis, Babesiosis
Old engine oil- Topical application	Sheep pox, Mange
Bleeding animal- Cut the base of the ears or the bridge of the nose near the eyes	Enterotoxaemia
Chewing tobacco ground to powder. Powder given as an oral drench or animal is forced to inhale it. The tobacco can also be chewed and spat into the animal's mouth	Enterotoxaemia, PPR, Babesiosis. PIP
Hot Iron or stone cauterization of affected body region	Enterotoxaemia, Sheep pox, FMD
Cooking oil	FMD
Honey	FMD
Local alcohol brew (Changaa)	Enterotoxaemia
Culling	PIP, Coenurosis
Pepper powder	PPR, Haemonchosis, Sheep pox, PIP, Babesiosis

NB: Text in red highlights practices that go against animal welfare principles

Type of VMP	Active ingredient	Purpose	Target sheep diseases						
Antibiotic	Oxytetracycline	Curative and preventive for secondary bacterial infections	PPR, Sheep pox, Babesiosis, PIP, FMD, Ovine Mycoplasma Pneumonia, Foot rot, Anaplasmosis						
Antibiotic	Penstrep® procaine penicillin G and dihydrostreptomycin	Curative and preventive for secondary bacterial infections	Sheep pox, PIP, Foot rot, FMD						
Anthelmintic	Albendazole / Levamisole	Curative	PPR, Haemonchosis, Mange						
Anthelmintic	Ivermectin	Curative	Haemonchosis, <mark>Sheep pox</mark> , Mange						
Antibiotic	Diseptoprim ® boluses Sulphadiazine and Trimethoprim	Curative	PPR						
Metabolic support	Multivitamin	Supportive therapy to boost immunity	PPR, Haemonchosis, Mange						
NB: The drug of c	NR: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy								

Table 19: Traditional practices mentioned by livestock keepers as effective in treating sheep diseases in Marsabit county

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

## Sheep Diseases: Bacterial Sheep Diseases

## 6.1 Enterotoxaemia

Other names; Over eating or Pulpy Kidney disease

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dukub Tiru,	Dukub Tiru	Lbus	Lbus /	Lobus	Suony
Qurtam,			Nadomanyita		
Marchaqas					

#### **Case Definition:**

- An acute disease of sheep and goats that is characterised by sudden and high death rates in lambs and kids and at post mortem haemorrhagic enteritis where sections of the small and large intestinal walls have a deep blue-purple appearance and intestinal contents are watery and bloody. The kidneys on palpation have abnormally soft consistency described as being pulpy.
- Enterotoxaemia as the name suggests is a blood poisoning due to a toxin (toxaemia) produced by an intestinal dwelling bacteria.

#### Aetiology and Pathophysiology

- Enterotoxaemia is caused by a gram-positive, rod-shaped bacteria called Clostridium perfringens type D.
- The bacteria are normally found in the soil and as part of the normal microflora of the gastrointestinal tract of healthy sheep and goats.
- However, under specific predisposing conditions, the bacteria rapidly reproduce in the animal's intestine, producing large quantities of the epsilon toxin.

• Young animals are more susceptible to the toxin when compared to adult animals that acquire a toxin tolerance with age following low dose exposure over time.

#### Predisposing factors and Epidemiology

- Overgrowth of Clostridium perfringens type D bacteria occur in the following conditions:
- Excessive consumption of milk, consumption of lush green pasture at the beginning of the rainy season and feeding on high grain concentrates.
- Reduced Immunity due to disease or heavy gastrointestinal parasitism such as nematodes, cestodes (tapeworms) and coccidia. Heavy tape worm infestation causes a reduction in the motility of the intestinal tract.
- Disease is distributed worldwide with an estimated morbidity rate of 2%, mortality rate of 3% and case fatality rate of 30%. Disease incidence reports of unvaccinated sheep and goats in India have found a prevalence rate 56% in lambs, 46% in kids and 4% in adult sheep.
- In Marsabit county, livestock keepers in the year 2022 estimated an apparent morbidity

and mortality rate of 3% and case fatality rate of 100%. The livestock keepers indicated the disease was common during the long rainy season between the months of April to June.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

#### **Clinical signs**

- Sudden death is the main manifestation of the per acute form of the disease in young lambs although some of the animals may be dull, depressed and anorexic.
- In acute cases, there is frothy salivation, green or pasty diarrhoea, staggering, recumbency, opisthotonus, colonic convulsions coma and death.
- Colic and bloat may occur. Affected adult sheep often lag behind the rest of the flock and show nervous signs. Champing of the jaws, blindness, salivation, rapid and shallow respiration, atonic rumen and pasty faces may also be evident.

#### Post-mortem lesions

- Haemorrhagic enteritis with ulceration of the mucosa.
- Fluid-filled pericardial sac in young lambs and in older animals, haemorrhagic areas on the myocardium.
- Petechiae and ecchymosis haemorrhages of the abdominal muscles and serosa of the intestine.
- Bilateral pulmonary oedema and congestion is also common.
- Rumen and abomasum contains feed. There is also undigested feed in the Ileum.
- Rapid post-mortem autolysis of the kidneys in adult sheep has led to the popular term pulpy kidney disease. Pulpy kidney is not a common feature in young lambs and is rarely found in goats and cattle. In goats the disease can also cause haemorrhagic or necrotic enterocolitis.

released to graze on lush pastures.

Diagnosis	Treatment	Control
Field based diagnosis can be made	Treatment of the animals is	The most effective control method
through making Giemsa stained	usually ineffective, due to the	is annual vaccination.
smears of intestinal contents	rapid progression of the disease in	KEVEVAPI does not have the
that can be examined for large	the animal.	vaccine source from private
numbers of gram-positive, rod-	Attempts can be made to treat	sources.
shaped bacteria	animals that are not too sick with;	Ethnoveterinary practice
	• Clostridium perfringens C & D	Restrict lambs from suckling all
Confirmatory diagnosis made	antitoxin (5 mL subcutaneously)	the time by ensuring the lambs are
through ELISA and PCR analysis	• Antibiotics, especially penicillin	separated from ewes most of the
of faecal and intestinal contents	<ul> <li>Anti-bloating medication</li> </ul>	day.
	Intramuscular thiamine	Restrict access to large
Differential Diagnosis	(vitamin B1) to prevent or treat	quantities of lush pasture by
<ul> <li>Plant poisoning</li> </ul>	the encephalomalacia	delaying the time the animals are
• Bloat	Ethnoveterinary practice	released in the morning or feeding
Thiamine deficiency	• Refer to tables 18 and 19	the animals on hay before being

#### References

1. MSD Veterinary Manual https://www.msdvetmanual.com/

2. Mohammad, Tooloei & Masoudi, Mohammadhossein. (2008). The Distribution and Prevalence Rate of Enterotoxemia in Sheep in East Azerbaijan Province, Northwestern Iran, in Spring 2008. Journal of Animal and Veterinary Advances. 7.

3. Hussain, R., Guangbin, Z., Abbas, R. Z., Siddique, A. B., Mohiuddin, M., Khan, I., Rehman, T. U., & Khan, A. (2022). Clostridium perfringens Types A and D Involved in Peracute Deaths in Goats Kept in Cholistan Ecosystem During Winter Season. Frontiers in veterinary science, 9, 849856. https://doi.org/10.3389/ fvets.2022.849856

lack spores.



Figure 87 Small intestine distended with gas (white arrow) and congested in a sheep with enterotoxaemia.

Picture source: Hassanein et al., 2017



#### Figure 88 C. perfringens, Gram-stained smear from intestinal epithelial surface using high power objective to visualise the intensely gram-positive large rods that

## 6.2 Foot rot

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Bargao	Bargao	Dokoho	Ngojini	Ebaibai	Aiy Komfo / Konof

#### Case definition and Aetiology

- Foot rot is a contagious bacterial hoof infection that causes severe lameness in sheep and goats.
- The bacteria are normally found in the soil and are easily carried onto a farm by infected animals or the soles of the shoes. Two types of soil borne bacteria cause the disease -Dichelobacter nodosus (formerly Bacteroides nodosus, a Gram-negative, obligate anaerobe) and Fusobacterium necrophorum).
- Both thrive in moist soil conditions and are difficult to control or eliminate once they contaminate the soil in farms where sheep and goats are kept.
- In Marsabit county, livestock keepers in the year 2022 estimated an apparent morbidity rate of 3% with no mortality rate.
- The disease is most common during the long rainy season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

#### Clinical signs and Field based diagnosis

- Foot rot is an inflammation between the toes that usually affects one foot.
- Foot rot can occur in one or more feet, causing severe lameness. Typically, animals are seen grazing on their knees. It occurs when both bacteria cause a dual infection of the tissues of the foot.
- The interdigital space of the foot has dark exudate and the discharge is foul smelling.
- If not treated early, the bacterial toxins break down the hoof wall and sole of the foot, resulting in the hoof wall loosening and detaching from the foot.
- Predisposing factors to foot rot infection include; overgrown, cracked or damaged hooves; poor diets deficient in certain minerals also predispose animals to poor hoof health.

#### Treatment

- Systemic treatment with antibiotics with or without trimming of the hoof is effective. One injection of 20% long acting oxytetracycline at 1ml per 10kgs deep intramuscular is effective. One can also administer an analgesic like phenylbutazone.
- Trimming of the claws is recommended to remove excess tissue that provides a place for the bacteria to thrive.
- After feet have been trimmed, affected animals should stand for at least 5 minutes with all feet in a medicated foot bath (10% copper or zinc sulphate) and feet should be dry before going back to pasture.
- Foot dipping should be repeated once a week for four weeks and non-responding animals should be culled out of the herd.

#### Ethno veterinary treatment and control

- Wash affected leg interdigital space with salty water
- · Ensure animals night boma area drains well especially during the rainy season.

### Control

- Do not purchase lame animals. Thoroughly inspect feet before purchase. Observe herd of origin for lameness of other animals.
- Trim hooves regularly.
- · Provide good drainage to all areas in pastures and paddocks where water tends to pool, or fence these areas off. This is where the bacteria often collect.

- Keep goat houses or bomas dry and clean.
- · Ensure regular hoof care management and always check feet of limping goats.

#### **Differential diagnosis**

- · Foot and mouth disease
- · Tick infestation of the lower limb (dew claw area or interdigital space)
- Foot scald
- · Foot trauma- thorns pricking interdigital space
- Foot abscess with other bacteria



## Figure 89

Common hoof conditions in goats and sheep



#### Picture source:

https://www.canr. msu.edu/news/ tactics-to-win-thebattle-against-foot-







Figure 91 Comparing hoof that is overdue for trimming and one that is properly trimmed

The picture shows an overdue hoof that has the hoof walls curling inwards and is long at the toe. The in curling hoof wall traps mud, manure thus predisposing animal to foot rot. A properly trimmed hoof is flat at the bottom and has a box shaped hoof shape where both toes are the same length.



Hoof trimming procedure in sheep and goats

## Sheep Diseases: Viral Sheep Diseases

## 6.3 Peste des Petits Ruminants (PPR)

Other name: Sheep and goat plague

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Thadi Ree	Thadi Ree	Siim / Ilmokhor	Lodua, Nagumoo	Lomaa /Lonoo,	Mokodo /
			E Ntare	Lokiyo	Lokotokonyan

#### Case definition

• PPR is a highly contagious disease affecting domestic and wild small ruminants. It is characterised by multisystemic clinical signs that include bilateral nasal and ocular discharge, pneumoniae, necrotic stomatitis and diarrhoea.

#### Aetiology and Pathophysiology

- PPR is caused by a virus of the family Paramyxoviridae, genus Morbillivirus.
- In 2016 the virus name was changed from Peste des Petits Ruminants virus (PPRV) to Small ruminant morbillivirus (SRM). However, PPRV is still commonly used by people working in the field.
- The virus through nucleic acid sequencing is differentiated into a single serotype that has four lineages (1–4).
- The virus is antigenically similar to rinderpest virus, measles virus and canine distemper virus.
- Virus is susceptible and is inactivated by alcohol, ether and common detergents and disinfectants like phenol and 2% Sodium hydroxide (Jik). The virus can survive for long periods in chilled and frozen tissues.
- · PPR virus enters the body through the

respiratory tract and spreads throughout the body via both the lymphatic and vascular systems. As the PPR virus is both lymphoand epithelio-tropic, infection usually results in conjunctivitis, rhinotracheitis, ulcerative stomatitis, gastro-enteritis and pneumonia.

#### **Epidemiology and Transmission**

- PPR was first described in 1942 in Côte d'Ivoire, West Africa. Since then the disease has spread to the rest of Africa, the Middle East, Asia and Europe.
- Based on the World organisation for Animal Health (WOAH) reports, the disease is found in more than 70 countries globally which are home to 1.7 billion heads or 80 % of the global population of sheep and goats.
- WOAH estimates that PPR when introduced into a new area can cause a high morbidity mortality rate of 90 and 70% respectively. In endemic areas, morbidity rates are lower at 30% while mortality rates can be as high as 50% in young lambs and kids that are under 1-year-old.
- Sheep and goats that recover from PPR develop an active immunity and have antibodies that last for more than 4 years. Studies indicate that PPR immunity may be

life-long.

- Marsabit county livestock keepers in the year 2022 estimated that PPR had an apparent annual morbidity rate of 20%, mortality rate of 14% and case fatality rate of 65%.
- PPR virus is found in tears, nasal discharge, coughed secretions and milk. Transmission is mainly through direct contact with aerosol droplets between animals living in close proximity and indirect transmission through contaminated fomites such as feed, pasture and water troughs.
- There is no disease carrier state.
- PPR outbreaks are common during the rainy season or the dry cold season that coincides with increased local livestock movement or trade.
- PPR is endemic in Marsabit county with cases reported all year round. However, explosive outbreaks occur during the long dry season between the months of July and September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

#### **Clinical signs**

- Incubation period is between 3 and 6 days. WOAH has set the period for trade purposes to be 21 days.
- PPR clinical severity depends on PPRV lineage, age (young have a more severe disease), host species (severe in goats) and breed (indigenous breeds less susceptible) and the health status (nutrition level and parasitism).
- Infected animals present clinical signs similar to those that were once seen with rinderpest (RP) in cattle. However, in PPR there are two differing signs, development of crusting scabs along the lips and pneumonia in later stages of the disease.
- PPR has various clinical forms described in literature. Peracute form that is frequent in goats from immuno-naïve herd. Animals develop a high fever (40-41°C) are depressed and die in 1 or 2 days. This form has high mortality.
- Acute form is characterised by fever (40–41°C) for 3–5 days, depression, anorexia, serous ocular and nasal discharge that becomes mucopurulent and profuse. The discharges crusts over and occludes the nostrils and eyes causing respiratory distress. The mucopurulent discharge may persist for up to 14 days. Within 4 days of onset of fever, the gums become hyperaemic, and erosive

lesions develop in the oral cavity with excessive salivation. Severe, watery, bloodstained diarrhoea is common in later stages that also occurs with bronchopneumonia that is evidenced by coughing and abdominal breathing. Abortions may also occur. Dehydration, emaciation, hypothermia and death may occur within 5–10 days. Survivors undergo long convalescence.

 Sub-acute form- common in endemic areas animals develop fever and serous ocular and nasal discharge, diarrhoea and if severe the animal may develop dehydration and prostration. Mortality is low.

#### Post-mortem lesions

- Erosive stomatitis of lower lips, gum, free portion of the tongue, hard palate, pharynx and upper third of the oesophagus in severe cases. In Marsabit, livestock keepers described the oral lesions as those seen when one consumes a hot liquid like tea.
- The rumen, reticulum and omasum rarely have lesions most lesions are found in the small and large intestine.
- There are streaks of haemorrhages and sometimes erosions: in the first portion of the duodenum and the terminal ileum. Severe cases have necrotic or haemorrhagic enteritis with ulceration of Peyer's patches.

• Linear haemorrhages of the lining of the cecum and colon give a stripe appearance called "zebra striping" of the large intestine.

#### PPR eradication

WOAH and FAO, in their joint strategy for control and eradication of PPR, have set the goal of eradicating the disease by 2030. The strategy has three components,
(i) the technical step-wise approach (Stage 1 to Stage 4) to control and eradicate the disease (ii) the strengthening of Veterinary Services in order to be able to carry out the technical component and (iii) the control of other priority small ruminant diseases like CCPP and SGP together with PPR in view of increasing the impact of the control efforts.

#### Diagnosis

• Field based diagnosis- PPR rapid diagnostic test using ocular conjunctival or nasal swabs.

#### **Confirmatory diagnosis**

• Specimens to be collected include whole blood and tissue samples such as lymph node (mesenteric and bronchial), spleen, and lung for antigen isolation and identification using PCR and ELISA for antibody analysis. ELISA does not differentiate antibodies due to vaccination or disease.

#### Differential diagnosis

- Contagious caprine pleuropneumonia
- Coccidiosis
- Pasteurellosis (may occur as secondary

infection to PPR)

- Contagious ecthyma
- Foot and mouth disease

#### Treatment

• Viral diseases have no treatment. Treatment is mainly to prevent adverse effects of secondary bacterial infection with the main bacteria involved being Pasteurella. Broad spectrum antibiotics like long acting Oxytetracycline, Penicillin streptomycin and Sulphonamides may be used at the recommended dosage given by the manufacturer.

#### Control

- Vaccination is the most effective method of controlling PPR. Isolation, quarantine and movement control are used to limit its spread during an outbreak.
- KEVEVAPI has a vaccine called PESTEVAX<sup>™</sup>

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals
- Migrate out of outbreak area or avoid areas with active PPR outbreak
- Avoid sharing of pasture of watering points with sick herds
- Report outbreak to CDR, ward administrator or government vet officer
- Present animals for vaccination

#### Zoonotic potential - None

#### References

1. BARON, J., FISHBOURNE, E., COUACY-HYMAN, E., ABUBAKA, M., JONES, B.A., FROST, L., HERBERT, R., CHIBSSA, T.R., VAN'T FLOOSTER, G., AFZAL, M., AYEBAZIBWE, C., TOYE, P., BASHIRUDDIN, J.M BARON, M.D. (2014). Development and Testing of a Field Diagnostic Assay for Peste des Petits Ruminants Virus. Transboundary and Emerging Diseases 61, 390 – 396.

2. FAO and OIE (2016) – Global Strategy for the Control and Eradication of PPR https://www.oie.int/app/uploads/2021/03/en-gep-ppr-finalweb.pdf

3. OIE (2020). Technical Disease Card https://www.oie.int/fileadmin/Home/eng/Animal\_Health\_in\_the\_ World/docs/pdf/Disease\_cards/PESTE\_DES\_PETITS\_RUMINANTS.pdf



## Figure 93

Buccal lesions, including vesicular to erosive lesions in an 8 month lamb (Left) and mouth lesions showing areas of dead cells in a goat (Right) Lesions commonly seen during the early stages of the diseases

#### Picture source:

Mohebbi et al (2018). Report of an Outbreak of Peste Des Petits Ruminants in Iran, Clinical, Epidemiological and Pathological Study. Recognizing PPR: A field manual http://www.fao. org/3/x1703e/x1703e00. htm



### Figure 94

Nodular lesions around the mouth are a common finding in goats in the later stages of PPR infection

> Picture source: Recognizing PPR: A field manual http://www.fao.org/3/x1703e/ x1703e00.htm



## Figure 95

"Zebra striping" in the large intestine due to linear haemorrhage along the tips of the folds of the lining of the caecum and colon.

#### Picture source:

Recognizing PPR: A field manual http://www.fao.org/3/x1703e/ x1703e00.htm



RAPID:       Results within 20 minutes         EASY:       Requires no laboratory equipment         SIMPLE:       Only requires swab sample from eyes or nose of animal         DETECTS:       All four known genetic lineages of PPRV         VALIDATED:       With experimental and field samples
EASY:       Requires no laboratory equipment         SIMPLE:       Only requires swab sample from eyes or nose of animal         DETECTS:       All four known genetic lineages of PPRV         VALIDATED:       With experimental and field samples
SIMPLE:         Only requires swab sample from eyes or nose of animal           DETECTS:         All four known genetic lineages of PPRV           VALIDATED:         With experimental and field samples
DETECTS: All four known genetic lineages of PPRV VALIDATED: With experimental and field samples
VALIDATED: With experimental and field samples
PROPERTY AND ADDRESS OF THE PARTY OF THE PAR
SENSITIVE: 84% (relative to PCR) and can detect before clinical signs
SPECIFIC: 95% (100% with ocular sample)

## 6.4 Sheep and Goat pox (SGP)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Kurtubale	Kurtubale	Nyinyilo	Nariri /Tune	Etune/ Tune	Verte

#### Case definition

• Sheep pox and goat pox are viral diseases of sheep and goats characterised by fever and sudden appearance of generalised skin nodules that extend into internal organs (particularly the lungs) and death.

#### Aetiology, Epidemiology and Transmission

- Sheep pox virus (SPPV) and goat pox virus (GTPV) are the causative agents of SGP and they belong together with lumpy skin disease virus to the genus Capripox virus from the family Poxviridae.
- SGP is endemic in Africa, the Middle East, Asia and Southern Europe countries of Bulgaria, Kazakhstan, Mongolia, Greece and Russia.
- Based on WOAH reports, SGP in endemic areas has a morbidity rate of morbidity rate:

of 70 to 90% and mortality rate of 5 to 10%. In immune naïve herds with exotic breeds, mortality can reach 100%.

- All breeds of domestic and wild sheep and goats are susceptible to infection.
- Direct transmission occurs through the aerosol route while indirect transmission occurs through contact with contaminated fomites or through mechanical routes via bites of insects or iatrogenic through hypodermic injection.
- Infection results in solid and enduring immunity. In Marsabit county, livestock keepers estimated an apparent annual morbidity rate of 8%, mortality rate of 3% and case fatality rate of 37%.
- SGP is endemic in the county with most outbreaks occurring during the long dry season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry (July- September)	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu Nakooyan	Mar Me Mederka

#### Clinical signs

- Incubation period is between 8 and 13 days
- Fever, dullness and Inappetance rarely anorexia
- There is development of rhinitis and conjunctivitis that manifests as increased mucopurulent nasal and ocular discharge.
- There is enlargement of all the superficial lymph nodes, in particular the prescapular lymph nodes.
- Breathing becomes laboured and noisy due to pressure on the upper respiratory tract from

the swollen retropharyngeal lymph nodes and developing lung lesions.

- Development of widespread nodular skin lesions that are readily seen on the muzzle, ears and areas free of wool or long hair. Palpation of animal detects skin lesions not readily seen.
- If affected animal survives the acute phase of the disease, the skin lesions become necrotic and form scabs that persist for up to 6 weeks, leaving small scars. The skin lesions are susceptible to fly strike.

#### Post mortem lesions

- Skin, oral and respiratory mucous membranes and organs have the characteristic nodules. Nodules are seen on the surface of the lungs, kidney, liver and testicles.
- All superficial and internal lymph nodes are enlarged.

#### Diagnosis

- Field based diagnosis- clinical signs can be used as tentative diagnosis
- Confirmatory- biopsy skin lesions and whole blood in live animals and lymphnodes and affected tissue samples like lungs for dead animals. ELISA for antibody detection should be done within the first week after clinical signs begin. After that PCR diagnosis to detect viral genome.

#### Treatment

• Viral diseases have no treatment. Treatment is mainly to prevent adverse effects of secondary bacterial infection with the main bacteria involved being Pasteurella. Broad spectrum antibiotics like long acting Oxytetracycline, Penicillin streptomycin and Sulphonamides may be used at the recommended dosage given by the manufacturer.

#### Control

- Quarantine and movement control during outbreaks
- Isolate sick animals
- Vaccination is the most effective control strategy. Live, attenuated virus vaccines induce longer immunity than inactivated virus vaccines. KEVEVAPI has a SGP vaccine called S&G Vax<sup>™</sup>.

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals
- Present animals for vaccination
- Report outbreak to authorities

#### References

1. MSD Veterinary Manual https://www. msdvetmanual.com/

2. OIE (2020). Technical Disease Card https://www. oie.int/en/what-we-do/animal-health-and-welfare/ animal-diseases/technical-disease-cards/#searchformheader



Figure 97 Lung lesions in sheep and goat pox

Picture source: Flock&herd at flockandherd.net.au

Figure 98 sheep pox skin lesions

Picture source: Hurisa et al., 2018



Figure 99 SGP lesions in a goat kid and adult in Marsabit county

Picture source: Diba Denge



Figure 100 Early SGP skin lesions in a goat in Marsabit county

Picture source: Elema Bule, Illeret ward, Marsabit County

## 6.5 Foot and mouth disease (FMD)

Refer to chapter 4: Cattle diseases, section 4.1 Foot and Mouth Disease (FMD)

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals and avoid areas with outbreaks
- Present animals for vaccination
- Do not buy animals from the markets during an outbreak (especially goats as they do not show clinical signs but are carriers of disease). Disease in cattle is more severe when compared to the disease in sheep. Goats are more resistant to FMDV as they show no or less severe clinical signs.

## 6.6 Contagious ecthyma (Orf)

Other names- scabby mouth, contagious pustular dermatitis, sore mouth

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Umburur	Abdhara	Afturo	Apturo	Ngiboruok	Dicho

#### Case definition

- Contagious viral skin and mucosa disease which causes scabby lesions usually around the area of the mouth, lips and nose. Orf virus infects mainly sheep and goats (goats have a more severe disease).
- Orf also occurs in camelids, deer, reindeer, dogs, cats and squirrels.
- Orf is very contagious, it is spread through direct animal-to-animal contact entering through damaged skin. It is often self-limiting but is zoonotic, although is considered more of an occupation hazard for people working with livestock.

#### Aetiology

- Caused by a virus from the parapoxvirus genus in the Poxvirus family.
- Humans infected with orf virus develop ulcerative lesions or nodules on their hands.
- In Marsabit county, livestock keepers estimated an apparent morbidity rate is 9%, mortality rate 6% and case fatality rate of 67%. Death occurs mainly in goat kids that do not receive traditional or conventional treatment.
- Orf in Marsabit county is common during the long rainy season that occurs from the month of April to June.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

#### Clinical signs

- Mouth lesions are typically found on the lips, muzzle, and in the mouth.
- Early in the infection, lesions appear as blisters that develop into crusty scabs.
- Sheep and goats may get sores on their lower legs and teats, especially when ewes or does are nursing infected lambs or kids. Young animals may have difficulty nursing and may require bottle or tube feeding. Nursing ewes or does with lesions on their udders may abandon their lambs, and older animals with oral lesions may also require nutritional support.
- Except in rare cases, animals recover completely from sore mouth infections within a month. Particular breeds, especially Boer

goats, may be especially susceptible and may have severe infections.

• Animals can become infected more than once in their lifetime but repeat infections usually occur after a year's time and are generally less severe.

#### Diagnosis

- Field based diagnosis using clinical signs
- Mouth lesions and whole blood can be submitted for ELISA and PCR analysis

#### Differential diagnosis

• PPR- in goats especially due to nodular/ scab like lesions around the lip commissures (junction). However, in PPR the animals also have bilateral nasal and ocular mucopurulent discharge and diarrhoea and are very sick.

- Dermatophilosis- especially in sheep, lesions appear as discrete "bottle-brush" / "paint brush or lumpy skin lesions on muzzle and margins of the ears.
- Warts- discrete growth that have a narrow base attachment (neck) to the skin and are not contagious. Only 1 or 2 animals gets warts in a flock.

#### Treatment

- Treatment of individually infected animals is not necessary unless lesions are severe. In which case topical disinfecting with iodine based products or application of milking salve on mouth and udder lesions can be used. Oxytetracycline Gentian Violet spray has also been used to prevent fly struck, control secondary bacterial infection and dry scabs.
- Severely affected kids require good nursing care to ensure that they are eating and drinking and can be an antibiotic combination of 200mg/kg BWT procaine penicillin and 250mg/kg BWT dihydrostreptomycin sulphate once a day for 3 days. A petroleum based soft emollient cream can also be applied once a day until lesions resolve. Does and ewes may require one treatment of long acting antibiotic either oxytetracycline or amoxicillin based if they develop mastitis. Oxytetracylcine is not indicated for young animals as the molecule is deposited in bones and teeth (binds to calcium) causing discolouration of teeth and delays bone growth.

#### Prevention

- Isolate newly purchased animals.
- Ensure young kids drink colostrum.
- KEVEVAPI has a vaccine called ORFVAX. The vaccine is applied to scarified skin on the inside of the thigh. This is done by making 3 to 4 short superficial scratches about ½" apart with a sterile hypodermic needle. These scratches should break the skin but should not cause bleeding. The vaccine is applied using a small piece of cotton wool moistened with the reconstituted vaccine. A mild local reaction occurs within one week and immunity develops within 14 days. Annual revaccination is recommended.

Zoonosis potential- Yes, wear gloves when treating sick animals

#### Notifiable disease - No

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Prompt treatment of cases using traditional or conventional methods so as to avoid the disease spreading to other animals
- Prevent animals from accessing thorny browse during the rainy season.

#### References

 Hosamani, Madhusudan & Scagliarini, Alessandra & Veerakyathappa, Bhanuprakash & Mcinnes, Colin J & Singh, Raj. (2009). Orf: An update on current research and future perspectives. http://dx.doi.org/10.1586/eri.09.64
 MSD Veterinary Manual https://www. msdvetmanual.com/







Figure 102 Contagious ecthyma mouth lesions in goats



Figure 101 shows orf infection in goats. Clinical picture of orf on the oral commissures of an adult goat (A), which recovered completely after 4-5 weeks (B) after first appearance of the clinical lesions. Proliferative Orf lesions on the gums and tongue in a 1-week-old kid in a small goat flock owned by a farmer in Bidar, India (C). Lesions have covered the entire anterior and lateral portion of the tongue (T), while lesions on the lower jaw (J), particularly on the gum (G) portion, have covered the entire lower incisors. Two kids with oral lesions died 3-4 days after showing clinical signs.

#### Picture source:

Hosamani, Madhusudan & Scagliarini, Alessandra & Veerakyathappa, Bhanuprakash & Mcinnes, Colin J & Singh, Raj. (2009). Orf: An update on current research and future perspectives. http://dx.doi.org/10.1586/eri.09.64

# 6.7 Progressive Interstitial Pneumoniae (PIP)/Chronic Interstitial Pneumonia (CIP)

Other names: Maedi, Maedi-Visna, Zwoegersiekte, La bouhite, Graaff-Reinet disease

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qufaa Ola	Qufaa Ola /	Chur	Chur	Lowala / Lotai	Lejuo
	Suutes				

#### Case definition

- A chronic viral disease in sheep that clinically manifests as progressive interstitial pneumonia characterised by dyspnoea (Maedi) and wasting (Visna). Visna is the neurologic form of the disease in sheep, resulting initially in unilateral pelvic paresis that progresses to hind limb paralysis. In goats, a similar chronic disease called Caprine Arthritis Encephalitis (CAE) affects the nervous system and joints resulting in progressive arthritis and neurological signs.
- In both CIP and CAE clinical disease is not treatable and animals have to be culled for lack of production or animal welfare reasons.

#### Aetiology and Epidemiology

- Caprine arthritis-encephalitis (CAE) and Maedi-Visna (MV) are persistent lentivirus infections of goats and sheep. The viruses are collectively called the Small Ruminant Lentiviruses (SRLVs).
- Reported seroprevalence for (CIP/PIP) in sheep varies widely from 1 to 50%. In the Western parts of North America, a seroprevalence of 49% has been reported, in north Atlantic region a prevalence of 9% has been documented and in Laikipia Kenya a prevalence of 29% is reported.
- CAE has a worldwide distribution with most studies reporting a high prevalence level of above 70%.
- In Marsabit county, livestock keepers in 2022

estimated a CIP apparent morbidity and mortality rate of 8% and 100% case fatality rate. The disease has no seasonal occurrence it is seen throughout the year and affects in a year 2 to 3 animals in a flock.

- The causal lentivirus, persists in lymphocytes, monocytes, and macrophages of infected sheep and goats in the presence of a humoral and cell-mediated immune responses.
- Seropositive sheep and goats must be considered infected and capable of transmitting the virus. Transmission occurs via the oral route, usually by ingestion of colostrum or milk that contains virus particles, or by inhalation of infected aerosol droplets. Intrauterine infection may also occur.
- Direct contact with blood or other body secretions, which might contain free viral particles and infected macrophages or epithelial cells may also occur

#### **Clinical signs**

 CIP/PIP clinical signs rarely occur in sheep under 2-years-old and are most common in sheep over 4 years old. The disease progresses slowly, with wasting and increasing respiratory distress as the main signs. Coughing, bronchial exudate, depression, and fever are seldom evident unless secondary bacterial infection occurs. A non- inflammatory, indurative mastitis (udder becomes hard and firm) may occur. The encephalitic form of visna causes head tilt and circling, whereas the spinal form causes unilateral pelvic limb proprioceptive deficits progressing to paresis and eventually to complete paralysis.

- Caprine arthritis encephalitis (CAE) is a multisystemic contagious viral disease of goats. The disease is typically spread from mother to kid through the ingestion of colostrum or milk. CAE virus may also be spread among adult goats through contact with body secretions including blood and faeces of infected goats.
- There are 5 major forms of CAE in goats: arthritis, encephalitis (inflammation of the brain), pneumonia, mastitis, and chronic wasting. The arthritic form of the disease is most common in adult goats, while the encephalitic form is common in kids. The chronic wasting form of the disease can occur either separately or in addition to any other form of CAE.
- Arthritic CAE signs in adults include-Lameness (may be sudden), stiffness, reluctance to walk, abnormal posture, reluctance to rise, weight loss, swollen joints and walking on knees
- Encephalitic CAE seen mostly in kids 1 to 6 months old signs include- Incoordination, inappropriate placement of limbs, progressive paralysis, depression, blindness, head tilt, seizures and death.
- Pneumonic CAE is common in adults, signs include- deep, chronic cough, difficulty breathing and weight loss.
- Mastitic CAE signs include- hard, swollen udder(s), decreased milk production
- In addition, CAE virus may also cause a chronic wasting disease in which goats continue to lose weight although appetite is unaffected.

#### Post-mortem lesions

 Macroscopic lesions of CIP/PIP are confined to the lungs and associated lymph nodes. The lungs do not collapse when the thorax (with obvious rib indentations) is opened and are abnormally firm and heavy (~2 kg; 2-4 times normal weight). Early lung changes may be difficult to detect, but later in the disease, lungs are mottled by gray and brown areas of consolidation. The mediastinal and tracheobronchial lymph nodes are greatly enlarged and oedematous. All lesions are progressive and result from the cellular immune response of the host, and not directly from viral damage.

• CAE gross lesions include swelling of carpal joints due to increased synovial fluid, and enlarged and hard joint heads, there is also articular cartilage ulceration on the joint cavity, and severely swollen mesenteric lymph nodes. In the neurological form there is asymmetric, brownish pink, swollen areas, most commonly located in the cervical and lumbosacral spinal cord segments.

#### **Diagnosis:**

- Field based diagnosis using clinical signs and post mortem lesions.
- Confirmatory diagnosis is collection of whole blood in live animal for ELISA and PCR testing or post mortem collection of lung, lymph node and joint tissues for virus genetic identification through PCR.
- Differential diagnoses of progressive pneumonia include pulmonary adenocarcinoma, pleural abscesses, and pulmonary caseous lymphadenitis. Listeriosis, scrapie, rabies cerebrospinal nematodiasis, and space-occupying lesions should be considered when the neurologic form (visna) of the disease is seen.
- Differential diagnosis for progressive paresis and paralysis exhibited by young kids should include enzootic ataxia, spinal cord abscess, cerebrospinal nematodiasis, spinal cord trauma, and congenital anomalies of the spinal cord and vertebral column. If a neurologic examination indicates brain involvement, polioencephalomalacia, listeriosis, and rabies should be considered. The pulmonary form of caseous lymphadenitis may have a similar clinical presentation to the pulmonary form of CAEV in adult goats.

#### Control

• Currently, there is no practical, effective treatment, and no vaccines are available.

Therefore, the only means for control and prevention is serologic testing and culling of positive animals. This is not practical in Marsabit and animal service providers should advise livestock keepers to cull animals with CIP or CAE as they will continuously shed the virus in the environment and pass it to off springs.

- Because of the long incubation period and time to seroconversion once signs appear it is advisable to cull animals.
- Pregnant does and ewes showing signs once they deliver young. The livestock keeper should immediately separate neonates from dam to prevent suckling of colostrum.
- Isolated dams should be fostered to other health dams or fed on heat-treated colostrum, and later raised on milk replacer.

Zoonotic potential- None identified

Notifiable disease- Yes, WOAH listed disease, report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Cull sick animals as there is no treatment and the longer the sick animal stays in the flock the more it infects other.

#### References

- Son, G. I., Hong, E. J., & Shin, H. J. (2021). Case Report: A Case of Caprine Arthritis Encephalitis in Dairy Goat Farms in South Korea. Frontiers in veterinary science, 8, 773039. https://doi.org/10.3389/ fvets.2021.773039
- Moroz, A.; Czopowicz, M.;Sobczak-Filipiak, M.; Dolka, I.;Rzewuska, M.; Kizerwetter-Swida, M.; Chrobak-Chmiel, D.; Mickiewicz, M.; Witkowski, L.; Szalu's-Jordanow, O.; et al. The Prevalence of Histopathological Features of Pneumonia in Goats with Symptomatic Caprine
- Arthritis-Encephalitis. Pathogens 2022,11, 629. https://www.mdpi.com/2076-0817/11/6/629/ pdf?version=1653905990
- Rwambo, P., Brodie, S. & DeMartini, J. Ovine Lentivirus is Aetiologically Associated with Chronic Respiratory Disease of Sheep on the Laikipia Plateau in Kenya. Tropical Animal Health and Production 33, 471–487 (2001). https://doi.org/10.1023/A:1012728528924
- 5. MSD Veterinary Manual https://www.msdvetmanual.com/
- 6. OIE (2020). Technical Disease Card



Limb paralysis due to infection with maedi-visna virus (MVV). Unilateral hindlimb paresis (a) early stages of the disease and later ataxia and hindlimb paralysis (b).

#### Picture source:

Diagnosing limb paresis and paralysis in sheep ( 2015) http://dx.doi.org/10.1136/inp.h5547



### Figure 104

Appearance of a goat infected with CAE (advanced stage) in which the animal is hitting its head against the pen wall (left picture), has depressive behaviour (middle picture), and has joint inflammation (right picture).

#### Picture source:

Short communication: Genetic parameter estimates for caprine arthritis encephalitis in dairy goats (2020) https://doi.org/10.3168/jds.2019-17740



## Figure 105

CIP/PIP affected lung is enlarged and heavy and has does not collapse when thoracic cavity is open. Note rib impressions on the costal surfaces of the diaphragmatic lobes (black arrow)

#### Picture source:

Ovine Progressive Pneumonia: Diagnosis and Seroprevalence in the South of Sonora, Mexico (2021) https://doi.org/10.1155/2021/6623888

## Sheep Diseases: Protozoal diseases

## 6.8 Babesiosis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Birte	Bira	Kati Ti Digee	Nkula	Lodip	Sin Bura

Refer to chapter 4: Cattle diseases, section 4.11 Babesiosis

#### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

## Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Clear bush and infested pasture through burning
- Migrate out of heavily tick infested areas.

## Sheep Diseases: Parasitic Diseases

## 6.9 Helminthiasis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Minii	Minni	Deyah	Ntumwa	Ngipelei	Chade

#### Epidemiology

- Helminths affecting sheep and goats can be divided into nematodes (round worms), trematodes (flukes) and cestodes (tape worms).
- Nematodes include: Haemonchus contortus, Nematodirus species, Bunostomum species, Ostertagia circumcinta, Trichostrongylus species
- Cestodes include: Monenzia expanza is the most common cestode of sheep
- Trematodes include: Fasciola hepatica (liver fluke) is the most common trematode of sheep
- Important helminths of sheep and goats are Haemonchus contortus and liver flukes-Fasciola hepatica.
- In Marsabit county, livestock keepers in the year 2022 indicated that helminthiasis was endemic and occurred throughout the year with an apparent annual morbidity and mortality rates of less than 7% and case fatality rate of 47%.

Haemonchosis- Barber's pole worm

#### Transmission and Pathophysiology

• H. contortus passes through six stages of life which include egg, four larval stages, and adult. Female adults lay numerous eggs with an average of more than 1200 eggs a day. The eggs are passed through faeces to pastures. The infective larval stage is L3.

- The L3 move out of the faeces, onto the soil, then randomly migrate horizontally and vertically up the grass sward independent of free water. The larvae do not feed, relying on stored energy for survival. In tropical climates like in Marsabit, the survival of L3 is less than five weeks, as the larvae are more active and consume energy stores. This provides an opportunity for spelling of pastures (that is periods without grazing) that can be used as a control method.
- The L3 larvae infective stage of H. contortus are consumed when the animals graze the pasture sward. Once ingested, a range of factors derived from the host sheep will influence survival and establishment of the L3 larvae within the abomasum. These include age, breed, health, and nutritional status of the animal as they impact on an individual's animal ability to mount an immune reaction to the H. contortus larvae.
- In the abomasum, the larvae burrow into the mucosa into the gastric pits where they use their lancet to cut the abomasal tissue and induce haemorrhages and ingest the leaking blood. The larvae then mature into adults.

Female worms start producing eggs 12 to 15 days after ingestion and produce. The infected animals may lose up to 30  $\mu$ L of blood every day due to one parasite.

• Previous research had indicated that hypobiosis, arrested development of ingested larvae occurred in the host during dry or drought conditions. However, a recent study in West Africa that found a high incidence rate of 77.8 to 100%) with no definite seasonal distinction. There was however, a clear seasonal trend in the worm burden of animals; higher burdens were evident during the rainy rather than the dry season. The study found no significant hypobiosis during the unfavourable dry season (November to March).

• This finding may explain why Marsabit livestock keepers indicated that bottle jaw which is an indication of hemonchosis in sheep and goats occurred throughout the year with more cases reported during the long dry season that occurs from July to September.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

#### Clinical signs

- Clinical signs are associated with anaemia and hypoalbuminemia. The magnitude and consequences of the anaemia and hypoalbuminemia depend on parasite burden, their stage of development, the age of animal and immune response.
- Haemonchosis clinical syndrome is categorised into three stages; hyper acute, acute, and chronic
- Hyper acute haemonchosis is rare and occurs in animals exposed to heavy burdens of
- o H. contortus (up to 30,000 larvae per animal) over a short period of time. This form is common in young animals. Death is sudden with or no previous clinical signs of anaemia. Bloody diarrhoea may be the only sign.
- Acute haemonchosis is the most common form and is characterized by lower burdens of H. contortus (approximately 2000–20,000 larvae per animal), resulting in a less severe loss of blood and anaemia that develops over a longer period of time. Animals show clinical signs associated with anaemia (conjunctiva colour that varies from the normal, red-pink colour to extreme white) and hyperproteinaemia (specifically hypoalbuminemia). The main clinical signs are lethargy, weakness, increased respiratory and heart rates, pale mucous membranes and bottle jaw. Hyperproteinaemia

occurs due to blood loss, leakage of protein into the abomasal lumen through disrupted cell junctions and increased permeability and decreased protein absorption due to abomasal epithelial cell loss. There is also increased protein use to repair damaged tissues and mount an immune response. Hypoalbuminemia results in decreased intravascular oncotic osmotic pressure and oedema that may be generalized or localized in the intermandibular space (referred to as "bottle jaw" or cervical region. Diarrhoea is not a key feature of Haemonchus infection unless there is concurrent infection with other helminth species or coccidiosis.

• In chronic or subclinical haemonchosis, sheep and goats ingest few numbers of H. contortus as the environment is not suitable for the development of the infective larvae. Infection manifest as a reduction in productivity, feed conversion and growth rate.

#### Diagnosis and Post mortem lesions

• Field based diagnosis – clinical signs of anaemia and bottle jaw that is supported with the use of FAMACHA Score card. The score card allows rapid anaemia classification in the field. The FAMACHA system was developed in South Africa and is based on conjunctival mucous membrane colour in small ruminants on a scale of 1–5 with various stages of anaemia related to haemonchosis with their measured Packed Cell Volume (PCV). A value of 1 (red) is considered non-anaemic and a PCV of 35%, 2 a PCV of 25%, 3 a PCV of 20%, 4 a PCV of 15%, through to a value of 5 (white) and a PCV of 5%, being severely anaemic.

- Post-Mortem diagnosis- Examination of dead small ruminants is another rapid, accurate method of confirming a diagnosis, as it allows the visualization of the H. contortus parasites in the abomasum of affected animals. The parasites have a characteristic red and white, twisted "barber's pole" appearance related to the blood-filled intestine spiralling around the white reproductive tract of the female nematodes.
- Confirmatory diagnosis- Confirming the presence of parasites through faecal egg counts and larval culture and haematology testing of EDTA blood for anaemia indicators: that include Complete Blood Cell Count (CBC), PCV and evaluation of Mean Corpuscular Volume (MCV) and Mean Corpuscular Haemoglobin Concentration (MCHC) and reticulocyte (immature RBC) counts. Molecular methods as a confirmatory test have a high sensitivity but have limitations due to the inhibition effects of faeces on the polymerase chain reaction (PCR) and their accessibility to veterinarians in the field. Development of suitable PCR tests is on-going, with droplet digital PCR showing promise. using the McMaster method for faecal egg count has a specificity but a lower sensitivity when compared to molecular methods.

#### Treatment and control

- Widespread anthelmintic resistance limits the effectiveness of both treatment and prevention of haemonchosis. Successful management of haemonchosis relies on the early recognition of infection through periodic monitoring of H. contortus burdens (FAMACHA score and or faecal egg count), strategic use of anthelmintics and grazing management.
- · Regular deworming with broad-spectrum

anthelmintics 2 weeks after the onset and at the end of the rainy seasons. Proper dosing based on manufacturers instruction that is guided by the weight of animal is important. Use of a weigh band can help minimise under dosing or overdosing incidences.

- Broad-spectrum anthelmintics currently available belong to five different chemical groups:
- 1) Benzimidazoles (white or blue drenches) like fenbendazole, oxfendazole and albendazole,
- 2) Imidazothiazoles (yellow drenches) like levamisole
- Macrocyclic lactones (clear or watery drenches or injectables like Ivermectin and doramectin.
- Amino-acetonitrile derivatives-is not readily available as it a new anthelmintic compound (2010). Monepantel effectively controls adult and L4 stages of ruminant nematodes, including

multidrug resistant helminths.

- 5) Spiroindoles like Derquantel has been approved as a combination anthelmintic product with
  - abamectin. This is also a new anthelmintic that is not readily available. The combination is effective against adult and L4 stages of ruminant nematodes that have shown multidrug
  - resistance.
- In Marsabit county, strategic use of anthelmintic can be used due to the high cost of treating the large small ruminant herds. Animals with FAMACHA score of 3 and above should be the only ones treated at the correct dosage. In addition, the timing of treatment should be at the beginning of the rainy season and at the end. Pasture management such as pasture rotation to allow desiccation of larvae to high environmental temperatures and delaying animals from accessing pasture when there is morning dew are good strategies used by livestock keepers.
- Barbervax®, a vaccine containing Haemonchus contortus gut membrane glycoprotein antigens have been developed and efficacy trials conducted. Results indicated that naturally infected vaccinated sheep had

significant egg count reductions compared with controls. Vaccination also prevented the periparturient rise in egg shedding of ewes, as well as egg shedding in lambs.

#### Ethnoveterinary practices

· See tables 18 and 19

**Ethnoveterinary control practices**- Delay animals grazing early morning during the rainy season, avoid contaminated pastures.



Anemia

Figure 106 Anaemia and bottle jaw due to haemonchosis



Picture source: Anaemia and bottle jaw due to haemonchosis Photo credit- MSD





### Figure 107

Haemonchus contortus parasites in the abomasum of an affected lamb, demonstrating the characteristic red and white twisted "barber's pole" appearance.

Picture source: Professor K G Thompson, Massey University, New Zealand

#### References

- Bello HJS, Lins JGG, Albuquerque ACA, Ferreira GB, Amarante MRV and Amarante AFT (2022) Prophylactic Effects of Ivermectin and Closantel Treatment in the Control of Oestrus ovis Infestation in Sheep. Front. Vet. Sci. 8:798942. https://doi.org/10.3389/fvets.2021.798942
- Flay, K.J.; Hill, F.I.; Muguiro, D.H. A Review: Haemonchus contortus Infection in Pasture-Based Sheep Production Systems, with a Focus on the Pathogenesis of Anaemia and Changes in Haematological Parameters. Animals 2022, 12, 1238. https://doi.org/10.3390/ani121012
- MSD Veterinary Manual https://www. msdvetmanual.com/
- Teixeira, M., Matos, A.F.I.M., Albuquerque, F.H.M.A. et al. Strategic vaccination of hair sheep against Haemonchus contortus. Parasitol Res 118, 2383–2388 (2019). https://doi.org/10.1007/ s00436-019-06367-x



Figure 108 Tapeworm segments being expelled with diarrhoea in a goat

Picture source: Dr. Ann Zajac Virginia-Maryland College of Veterinary Medicine

Oestrus ovis larvae cause cavitary myiasis in small ruminants. The larvae are an obligate parasite of the nasal and sinus cavities of sheep and goats. In endemic areas, the O. ovis larvae have been found in humans' eyes and nasopharyngeal airway. The female fly (adult female fly (Oestrus ovis) commonly known as the sheep nasal bot fly. Flies around the head of its host to deposits larvae at a few centimetres from sheep nostrils. In case of the fly presence, the animals shake their heads and hide the muzzle on the soil or in the others sheep's wool. Once deposited the larvae enter the nostrils where they use the hooks and spines to provoke inflammation and mucous nasal discharge which the larvae feed on. Sheep infested with larvae are often seeing sneezing and have muco purulent nasal discharge. Animals may also lose weight. Treatment involves injecting Ivermectin at a dose rate of 0.2mg/kg BWT given SC once or repeated after 2 weeks if the clinical signs persist. In heavy infestation diagnosed through post mortem of the nasal cavity a combination of Ivermectin and closantel (10mg/ kg orally) can be used.



#### Figure 109

Photos of lambs with oestrus ovis larva infestation showing different nasal discharge scores.

No discharge (A), serous discharge (B), sero-mucous discharge (C), thick mucous discharge (D) and mucopurulent thick discharge (E)



## Figure 110

Cross section of the nasal sinuses for the recovery of O. ovis larvae from naturally infested lambs (B)

#### Picture source:

Prophylactic Effects of Ivermectin and Closantel Treatment in the Control of Oestrus ovis Infestation in Sheep (2022) https://doi.org/10.3389/fvets.2021.798942https://doi. org/10.3389/fvets.2021.798942

### Figure 111

Cephalopina titillator larvae (camel nasal bot fly) in the nasal cavity of a camel, Photo credit, Diba Denge, Animal Health Technician, Marsabit County

## Figure 113

FAMACH score card showing when to administer anthelmintics



Picture source: https://link.springer.com/10.1007/978-3-642-27769-6\_4307-1



Picture source: https://u.osu.edu/sheep/2010/06/16/usefamacha-correctly/
## 6.10 Coenurosis

Other name: Epilepsy of sheep and goats, acute or chronic gid or sturdy

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Sirgo	Sirgo	Sirgo	Sirgo	Nkerep	Sigire

### Case definition

- Fatal parasitic disease affecting sheep and goats. It is caused by the larval stage of Taenia multiceps, the adult tapeworm is found in the intestines of dogs. Sheep and goats are intermediate hosts and do not support the larvae that hatches from the eggs to mature to adult forms. Instead the aberrant larvae migrate to the nervous tissue and forms cysts in the brain.
- The larval stage, Coenurus cerebralis, is typically found in the central nervous system (CNS) of wide range of livestock. Cysts in sheep are mainly in the brain and spinal cord. However, in goats, in addition to the central nervous system (CNS) they can have cysts in musculoskeletal system, subcutaneous, connective tissue, peritoneal and pelvic cavities, the liver and the lungs.
- Zoonotic parasite as evidenced by multiple cases of coenurosis in humans caused by T. multiceps.

### Aetiology and Epidemiology

- The causative agent is Coenurus cerebralis, the larval stage of Taenia multiceps, a tapeworm that infests the small intestine of carnivores.
- Contamination of pastures grazed by sheep by dog faeces can result in larval invasion of the central nervous system and clinical disease. The life cycle is completed when the carnivorous definitive host ingests infested sheep's brain.
- In Marsabit county, livestock keepers in the year 2022 estimated an apparent morbidity and mortality rates 1% and a 100% case fatality rate.

 In Marsabit county the disease can occur at any time of the year, but livestock keepers indicate the signs manifest more during the long dry season especially when animals are watered after a period of water depravation.

### Pathophysiology

- The adult tapeworm, Taenia multiceps, inhabits the dog guts where it produces eggs that are defecated onto pasture. The eggs can survive on pasture for several weeks.
- Sheep and other intermediate hosts become infected by consuming pasture contaminated with Taenia multiceps eggs.
- The eggs hatch into larvae within the gut of the intermediate host. The larvae migrate into the brain of the intermediate host, a process that may last several months, where they form cysts. The symptoms associated with Coenurosis vary with the location and size of the cysts as well as with the pressure the cysts on the cerebrum.
- Molecular evaluation of cerebral and noncerebral coenuri from sheep and goats established that the larval stage of Taenia multiceps in the brain and other aberrant sites are different. Larvae found away from CNS in goats are now called Taenia gaigeri and Taenia skrjabini in sheep.

### Clinical signs

- Circling in one direction, Tilting, lifting or lowering the head
- Constant loud bleating and excitement,
- Weight loss
- Cysts found on post mortem on brain or spinal cord and in goats in other tissue.

Diagnosis	Treatment	Control
Diagnosis is confirmed on necropsy by the presence of Coenurus cerebralis cysts in the brain of sheep. Clinical signs alone cannot be used as a diagnostic method. The best diagnostic method that is not feasible in Marsabit is clinical signs combined with accurate localization of the cyst using diagnostic imaging.	Treatment with medication is not usually successful, farmers resort into culling affected animals	The most effective means of controlling coenurosis, is through the regular anthelmintic treatment of farm dogs with an effective taeniacide (i.e. praziquantel at 5 mg/kg body weight) at 6- to 8-week intervals and the proper disposal of sheep carcasses to prevent scavenging by herding and stray dog and wild carnivores Most of the naturally occurring cases of clinical coenurosis in sheep have been observed in young animals. Suggesting the existence of an age-related resistance to T. multiceps infection in sheep, although the mechanism by which this may occur is unknown. There is a vaccine under development
Differential diagnosis		Zoonotic potential- Yes for only coenurosis

- Listeriosis, nasal bots syndrome, louping ill, scrapie, sarcocystosis, polioencephalomalacia and cerebral echinococcosis.
- The age of an infected animal may aid diagnosis, since the symptoms of cerebral coenurosis typically manifest in young animals; however, immunocompromised animals as well as animals raised in isolation may develop coenurosis when adults.

**Zoonotic potential**- Yes for only coenurosis Notifiable disease- No

### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

### Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Cull affected animals

### References

1. Varcasia, A., Tamponi, C., Ahmed, F. et al. Taenia multiceps coenurosis: a review. Parasites Vectors 15, 84 (2022). https://doi.org/10.1186/s13071-022-05210-0



Figure 114 Coenurus cerebralis larval cyst in sheep brain.



Figure 115 Adult Taenia multiceps under light microscopy. b Proglottids of T. multiceps in dog faeces. c Mature coenurus isolated from sheep; clusters of protoscoleces are clearly visible without magnification



## Figure 116

Graphical representation of the life cycle of Taenia multiceps

### Picture source:

Varcasia, A., Tamponi, C., Ahmed, F. et al. Taenia multiceps coenurosis: a review. Parasites Vectors 15, 84 (2022). https://doi.org/10.1186/s13071-022-05210-0

## 6.11 Mange

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
D'oq	D'oq	Haddo	Lpepedo	Ekoikoi / Pepedo	Gogosich

### Case definition

- Mite infestations cause skin irritation leading to oozing (exudation) and skin thickening. The intense irritation leads to disruption of feeding patterns and weight loss.
- Mange occurs worldwide and in all seasons of the year in the tropics.

### Aetiology

The most important parasitic mite species of sheep are:

- Psoroptes (P) ovis that causes psoroptic mange, also called sheep scab occurs worldwide.
- Sarcoptes (S) scabiei var. ovis that causes sarcoptic mange, also called scabies occurs worldwide.

- Chorioptes (C) ovis that causes chorioptic mange, also called leg mite, foot scab occurs worldwide.
- Demodex (D) ovis, responsible for sheep demodectic mange

The most important parasitic mite species of goats are:

- S scabiei var caprae
- C. bovis
- Demodex caprae infests goats.
- In Marsabit county, livestock keepers in the year 2022 estimated the apparent morbidity rate on 3% with no mortality. Mange cases occurred through the year but more cases are seen when animals are in nutritional stress during the long dry season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Dry	Adholes	Adholes	Nabhai Deer	Lamei-oodo	Akamu	Mar Me
(July- September)					Nakooyan	Mederka

### Clinical signs goats

- This mite infests the head and face. Lesions manifest with formation of crusts and intense pruritus.
- Affected animals have decreased reproduction, meat gain, and milk yield.
- In goats, S scabiei var caprae is responsible for a generalized skin condition characterized by marked hyperkeratosis. Lesions start usually on the head and neck and can extend to the inner thighs, hocks, brisket, ventral abdomen, and axillary region.
- S scabiei var caprae has zoonotic. Consistent with other animal variants of Sarcoptes, zoonoses are initiated from direct contact

with infected animals but are self-limiting infestations.

• C bovis in goats is similar to that in cattle, with papules and crusts seen on the feet and legs.

### Treatment and Control

- Hot lime sulphur spray or dip is labelled for use against sarcoptic, psoroptic, and chorioptic mites in sheep. Treatment should be repeated every 12 days if needed.
- Certain formulations of synthetic Pyrethroids or organochlorines (Amitraz) are labelled for use in sheep and goats. As with cattle, they are not considered the compound of choice but if

used, the animals should be thoroughly wetted with the product and re-treated in 10–14 days.

• Macrocyclic lactones (Ivermectin) is the drug of choice and given as two doses given 7 days apart at a dose rate of 0.2mg/kg BWT given SC.

Figure 117 Sarcoptic Mange in a goat showing alopecic areas of the skin



Picture source:

https://www.researchgate.net/publication/319454346\_THERAPEUTIC\_MANAGEMENT\_OF\_SARCOPTIC\_MANGE\_IN\_GOAT\_A\_COMPARATIVE\_STUDY\_ALLOPATHIC\_AND\_HERBAL\_PRODUCT/figures



Figure 118 Location of ectoparasite in goats indicates if it is a mite or flea infestation

> Picture source: https://www.farmhealthonline.com/US/ disease-management/goat-diseases/mange-ingoats/

# Pathophysiology and Clinical signs in sheep Psoroptes ovis (sheep scab)

- Adult Psoroptes female mite lays 1 to 3 eggs a day, a total of about 50 to 100 eggs in their lifetime. Adult life lasts for about 50 days.
- The shortest life-cycle duration from eggs to eggs of the next generation is about 10 to 14 days.
- Psoroptes mites do not dig tunnels in the skin, but the mite faeces cause an allergic reaction of the host's skin, which reacts producing exudations and skin thickening and hardening (lichenification) with formation of papules, scales and crusts (excoriations), mostly with wool loss.
- The mites feed on the exudates and secretions produced by the affected skin. Large scabs may develop that spread to cover the entire body in 2 to 3 months if left untreated. Mites concentrate at the edge of the growing scabs. Mites are one host parasites.
- Transmission within a herd is mostly by physical contact. Mites do not actively jump or crawl from one host to another one, but are passively transmitted when animals come in close contact. Psoroptic mites and eggs can survive 2 to 3 weeks off the host (in clumps of fallen wool or on inanimate objects like fence posts).
- Sheep scab lesions affect the back, the flanks and the shoulders. Infestations remains unnoticed until wool loss becomes evident, which mostly means that the whole flock is already infested. Affected animals suffer from intense itching (pruritus) and react vigorously scratching, biting and rubbing against objects, which causes injuries that can be infected with secondary bacteria. All this leads to weight loss and wool loss, reduced milk production, and general weakness that makes the affected animals more susceptible to other diseases. Left untreated it is often fatal, especially for lambs. Hides of affected animals are downgraded or rejected at slaughter.
- Diagnosis based on clinical symptoms, confirmed by examining skin scrappings of affected parts under the microscope for visualization of the mites.

• Psoroptic mites are not infectious to humans.

### Sarcoptes scabiei var. ovis

- Sarcoptic mites of sheep are a species-specific strain of Sarcoptes scabiei, a mite species that infests also cattle, pigs, other livestock and also humans. (zoonosis) causes scabies.
- Sarcoptic mites spend their whole life on the same host. Mites do not actively jump or crawl from one host to another one, but are passively transmitted when animals come in close physical contact. However, sheep can pick mites from the immediate environment or fomites. There are no external vectors that transmit the mites.
- The mites dig tunnels beneath the skin. Their saliva has potent digestive enzymes that dissolve the skin tissues. They feed on the resulting liquids. They do not suck blood.
- Adult females deposit their eggs in tunnels, which hatch in 3 to 5 days. The whole development through several larval and nymphal stages can be completed in less than 2 weeks.
- Adults live for 2 to 3 weeks. Off the host the mites survive only a few days.
- Disease not as severe as that of Psoroptes mites. Mite digging causes skin irritation, which is enhanced by allergic reactions to the saliva. The affected skin develops pimples and papules that become crusty, and shows hardening, thickening, and folding.
- Skin lesions mainly seen in non-wooly skin and frequently start on the head, then later spread to sides of the neck and fore legs.

### Chorioptes ovis

- Chorioptic sheep mites (also called "leg mites", or "foot scab") are less harmful when compared to psoroptic or sarcoptic mites. They are not transmitted to humans.
- They have chewing mouthparts and neither suck blood, nor dig tunnels as sarcoptic mites, but bite the outer skin layers and feed on skin debris, fat, lymph or exudates. The whole development through several larval and nymphal stages can be completed in about 32

weeks. Off the host the mites survive only a few days.

- Preferential sites or chorioptic mites are the hoofs and lower part of the legs.
- Can affect the scrotum, the face and the lips. Affected parts show formation of scales and crusts. Rams are often more affected and spread the disease in the flock, especially if they are permanently confined. Severely infested rams may suffer from partial paralysis and low reproductive performance.
- Itching is not as severe and the scratching and biting reactions of affected animals are less vigorous.

### **Treatment and Prevention**

- Two injections with a macrocyclic lactone (Ivermectin) with 7 to 10 days' interval.
- Topical acaricide and pour-ons are not reliable

for controlling psoroptic mites.

- Infected premises/ boma should be kept vacant for at least 2 weeks. This allows surviving mites or eggs to die.
- There are no vaccines that will protect sheep by making them immune to the mites.
- There are no repellents, natural or synthetic that will keep mites away from sheep.

### Ethnoveterinary treatment practices

• Refer to tables 18 and 19

# Ethnoveterinary control practices (refer to table 10 for local dialect names)

- Isolate sick animals and if possible build a separate night boma for them
- Migrate from boma and leave after you have burned the infested livestock boma





Picture source: https://link.springer.com/article/10.1007/s12639-012-0157-5

Further Reading Merck Veterinary Manual. https://www.merckvetmanual.com

## Chapter 7: Goat diseases

### Table 21: Local dialect name of priority goat diseases in Marsabit county

Bacterial Goat Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Contagious Caprine Pleuropneumonia (CCPP)	Sombes Ree	Sombes Ree	Ilkibei	Ilkibei	Loukoi	Louke Ayiet
Anaplasmosis	Darabot	Darabot / Riqanot	Ndis	Ndis	Lonyang	Bash Kinne / Bash Raarka
Heart Water	Qando	Qando	Ririkhanyo	Lmilo	Amil / Lotai	Bash Chira
Enterotoxaemia	Dukub Tiru / Qurtam / Marchakas	Dukub Tiru	Lbus	Lbus / Nadomanyita	Lobus	Suony
Foot Rot	Bargao	Bargao	Dokoho	Ngojini	Ebaibai	Aiy Komfo / Konof
Mastitis	Budha	Budha	Budha	Ntiroto E Lkii	Edeke- Aeket	Bash Oto
Viral Goat Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Peste Des Petits Ruminants (PPR)	Thadi Ree	Thadi Ree	Siim / Lmokhor	Lodua Ntare / Nagumo	Lomaa / Lonoo / Lokiyo	Mokodo / Lokotokonyan/ Lojou
Sheep And Goat Pox (SGP)	Kurtubale	Kurtubale	Nyinyilo	Nairiri / Tune	Etune/ Tune	Verte / Ngerero
Contagious Ecthyma (Orf)	Umburur	Abthara	Afturo	Afturo	Ngiboruok	Dicho
Foot And Mouth Disease (FMD)	Oyale	Oyale	Lkulub	Lkulub	Lojaa / Lokituk / Lokulup	Huluf/ Guluf

Parasitic Goat Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Coenurosis	Sirgo	Sirgo	Sirgo	Sirgo	Ngithurko / Nkerep	Sigire
Helminthiasis	Minii	Minii	Deyah	Ntumwa	Ngipelei / Ngirtan	Chade
Fascioliasis / Liver Fluke	Minni Tiru	Minni Tiru	Deyah Ti Helesi	Lkurui Lemonywa	Lomany	Nakarkarsha
Mange	Chito	Chito	Haddo	Lpepedo	Ekoikoi / Emitina Pepedo	Nyaacho
Protozoal Goat Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Babesiosis	Birte	Bira	Kat Ti Digee	Nkula	Lodip	Sin Bura
Coccidiosis	Tumma	Tumma	Hari Dige	Sirr E Lodo	Lorenge- Malteny	Uu-Bura

### Table 21(cont): Local dialect name of priority goat diseases in Marsabit county

### Table 22: Plants identified by livestock keepers as having medicinal value in treating goat diseases

Medicinal plants	Target Goat diseases
Acacia Nubica	ССРР
Euphorbia species	ССРР
Aloe species	ССРР

### 190 Goat Diseases

Table 22: Plants identified by livestock keepers as having medicinal value in treating goat diseases

Traditional Practices	Target Goat disease				
Salty water from saline wells, springs or Lake	Goat pox, FMD, Foot rot				
Turkana- Topical application					
Old engine oil	Goat pox				
Bleeding animal from base of ear	Enterotoxaemia				
Chewing tobacco	CCPP, PPR				
Hot Iron or stone cauterization	Enterotoxaemia, Goat pox, PPR, CCPP				
Cooking oil	FMD				
Wood ash	Goat pox				
Alcohol- (Vodka)	Enterotoxaemia				
Boiled Tea leaves (Sturungi)	Enterotoxaemia, FMD, Foot rot				
Tetracycline antibiotic capsules intended for human	CCPP, PPR, Anaplasmosis				
use, the powder is removed from the capsule casing					
and mixed with cooled boiled water and injected into					
the muscles of the animals					
Pepper powder sometimes mixed with cattle urine	Enterotoxaemia, PPR				
NB: Text in <b>red</b> highlights practices that go against animal welfare principles or contribute to AMR or can serve as					

transmission route to a new infection

### Table 24: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority goat diseases

Type of VMP	Active ingredient	Purpose	Target Goat diseases
Antibiotic	Oxytetracycline	Curative and Preventive for secondary bacterial infections	CCPP, PPR, Heart water, Goat pox, FMD
Antibiotic	Penstrep® procaine penicillin G and dihydrostreptomycin	Curative and Preventive for secondary bacterial infections	CCPP, Goat pox, Mastitis
Anthelmintic	Albendazole / Levamisole	Curative	Haemonchosis
Anthelmintic	Ivermectin	Curative	Goat pox, PPR
Antibiotic	Diseptoprim ® boluses Sulphadiazine and Trimethoprim	Curative	Enterotoxaemia, Coccidiosis
Metabolic support	Multivitamin	Supportive treatment	CCPP, PPR, Goat pox
Acaricides	Amitraz, synthetic pyrethroids, organophosphate and carbamates	Curative	Ectoparasite infestation (Lice and Fleas)
Antibiotic	Tylosin	Curative and Preventive for secondary bacterial infections	CCPP, PPR, Goat pox, FMD

NB: The drug of choice used by livestock keepers to treat the diseases highlighted in red do not have therapeutic efficacy on the causative microbes. The extra label use of drugs promotes antimicrobial resistance.

## Goat Diseases: Viral diseases

## 7.1 Peste des Petits Ruminants (PPR)

Refer to Chapter 6: Sheep diseases section 6.3 Peste des Petits Ruminants (PPR) including for ethnoveterinary control practice information

Ethnoveterinary treatment practices

Refer to tables 22 and 23

## 7.2 Sheep and Goat Pox (SGP)

Refer to Chapter 6: Sheep diseases section 6.4 Sheep and Goat Pox (SGP) including for ethnoveterinary control practice information

### Ethnoveterinary treatment practices

Refer to tables 22 and 23

## 7.3 Contagious ecthyma (orf)

Refer to Chapter 6: Sheep diseases section 6.6 Contagious ecthyma (orf) including for ethnoveterinary control practice information

### Ethnoveterinary treatment practices

Refer to tables 22 and 23

## 7.4 Foot and Mouth Disease (FMD)

Refer to Chapter 4: Cattle diseases section 4.1 Foot and Mouth Disease **Ethnoveterinary treatment practices** 

Refer to tables 22 and 23

### Ethnoveterinary control practices (refer to table 10 for local dialect names)

• Isolate sick animals and avoid areas with outbreaks

• Present animals for vaccination

• Do not buy animals from the markets during an outbreak (especially goats as they do not show clinical signs but are carriers of disease). Disease in cattle is more severe when compared to the disease in sheep. Goats are more resistant to FMDV as they show no or less severe clinical signs.

## Goat Diseases: Parasitic diseases

## 7.5 Helminthiasis

Refer to Chapter 6: Sheep diseases section 6.9 Helminthiasis including for ethnoveterinary control practice information Ethnoveterinary treatment practices Refer to tables 22 and 23

## 7.6 Coenurosis

Refer to Chapter 6: Sheep diseases section 6.10 Coenurosis including for ethnoveterinary control practice information Ethnoveterinary treatment practices Refer to tables 22 and 23

## 7.7 Fascioliasis (Liver fluke infestation)

Refer to Chapter 4: Cattle diseases section 4.13 Fascioliasis including for ethnoveterinary control practice information Ethnoveterinary treatment practices Refer to tables 22 and 23

## 7.8 Mange

Refer to Chapter 6: Sheep diseases section 6.11 Mange including for ethnoveterinary control practice information Ethnoveterinary treatment practices Refer to tables 22 and 23

## Goat Diseases: Protozoal diseases

## 7.9 Babesiosis

Refer to Chapter 4: Cattle diseases section 4.11 Babesiosis including for ethnoveterinary control practice information

### Ethnoveterinary treatment practices

Refer to tables 22 and 23

## 7.10 Coccidiosis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Tumma	Tumma	Hari Dige	Sirr E Lodo	Lorenge-Malteny	Uu-Bura

### Case definition

- Coccidia are single-celled, microscopic parasites that live and reproduce in animal cells. Coccidia of sheep and goats are host specific and almost all kids and lambs will become infected.
- Significant damage to the cells lining the small intestine, caecum and large intestine results in diarrhoea which may contain mucus or blood. This is often accompanied by straining, pain, weight loss and sometimes death of the lamb and kids.
- Recovered animals have poor growth rates due to damage of epithelial lining of the intestine and reduced ability to absorb nutrients from ingested food.
- Most of the different Eimeria species found in sheep are harmless and may even provide beneficial effects. Distinguishing between an infection with coccidia and the disease coccidiosis is very important in lambs.

### Aetiology, Epidemiology and Pathogenesis

• Numerous species of Eimeria species cause disease in goats in Kenya. A study estimated

the species prevalence in goats to be E. arloingi (71%) followed by E. ninakohlyakimovae (65%) and E. hirci (59% percent).

- The disease is severe and often fatal in kids.
- In sheep, only two harmful (or pathogenic) species cause clinical disease (Eimeria ovinoidalis and Eimeria crandallis) when there is either a heavy infestation in lambs or they have lowered immunity. Disease is mainly seen in lambs 4 to 8 weeks old and rarely seen in lambs over 3 months old.
- Lambs and kids take in pathogenic coccidia oocysts (eggs) by mouth. Inside the intestine, the oocysts hatch, invade the mucosal intestinal cells and multiply dramatically. The coccidia emerge by bursting the cells open damaging the inside lining of the intestine. A dramatic number of oocysts are shed in faeces and this can be many million times higher than the number that were ingested. It takes two to three weeks from infection via the mouth to the passing of oocysts from the rectum.
- · In Marsabit county, livestock keepers in the

year 2022, estimated an apparent annual morbidity rate of 10% with a mortality rate of 8% and a case fatality rate of 80%. The cases were common during the long rainy season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

### **Clinical Signs**

- Coccidia are host-specific and each animal species is susceptible to infection with various coccidia species. In goats, Eimeria spp. are most common.
- The protozoan parasite goes through its life cycle in the small intestine, destroying the epithelial cells.
- Transmission of oocytes is via the faecal-oral route. This process involves oocysts being passed when an animal defecates, and another animal ingests the contaminated substrate.
- Clinical cases vary from loss of appetite and decrease in weight gain to severe cases involving chronic diarrhoea, fluid faeces containing mucus and blood, straining in attempt to pass faeces, loss of weight, and dehydration and death.

### Post-mortem lesions

• Lesions are confined to the small intestine, which may appear congested, haemorrhagic, or ulcerated, and have scattered pale, yellow to white macroscopic plaques in the mucosa.

### Diagnosis

- Clinical signs of bloody diarrhoea and post mortem lesions
- Microscopic analysis of faeces to observes oocysts.
- In lambs, clinical signs of severe, bloody diarrhoea and straining can be highly suggestive of coccidiosis but the disease should be confirmed. Faecal samples can be taken to identify a significant oocyst count of

pathogenic species of coccidia. Samples with a high faecal coccidia count alone may not be significant and they should be speciated to confirm the oocysts are the harmful type at an accredited laboratory. Many lambs have a very high count of harmless oocysts.

### Treatment

- Oral solution or bolus of Sulfadiazine400 mg Trimethoprim80 mg at 1 g per 10 kg body weight for 4 - 7 days.
- Extra label use of the poultry product Bayticol® that contains Toltrazuril is effective as a single dose at 1 ml/2.5 kg. It is reported to be very effective at reducing oocyte shedding as the drug is effective at all intracellular developmental stages of the parasite. Poultry products containing Amprolium have also been used effectively in goats.

### Control

- Good hygiene is essential in livestock bomas especially where lambing and kidding occurs.
- Feeding troughs should be elevated or located outside the sleeping areas
- Ensure new born kids feed on colostrum as it provides immunity to coccidiosis for the first several weeks of the neonate's life.
- If possible, separate young animals from older animals
- Stress contributes to outbreaks of coccidiosis. Sheep and goats should be handled minimally and handled calmly and gently. It is particularly important to minimize stress at weaning.

Figure 120 Eimeria species unsporulated oocysts in goat kid faecal sample



Picture source: https://www.boergoatprofitsguide. com/goat-care-for-beginners/

> Figure 121 Small intestine of a goat with multiple raised nodules consistent with coccidiosis.



Picture source: https://tvmdl.tamu. edu/2020/06/15/pathologicdiagnosis-of-coccidiosis-in-goats/

## Goat Diseases: Bacterial diseases

## 7.11 Contagious Caprine Pleuropneumonia (CCPP) and Ovine Mycoplasma Pneumonia

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Sombes Ree	Sombes Ree	Ilkibei	Lkipei	Loukoi	Louke Ayiet

\*\*Notifiable disease and should be reported to national and county DVS through KABS.

### Case definition and Epidemiology

- CCPP is a severe highly contagious fatal disease that affects the respiratory tract.
- In immune naive herds the morbidity rate may reach 100% and the mortality rate can be as high as 80%. CCPP causes major economic losses in East Africa and the Middle East, where it is endemic
- During the only confirmed outbreak in wild ruminants, the morbidity rate was 100% in wild goats and 83% in Nubian ibex. The mortality rates in these two species were 82% and 58%, respectively.
- Goats are the primary hosts but Sheep may be affected, during CCPP outbreaks affecting mixed goat and sheep herds the bacteria has been isolated from healthy sheep, and their role as a possible reservoir must be considered.
- CCPP was first reported in Europe in Thrace, Turkey, in 2002, but it did not spread to neighbouring countries of Greece and Bulgaria.

### Causative agent

- CCPP is caused by bacterium Mycoplasma capricolum subspecies capripneumoniae (Mccp). Formerly known as Mycoplasma sp. type F-38.
- · Genetic studies have grouped Mccp isolates

into two major clusters (2 or 3) depending on the geographic region.

- Mccp is closely related to M. capricolum subsp. capricolum and more distantly related to other members of the "Mycoplasma mycoides cluster" such as M. mycoides subsp. capri or M. leachii. Mccp lesions are only confined to the thoracic cavity. However, mycoplasmas of the mycoides cluster have thoracic as well as prominent lesions in other organs and/or parts of the body.
- Mccp is inactivated by most of the routinely used disinfectants for example phenol (1%) inactivates it in 3 minutes. The bacteria cannot survive for long in the external environment. on average it only survives outside the host for up to 3 days in tropical areas.

### Morbidity and Mortality rates

- In Marsabit county, livestock keeper in the year2022, estimated an apparent annual morbidity rate of 18% with a 9% mortality and 41% case fatality rate.
- The disease is endemic in Marsabit and is most common during the long rainy season.

Season	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Long Rains	Gann	Gann	Guuh	Lng'erng'erwa	Akiru Napolan	IR Gutoka
(April- June)					or Akiporo	

### Transmission

- Transmitted during close contact and by inhalation of respiratory droplets.
- Chronic carriers may exist, but this remains unproven. Some outbreaks have occurred in endemic areas when apparently healthy goats were introduced into flocks.
- Outbreaks of the disease often occur after heavy rains (e.g. after the monsoons in India), after cold spells or after transportation over long distances. This may be because recovered carrier animals shed the infectious agent after the stress of sudden climatic or environmental changes.

### **Clinical signs**

• The incubation period is 6 to 10 days. Peracutely affected goats can die within one to three days with minimal clinical signs. Acute symptoms include fever, lethargy, violent coughing, extended necks, laboured breathing, loss of appetite and abortions.

### Control

- Treatment with Tylosin at 10 mg/kg/day, IM, for 3 days, has been effective, as has oxytetracycline (15 mg/kg). Tylosin dosage is 0.3 – 0.4 mL per 10 kg bodyweight for 3 to 5 days. On the first day two injections can be given 12 hours apart.
- Vaccines used to control disease in Kenya, KVEVAPI CAPRIVAX that is administered to animals over 3 months of age via subcutaneous injection at the rate of 1ml per animal. Revaccination should be carried out every 6 months



Figure 122 Loss of body condition (Left) and open mouth breathing in goats affected with CCPP

Picture source: https://www.semanticscholar.org/paper/Contagiouscaprine-pleuropneumonia-in-Beetal-goats-Hussain-Auon/ a2ffc6fb72ef9c23e40087760efade8e82d7ec85/figure/1



### Figure 123

CCPP lesions showing abundant fibrin on lung lobes (Left) and large quantities of pleural fluid and "port wine"-coloured hepatized lung lobes in a goat

### Picture source:

https://www.msdvetmanual.com/respiratory-system/ respiratory-diseases-of-sheep-and-goats/contagious-caprinepleuropneumonia

### Further Reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/ 2. OIE (2017). Technical Disease Card. https://www.woah.org/app/ uploads/2021/03/contagious-caprine-pleuro.pdf

## 7.12 Mastitis in Goats

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Budha	Budha	Budha	Ntiroto E Lkii	Edeke- Aeket	Bash Oto

### Case definition, Aetiology and Epidemiology

- Mastitis in goats is an acute to chronic inflammation of the udder mainly caused by bacterial infection. Trauma may be a predisposing factor to bacterial mastitis.
- The bacterial organisms that infect the udder of does are similar to those in cows. Coagulasenegative staphylococci are generally the most prevalent and can cause persistent infections that result in increased cell counts and lowgrade mastitis with some recurring clinical episodes. The level of infection and incidence of mastitis due to Staphylococcus aureus tends to be low (<5%) but can result in persistent infections that do not generally respond

to therapy. Streptococcal intramammary infections can occur in both subclinical and clinical cases but are less frequent than in cattle. Streptococcus agalactiae is not a common mastitis pathogen in does.

- Mycoplasma infections, primarily M mycoides (large colony type) and M putrefaciens, sometimes cause serious outbreaks of mastitis in goats (Contagious Agalactia). The latter also causes septicaemia, polyarthritis, pneumonia, and encephalitis, together with serious disease and mortality in suckling kids. M capricolum has also been reported to cause severe mastitis in goats and infection in kids. Does usually recover after 4 weeks.
- · Gram-negative organisms cause intermittent

infections that may be severe but are usually self-limiting. Trueperella (formerly Arcanobacterium) pyogenes sometimes produces multiple, nodular abscesses in the udder and loss in the functionality of the udder in does.

- Does can also signs of mastitis from caprine arthritis and encephalitis and ovine progressive pneumonia secondary to systemic infection. Agalactia is common due to the hardening of the udder from fibrosis.
- Does that have aborted and have uterine infection should be treated immediately as the same microorganisms that cause abortion in does also cause mastitis.

### Diagnosis

• Diagnosis, control, and treatment of bacterial mastitis in does are similar to those in cows. However, monitoring subclinical mastitis with somatic cell counts (SCCs) in does is difficult because of poor discrimination between infected and non-infected animals, especially in the later stages of lactation. This is partially because a of a higher proportion of epithelial cells in caprine milk than in bovine milk. As lactation progresses, shedding of epithelial cells into milk increases; thus, SCCs >1,000,000 cells/mL are common in uninfected does in late lactation.

### Treatment

- The treatment should be based on the results of the microbiologic culture obtained from milk samples. This is not practical in Marsabit setting.
- Before treatment the affected udder half should be dried off by stopping the kid from suckling the half or stop milking and infusing

Picture source: https://www.premier1supplies.com/w/mastitiscontrol-program/ commercially intramammary infusion of 2% chlorhexidine solution into affected half twice a day for 5 to 10 days. This many not also be practical in Marsabit setting.

- Intramammary infusions for dairy cows also work in goats but one should observe for developing irritation as the drug concentration may be strong for the doe's udder tissue.
- Most practitioners prefer systemic antibiotics combined with anti-inflammatory drugs like glucocorticoids. Correct dosage and duration as well as withdrawal period should be observed. Antibiotics like cloxacillin, amoxicillin plus clavulanic acid, erythromycin, and tetracycline have been recommended to treat mastitis in goats. However, cure rates vary from animal to animal and according to the severity of the case.
- Glucocorticoids, can be administered early in the course of disease. Administration of dexamethasone in the mammary gland has been reported to reduce swelling.
- Treatment during the dry-off period is an efficient method for the cure of subclinical mastitis

### Control

- Proper milking procedures and good environmental sanitation are needed to reduce the prevalence and spread of infection.
- Chronically infected does should be culled, including does with M mycoides infections and those that do not recover from M putrefaciens or M capricolum infections.
- Kidding pens must be disinfected and bedding removed daily.
- When purchasing animals palpate mammary glands to note if the consistency is normal.



## Chapter 8: Chicken diseases

### Viral Chicken Borana Gabra Rendille Samburu Turkana Daasanach Disease New Castle Disease Kotes Kotes Argabi Gurunkata Longendu Uu Gileba (NCD) Luku Korguma Keiri Fowl Pox Nariri Ee Finno Finno Nyinyilo Ti Ngajemei Verte Luge Luku Nkokon Infectious Lchama Lee Qufa Qufa Argabi Ipiyorit-Ufute Bronchitis (IB) Luku Nkokon Akienga Protozoal Chicken Borana Gabra Rendille Samburu Turkana Daasanach Disease Coccidiosis Tumma Tumma Hari Dige Ngiriata E Aremorit-Geerbura Luku Luku Lodo Keisirua Ng'aakot Rendille Parasitic Chicken Borana Gabra Samburu Turkana Daasanach Disease Helminths Minii Minii Deyaah Ntumwa Ngirtan / Chade Ngipelei

### Table 25: Local dialect name of priority chicken diseases in Marsabit county

### Table 26: Local dialect name of clinical signs for priority chicken diseases

Clinical Signs	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dropping Wings-	Khool Buft	Khool	Keilili	Barbare	Echakunit -	Kuol Hi
Chicken		Buft	Nkaipupo	Lugdha	Ngabeben	Reereme
Snoring/	Inkhorift	Inkhorift	Keirung	Ahuricha	Akingorare	Him Hi Chies
Respiratory Rales-						
Chicken						
Twisted Neck-	Qacher	Qacher	Kekordo	Kekordo	Kekordo	Luut Hi Gaa
Chicken	Thabsit	Thabsit	Murt	Murt	Murt	Gernete
Ruffled Feathers	Inshil	Inshil	Keitachoo	Tudube	Epuriarit	Dhuur Hi Gaa
	Khoft	Khoft	Nkopir	Tolosata		Firay
Paralysis- Chicken	Nafat Iraat	Nafat Iraat	Keikanga	Ahanjawa	Etergege	Heljeny
	Dhue	Dhue				
Loss Of Feathers	Kolat Luqa	Kolat	Kodoyio	Tudube	Lochud /	Balaam Hi Gaa
		Luqa	Nkopir	Abutamte	Echudanu	Okode

### Table 27: Medicinal plants and traditional treatment practices identified by livestock keepers

Ethnoveterinary Practice	Target Chicken disease
Aloe species	IB, Helminthiasis, NCD, Coccidiosis, Fowl pox
Old engine oil	Fowl pox, Ectoparasite infestation
Milk	IB, Helminthiasis
Ground pepper	IB, NCD, Helminthiasis, Coccidiosis
Detergent (Omo)	Coccidiosis, Ectoparasite infestation
	10

NB: Text in red highlights practices that go against animal welfare principles

### Table 28: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority chicken diseases

Type of VMP	Active ingredient	Target diseases based on the 7 informant group discussions
Antibiotic	doxycycline capsules indicated for human use and injectable oxytetracycline given orally in water	IB, NCD, Coccidiosis
Anthelmintic	Piperazine citrate / Pyrantel pamoate/ Albendazole/ Levamisole	Helminthiasis
Acaricides	Sevin® (Carbaryl	Ectoparasite infestation, Fowl pox

### Table 29: Ethnoveterinary Control Practices for priority chicken diseases

Livestock keepers in Marsabit county identified 4 control strategies that can be implemented to control priority diseases in chicken. These were;

- 1. Isolating sick birds and new birds purchased;
- 2. Relocating the chicken house or burning it if there is a heavy ectoparasite infestation or high mortality of birds as seen in New castle disease and Infectious bronchitis;
- 3. Regular removal of chicken droppings and application of wood ash to prevent helminthiasis, coccidiosis and ectoparasite;
- 4. Prompt carcass disposal;

## Chicken Diseases: Viral diseases

## 8.1 New Castle Disease (ND)

Other names- Avian pneumoencephalitis, Ranikhet disease, Fowl pest, Fowl plague

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Kotes	Kotes	Argabi Luku	Gurunkata	Longendu	Uu Gileba
			Korguma Keiri		

### Case definition

- ND is a highly contagious and fatal viral disease affecting over 250 bird species with chicken being the most susceptible.
- ND is a multisystemic disease involving the respiratory, neurological, reproductive and gastrointestinal body systems.

### Distribution (Epidemiology)

- Endemic globally, sero-surveillance studies in Kenya have shown a Morbidity rate of 5 to 17.8% and mortality rate of 50-100%.
- Based on Marsabit county livestock keepers' perception (2022) ND apparent annual Morbidity rate is 11% with a Mortality rate of 10% and Case fatality rate of 98%.
- ND in Marsabit county occurs throughout the year with most outbreaks reported during the long dry season between the months of July and September.
- ND, is the most significant disease of poultry in Kenya.

### Age susceptibility

• All ages, but young birds have the highest mortality rates.

### Aetiology

• Virulent strains of avian orthoavulavirus 1 (AOaV-1), causative agent formerly known as

avian paramyxovirus1 (APMV 1).

- It is a single-stranded RNA virus from Paramyxoviridae family.
- AOaV-1 has 11 serotypes
- Original classification was based on virulence; Lentogenic (low virulence viruses); Mesogenic (moderate virulence), and Velogenic (highly virulent) and tissue tropism (predilection). The virus exhibits tissue tropism where some strains only attack the nervous system, others the respiratory, and others the digestive systems.
- In the new classification Velogens and mesogens strains are now called virulent NDV (vNDV) they cause Newcastle disease and are WOAH reportable infections.
- Infections with lentogens are now called low virulence NDV (loNDV) and are widely used as live vaccines and are not reportable.

### Mode of transmission

- Main risk factors for ND outbreaks are introduction of new birds into the flock and purchase of birds from markets.
- AOaV-1 is stable in nature and remains infectious for weeks at low temperatures, and if protected by bird's feathers and within eggs survives for over 250 days.
- Direct transmission is via inhalation or ingestion (faecal/oral route). Birds shed virus

in faeces and respiratory secretions. Reservoir and carrier hosts are a source of direct infection as they shed the virus,

• Indirect transmission is via fomites. Virus survival is prolonged by the presence of faeces, for example on eggshells.

### **Clinical signs and Pathophysiology**

- Incubation period is 2 to 12 days.
- There is sudden death and high mortality within 24-48 hours
- Birds that survive are drowsy or extremely depressed with most birds are seen sitting on their back hock joints.
- Birds have ruffled feathers are anorexic and have a sudden drop in egg production.
- The most common gastro intestinal sign is greenish to yellowish diarrhoea.
- The main respiratory signs are dyspnoea (gasping) with rales (gurgling or rattling sounds), sneezing, coughing, nasal and ocular watery discharge. Swelling of head and neck tissues.
- Nervous signs include muscular tremors (twitching) of head and neck, partial paralysis of wings and legs, circling, spasms and torticollis (twisted neck) and opisthotonus (star gazing).
- Recovered birds beyond may have permanent nervous (tremor) or permanent reproductive impairment.
- Clinical signs may be seen in birds vaccinated with the live vaccine where they have a marked drop in egg production, some birds may produce eggs that are misshapen, abnormally pale, rough, or thin-shelled with watery albumin.

### Post-mortem Gross lesions

- Swelling of periorbital area or entire head.
- Oedema of the interstitial or peritracheal tissue of the neck especially at the thoracic inlet.
- Diphtheritic membranes in the oropharynx, trachea and oesophagus.
- Pulmonary congestion and oedema.
- Pinpoint haemorrhages of the proventriculus gland.

- Multifocal, necrotic haemorrhagic areas on the mucosal surface of intestines especially at lymphoid foci such as the gut-associated lymphoid tissues (GALT) of cecal, tonsils and Peyer's patches.
- Though not pathognomonic, ulceration/ necrosis of Peyer's patches is suggestive of Newcastle disease.

### Diagnosis

- Tentative/Presumptive- clinical signs and PM gross lesions but are not specific to ND.
- Laboratory investigation is needed.
- Samples collection should be from recently dead birds (less than 8 hours) or moribund birds that have been killed humanely.
- Samples- Tracheal, oropharyngeal and cloacal swabs or faeces obtained from live birds or pooled organ samples taken from dead birds. Contact laboratory to get advice on what transport media to use.
- Confirmatory diagnosis- antibody or viral antigen detection using ELISA, or other serological assays like Haemagglutination (HA) and Haemagglutination inhibition (HI) or molecular virus identification using RT-PCR for the detection and typing of virus.

### Public health risk

• Zoonotic disease - Causes conjunctivitis in humans, that is mild and self-limiting.

### Differential Diagnosis (DDx)

- Other viral diseases highly pathogenic avian influenza, laryngotracheitis, fowl pox (diphtheritic form) and infectious bronchitis.
- Bacterial diseases: fowl cholera, psittacosis (Chlamydiosis), Mycoplasmosis, salmonellosis
- Fungal diseases like Aspergillosis
- Management errors such as deprivation of water poor feed and air quality.

### Treatment

- Recovered birds may be immune but remain carriers that shed virus when stressed.
- No treatment as it is a viral disease
- Infected birds should be slaughtered.

### Prevention/Control

- Humane destruction of all infected birds.
- Thorough cleaning and disinfection of houses/ burning house
- Proper carcass disposal
- Endo and Ecto parasite control
- Allow 21 days without poultry before restocking
- Isolate new birds for 1-week
- Regular vaccination

### Vaccination Schedule

- Locally available live attenuated vaccine (AVIVAX-L<sup>™</sup> and AVIVAX-F<sup>™</sup>) is used for vaccinations against AOaV-1 in Kenya.
- Thermostable I-2 vaccine (derived from lentogenic LaSota/F strains) is administered in three doses (as eye drops): first dose 4 days to 3 weeks old, second dose at 4–8 weeks and the last dose at the age of 8–18 weeks.

- To overcome the problem of continuous ND outbreaks even in vaccinated flocks, some commercial farmers combine live and/or inactivated vaccines, which are based on an older AOav-1 genotypes that has been reported to be inefficient in reducing viral replication and shedding.
- Kenchic has introduced a hatchery vaccination in all day-old chicks against Gumboro, Newcastle and Infectious Bronchitis diseases. The TRANSMUNE vaccination against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases.

### **Reporting criteria**

- Notifiable disease- WOAH listed
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS)



Figure 125 Recap: Gross anatomy of the chicken



Figure 126 Clinical signs of Newcastle disease (clockwise order): 1) chicken showing severe depression, 2) torticollis 3) greenish watery diarrhoea and 4) death





Figure 128 Chicken proventriculus and ventriculus. Severe, acute multifocal mucosal haemorrhages



# Figure 129 Chicken, cecal tonsils.

Haemorrhage of the cecal tonsils visible through the serosa (Left) and necrosis of cecal tonsil (Right)

## 8.2 Infectious Bronchitis (IB)

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Qufa	Qufa	Argabi Luku	Lchama Lee	Ipiyorit- Akienga	Ufute
			Nkokon		

### Case definition:

- acute, highly contagious upper respiratory tract disease. characterised by coughing, sneezing and tracheal rales.
- In laying hens, causes decreased egg production and quality.
- Some virus strains cause nephritis (Kidney inflammation) and cystic oviduct syndrome (false layer syndrome).
- Causes clinical disease in chicken only and no other bird species.
- Second most important disease of chicken after New Castle Disease (ND). ND causes high mortality while Infectious Bronchitis (IB) causes low mortality but high production losses.

### Distribution (Epidemiology)

- Worldwide
- IB virus (IBV) has many antigenic types, some IBV types are widespread, others are regional.
- Severity of disease and body systems involved influenced by: strain of the virus, age, immune status, and diet of the chicken and exposure to cold stress.
- Co-infection with Mycoplasma (M) gallisepticum, M synoviae, Escherichia coli, and/or Avibacterium paragallinarum worsen infections.
- Morbidity rate in flocks with no immunity is 100% but morbidity is low 5% but can be as

high as 60% due to bacterial co-infections.

- Based on Marsabit county livestock keepers' perception (2022). IB apparent annual Morbidity rate is 6% with a Mortality rate of 4% and Case fatality rate of 73%.
- IB in Marsabit county occurs throughout the year with most outbreaks reported during the long dry season between the months of July and September.

### Age susceptibility

- All ages of birds.
- Young chicks more susceptible.

### Causative agent (Aetiology)

- IBV is an avian coronavirus that is an enveloped and single-stranded RNA virus with characteristic spike-like projections on the surface of its envelope.
- Variation of the viral spike protein gives rise to multiple strains of the virus, which may vary regionally.
- IBV quickly mutates and requires constant surveillance to identify IBV types circulating in a specific region.
- Different antigenic virus strains do not crossprotect.
- Recent discoveries of IBV and IBV-like strains in other bird species such as geese, ducks, and pigeons. May play a role in the spread of IBV to chickens.

### Mode of transmission

- Naturally infected chickens and those vaccinated with live IBV may shed virus intermittently for up to 20 weeks.
- Virus is shed via aerosol droplets and faeces. IBV can persist in the bird's intestinal tract for several weeks or months
- Frequency of airborne transmission is not known but studies have shown of transmission occurring in commercial farms over 1 kilometre apart.
- Incubation period is 24 to 48 hours, peak virus excretion from the respiratory tract occurs for 3 to 5 days after infection.
- Direct and indirect transmission via aerosol droplets, ingestion of contaminated feed and water, and contact with contaminated equipment and clothing (fomites).

### Clinical signs and Pathophysiology

- Symptoms manifest 18 to 24 hours' postexposure and may last for 2-3 weeks.
- Conjunctivitis, coughing, sneezing, tracheal rales and facial swelling.
- Respiratory distress is due to increased mucous in the trachea.
- In addition to above signs, adult birds, have a sharp decrease in egg production.
- Damage to the reproductive tract causes production of misshapen, wrinkled, thin soft eggs of poor internal quality for several weeks.
- Infection in birds that have not started laying (pullets) can cause false layer syndrome as the oviduct is damaged but ovaries are intact. The ovaries continue to produce and the yolks accumulate in the abdomen of the animal. With time, the non-functional oviduct fills with clear fluid and distends (cystic).
- In addition, accumulation of yolks, causes abdominal distention and forces the birds to stand upright (penguin-like posture).

### Post-mortem Gross lesions

- Trachea, sinuses, and nasal passages contain serous, catarrhal, or caseous exudates,
- Air sacs are cloudy and thickened. If complicated by infection with E coli, there may be caseous airsacculitis, perihepatitis, and pericarditis.
- Female birds infected when young have cystic oviducts, whereas those infected while in lay have an oviduct of reduced weight and length and ovaries with regressed follicles.
- Infection with nephropathogenic IBV strains cause swollen, pale kidneys, with the tubules and ureters distended with urates. Kidneys may be atrophied.

### Diagnosis

- Tentative/Presumptive- clinical signs and clinical history
- Laboratory investigation needed due to large number of diseases that show similar symptoms.
- Samples collection should be from recently dead birds (less than 8 hours), moribund birds that have been killed humanely or live birds.
- Samples for acute respiratory disease include swabs from the upper respiratory tract of live birds or tracheal and lung tissues from dead birds.
- Samples from nephritis or egg production problems should be tissue samples from the kidneys or oviduct, respectively in addition to respiratory specimens.
- Confirmatory diagnosis- antibody or viral antigen detection.
- Laboratory will inform if they can Conduct rising antibody analysis using ELISA or Molecular virus identification using RT-PCR.
- There is need for sustained molecular surveillance so as to ensure early detection of novel strains of IBV.

### Public health risk

• No data indicating zoonotic potential.

### Differential Diagnosis (DDx)

- Viral diseases: Newcastle Disease, Infectious laryngotracheitis, infectious coryza.
- Bacterial diseases: Mycoplasma gallisepticum (Mycoplasmosis)
- Management errors such as deprivation of water, poor feed and air quality.

### Treatment

- None as it is viral.
- Antibiotics can be used if there is evidence of secondary bacterial infection that manifests as increasing mortality rates with cloudy air sacs, peri hepatitis and pericarditis.

### Prevention/Control

- Attenuated live and killed vaccines are available, but different antigenic types of the avian coronavirus causing the disease do not cross-protect, complicating control efforts.
- No medication alters the course of IBV infection, as it is a viral disease. Antimicrobial therapy may reduce mortalities caused by complicating bacterial infections.
- In cold weather, increasing the ambient temperature may reduce mortalities, and reducing the protein concentrations in feed and providing electrolytes in drinking water may assist in outbreaks caused by nephropathogenic strains.
- The live-attenuated vaccines used for immunization may produce mild respiratory signs. These vaccines are initially given to 1- to 14-day-old chicks by spray, drinking water, or eye drop, and birds are commonly revaccinated approximately 2 weeks after the initial vaccination.
- Revaccination with a different serotype can induce broader protection. Attenuated or adjuvant inactivated vaccines can be used in breeders and layers to prevent egg production

losses as well as to pass protective maternal antibodies to progeny.

- There are many distinct types of IBV, and new or variant types, which are not fully controlled by existing vaccines, are identified relatively frequently.
- Variant viruses historically arise from mutations accumulating over time as the virus replicates (genetic drift). However, recombination can occur in coronaviruses and may result in unique viruses that may or may not cause disease.
- Selection of vaccines should be based on knowledge of the most prevalent virus type(s) in the area. The correlation between IBV type and protection is imperfect, and selection of the most appropriate vaccine, or combination of vaccines, may require experimental assessment in vivo.

### Vaccination Schedule

- Kenchic has introduced hatchery vaccination in all day-old broilers against Gumboro, Newcastle and Infectious Bronchitis diseases.
- TRANSMUNE vaccination against Gumboro and VITABRON against Newcastle and Infectious Bronchitis diseases in day-old chicks at the hatchery. The farmer will only do one single Newcastle disease vaccination in the Broiler farm at day 14 instead of 2 vaccinations as done previously

### Reporting criteria

- Notifiable disease- WOAH listed
- Report to county and national DVS using ND1 form and input outbreak event in the mobile surveillance app (Kenya Animal Bio Surveillance System (KABS)

### References

- Al-Rasheed, M., Ball, C. & Ganapathy, K. (2021). Route of infectious bronchitis virus vaccination determines the type and magnitude of immune responses in table egg laying hens. Vet Res 52, 139 (2021). https://doi.org/10.1186/s13567-021-01008-7
- Najimudeen, Shahnas, Mohamed S. H. Hassan, Susan C. Cork, and Mohamed F. Abdul-Careem. (2020). Infectious Bronchitis Coronavirus Infection in Chickens: Multiple System Disease with Immune Suppression. Pathogens 9, no. 10: 779. https://doi.org/10.3390/pathogens9100779





Picture source: https://www.researchgate.net/profile/Tofazzal-Md-Rakib



## Figure 131

Post-mortem lesion in different organs of chicken caused by infectious bronchitis virus

A) Congested trachea;
B) Swollen, pale or marbled kidneys with urate deposits in the tubules;
C) Visceral gout in chicken with wide spread deposits of uric acid on serosal surface of the body;
D) Misshappen ova and oviduct

Picture source: https://www.researchgate.net/profile/ Tofazzal-Md-Rakib

## 8.3 Fowl pox

Other name: Avian pox. There are three strains of virus that cause avian pox: Fowl pox, pigeon pox and canary pox.

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Finno	Finno	Nyinyilo Ti Luku	Nariri Ee Nkokon	Ngajemei	Verte Luge

### Causative agent:

An avipoxvirus. The virus is resistant and can survive in the environment for long periods in dried scabs.

### Transmission:

- · Direct contact through abrasions of the skin
- Aerosol infection from recovering birds that have skin lesions (scabs) and sharing same houses
- Indirectly through mechanical vectors like mosquitoes, other biting insects or individuals handling one bird after the other for immunization or other reasons.
- In Marsabit county the estimated apparent morbidity rate is 5% with a mortality rate of 2% and case fatality rate of 50%.
- The disease in the county is most common during the long dry season.

### Main clinical signs:

There are two forms of the disease:

 Skin or cutaneous form (dry pox): this is characterized by nodular wart-like lesions on various parts of the un-feathered skin of chicken's head, neck, comb, wattles, eyelids, legs, and feet. In some cases, lesions are limited to the feet and legs. The lesion is initially a raised, blanched, nodular area that enlarges, becomes yellowish, and progresses to a thick, dark scab. Localization of lesions around the nostrils may cause nasal discharge. Cutaneous lesions on the eyelids may cause complete closure of one or both eyes. Only a few birds develop cutaneous lesions at one time. Multiple lesions usually develop and often coalesce. Lesions in various stages of development may be found on the same bird.

2. Diphtheritic form (wet form): This is characterized by small white nodules in upper respiratory and digestive tracts. These nodules merge together to form raised-yellow white cheesy patches. These lesions cause difficulty in breathing. It has been found that most cases of diphtheritic fowl pox are characterized by the formation of massive yellow cheesy necrotic masses in the larynx and adjacent trachea The bird in such cases die suddenly from asphyxiation (lack of oxygen).

### Post mortem Findings:

• Wart-like lesions of the head particularly of the comb and around the eyes, or yellow cheesy lesions in the larynx and adjacent trachea on post-mortem, are diagnostic of fowl pox.

### **Differential Diagnosis:**

• Infectious laryngotracheitis, necrotic dermatitis, leg mites and thrush (Candidiasis) and canker (Trichomonas gallinae).

### Diagnosis:

• Tentative diagnosis is based on flock history and presence of lesions.

### Prevention and control:

• Vaccination effectively prevents the disease and may limit spread within actively infected

flocks. In high-risk areas, vaccination with an attenuated vaccine of cell-culture origin in the first few weeks of life and revaccination at 12–16 weeks is often sufficient. Vaccinated birds should be examined 1 week later for swelling and scab formation ("take") at the site of vaccination. Absence of "take" means the birds are not protected and should be vaccinated again.



Figure 132 Cutaneous form of fowl pox showing (white arrows) scab-like lesions on the combs of chicken



Figure 133 Diphtheric form of fowl pox infection showing yellow cheesy oral lesions

## Chicken Diseases: Protozoal diseases

## 8.4 Coccidiosis

### **Other Name: Coxy**

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Tumma Luku	Tumma Luku	Hari Dige	Ngiriata E Lodo, Keisirua	Aremorit- Ng'aakot	Geerbura

### Causative agent:

Protozoan parasite of the Eimeria species that infect various sites in the intestines where it multiplies causing damage. There are about nine species that infect chicken and about seven that infect turkeys. Eimeria species are considered host specific meaning those that infect chicken do not infect turkeys and vice versa. Eimeria life cycle takes 7 to 9 days.

### Transmission:

- The sporulated oocyst is the infective stage of the parasite and is shed by both Infected and recovered birds.
- Chicken get infected when they ingest the infective form of the oocysts (sporulated oocysts)
- Oocysts can be spread mechanically by animals, wild birds, insects, clothing and human footwear, contaminated equipment, or in some cases, by wind/dust while spreading poultry-house dust and litter over short distances.

### Main Clinical Signs:

Coccidiosis occurs in two forms: Large intestine/ Caecal coccidiosis is caused by Eimeria tenella. Disease is severe and signs include:

- Bloody diarrhoea
- · Reduced weight gain
- · High mortality

• Unthriftiness and emaciation

Small Intestinal coccidiosis is caused by Eimeria necatrix, E. acervulina, E. brunetti, E. maxima,

- E. mitis, E. mivati, E. necatrix and E. praecox.
- Severe weight loss
- Droppings contain blood
- High morbidity.
- Decreased egg production
- · Depressed feed and water consumption

### Post mortem Findings:

The main lesions are bloody intestinal content, enteritis and mottled intestinal wall. The lesions are different depending on the species as follows:

- E. acervuline: lesions include numerous whitish, oval or transverse patches in the upper half of the small intestine, which may be easily distinguished on gross examination.
- E. mivati: lesions develop in the anterior part of the small intestine. In severe infections, E. mivati may cause reddening of the duodenum that may be visible through the serosa of the distal duodenum and proximal jejunum.
- E. necatrix: major lesions are found in the anterior and middle portions of the small intestines. Small white spots, usually intermingled with rounded, bright- or dullred spots of various sizes, can be seen on the serosal surface. This appearance is sometimes described as "salt and pepper." The white spots are diagnostic for E. necatrix if clumps of large schizonts can be demonstrated

microscopically.

- E. maxima: develops in the small intestine, where it causes dilatation and thickening of the wall; petechial haemorrhage; and a reddish, orange, or pink viscous mucous exudate and fluid. The exterior of the midgut often has numerous whitish pinpoint foci, and the area may appear engorged.
- E. tenella: haemorrhagic typhlitis (inflammation of the cecum). Recognized by accumulation of blood in the ceca and by bloody droppings. There may be accumulations of clotted blood, tissue debris, and oocysts in ceca of birds surviving the acute stage.

### Diagnosis:

- Tentative diagnosis is based on flock history, clinical signs and lesions.
- Post mortem examination within one hour of death before the post mortem changes take effect in the intestinal mucosa.
- A definite diagnosis is made by observation of Eimeria Oocysts in the laboratory.



### Figure 134

Pictorial description of sites parasitized by Eimeria tenella (A), E. necatrix (B), E acervuline (C), E brunetti (D), E maxima (E), and E mitis (F). (Illustration by Dr. Gheorghe Constantinescu).

- Note that the finding of a few oocysts by microscopic examination of smears from the intestine indicates the presence of infection, but not a definite diagnosis of clinical coccidiosis.
- Coccidia and mild lesions are present in the intestines of birds 3–6 weeks old in most flocks. Diagnosis should be based on finding lesions that confirm the microscopic findings.

### Prevention and Control:

- Prevention is mainly with anti-coccidia drugs, good hygiene, disinfection and sanitation of premises and providing clean feed and water.
- Early treatment during outbreaks will prevent large scale bird losses.
- Use of antibiotics combined by supplemental provision of vitamins A and K are incorporated in the feed for treatment. Medication used include: amprolium, sulphonamides and toltrazuril.
- A species-specific immunity develops after natural infection, the degree of which largely depends on the extent of infection and the number of reinfections.



Figure 135 Coagulative necrosis and sloughing of intestinal mucosa as a result of severe infection with Eimeria brunetti

## Chicken Diseases: Parasitic diseases

## 8.5 Helminthiasis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Minii	Minii	Deyaah	Ntumwa	Ngirtan / Ngipelei	Chade

### Causative agents:

• Mainly nematodes (roundworms) and cestodes (tapeworms) Ascarids are the main round worms that infect chicken. Gapeworms are found in the trachea, capillaria in the intesines, heterakis in the ceca, trichostrongyles and tapeworms in the intestines while gizzard worms in the gizzard.

### Transmission:

- Direct transmission from bird to bird by ingestion of infective eggs or larvae.
- Indirectly through an intermediate host (insect, snail or slug).
- In Marsabit county the apparent morbidity rate due to helminths is 5% with a mortality rate of 3% and case fatality rate of 65%.
- Helminthiasis cases are reported throughout the year with no seasonal prevalence.

### Main Clinical Signs:

- · General unthriftiness and inactivity
- Depressed appetite
- Suppressed growth
- · In severe cases, may result in death
- Gapeworms cause chickens to gasp with neck extende

### Diagnosis:

• Detection of worm eggs by faecal flotation allows for the reliable confirmation of the

presence of worms. Note that absence of eggs in a sample does not necessarily mean worms are absent.

### Treatment:

- Fenbendazole is effective against nematodes when given in drinking water at 125 mg/litre for 5 days. Do not administer during moult as it interferes with feather re-growth
- Albendazole can be administered as a single oral suspension at a dose of 5 mg/kg bird weight. This is reported to be effective against A galli, H gallinarum, and C obsignata. It has also has been reported effective against cestodes if administered at 20 mg/kg
- Levamisole is reported to be effective against A dissimilis, H gallinarum, and C obsignata if given in drinking water at 0.03%–0.06%.
- Other medications such as piperazine citrate are available in the market and should be used according to manufacturer's instructions.

To reduce the potential spread of resistance, treatment should be limited to birds with severe infection that show clinical signs of disease.

### **Prevention and Control:**

• Improvement of management and sanitation is key. In free range birds, move the chicken, although the benefit that may result will be of short duration.



Figure 136 Chicken intestinal tract impacted by Ascarid worms



Figure 137 Life cycle of the large round worm of chicken Ascarid galli

Picture source: https://core.ac.uk/download/pdf/211564736.pdf The table below summarises important helminth parasites pathogenicity in chicken

Parasite	Intermediate host or lifecycle	Organ infected	Pathogenicity
Nematodes:			
Ascaridia galli	Direct	Small intestine	Moderate
Capillaria annulata (also known as Eucoleus annulata)	Earthworm	Esophagus, crop	Moderate to severe
Capillaria caudinflata (also known as Aonchotheca caudinflata)	Earthworms	Small intestine	Moderate to severe
Capillaria contorta (also known as Eucoleus contorta)	None or earthworms	Mouth, esophagus, crop	Severe
Capillaria obsignata (also known as Baruscapillaria obsignata)	Direct	Small intestine, ceca	Severe
Cheilospirura hamulosa	Grasshoppers, beetles	Gizzard	Moderate
Dispharynx nasuta	Sowbugs	Proventriculus	Moderate to severe
Gongylonema ingluvicola	Beetles, cockroaches	Crop, esophagus, proventriculus	Mild
Heterakis gallinarum	Direct	Ceca	Mild, but transmits agent of histomoniasis
Oxyspirura mansoni	Cockroaches	Eye	Moderate
Strongyloides avium	Direct	Ceca	Moderate
Subulura brumpti	Earwigs, grasshoppers, beetles, cockroaches	Ceca	Mild
Syngamus trachea	None or earthworm	Trachea	Severe
Tetrameres americana	Grasshoppers, cockroaches	Proventriculus	Moderate to severe
Trichostrongylus tenuis	Direct	Ceca	Severe
Cestodes:			
Choanotaenia infundibulum	House flies	Upper intestine	Moderate
Davainea proglottina	Slugs, snails	Duodenum	Severe
Raillietina cesticillus	Beetles	Duodenum, jejunum	Mild
Raillietina echinobothrida	Ants	Lower intestine	Severe, nodules
Raillietina tetragona	Ants	Lower intestine	Severe
# 8.6 Ectoparasites

There are a number of ecto-parasites that infest chicken as shown in the table:

English name	Causative agent
Fleas	The main flea pest is the sticktight flea; Echidnophaga gallinacean. Attach on the skin of the head
Lice	Chewing lice; Order Mallophaga. They feed on feathers, feather debris, skin scales and other dermal scurf.
Ticks	Argasid Ticks (soft-shelled ticks) are most common. Argasid ticks are nocturnal feeders and favor the soft un-feathered skin beneath the wings
Body or Scaly leg Mites	Ornithonyssus spp remain on chickens permanently; while Dermanyssus mites parasitize chickens nocturnally Knemidocoptes mutans- scaly leg mites

# NB: Refer to Table 7 for the local dialect name for ectoparasites

- In Marsabit county the apparent morbidity rate due to external parasites in chicken is estimated to be 20% with 7% mortality and 35% case fatality rate.
- External parasitism in chicken flocks in Marsabit county occurs throughout the year with no seasonal outbreaks reported.

## Main clinical signs:

Ectoparasites affect chicken by:

- Transmitting diseases to chicken- ticks transmit spirochetosis.
- Sucking blood causing birds to be weak, less productive and more vulnerable to disease. If too many, cause anaemia and death in young birds.
- Birds spend more time scratching leading to reduced growth and egg production.
- Lice cause irritation and damage to feathers that results in depluming.

### Diagnosis:

- Heavy mite infestation is characterized by anaemia and the appearance of black mite dirt that is the exoskeleton casts and excreta of the mites.
- Mites also cause dermatitis of the vent area.
- Birds parasitized with soft-shelled ticks (Argas species) show multiple hematomas associated with biting sites.
- Chronic infestation of the legs of free-roaming chickens with scaly leg mites, Knemidocoptes mutans, results in proliferation of scales overlying the shanks and feet. The gross appearance of the lesion is pathognomonic of Scaly Leg Mites. The diagnosis may be confirmed by microscopic examination of scales to detect the mite.
- Evidence of mature adult lice on examination of chicken which appear as brown tiny parasites attached to the skin of the head is diagnostic for lice infestation.
- Under the microscope lice egg clusters ("nits") are observed as spherical white structures adherent to the shafts of feathers.

#### Treatment:

- Ectoparasites may be treated with a carbamate insecticide such as Carbaryl 7.5% insecticide powder external use (Sevin Poultry dust) applied as a powder to birds at two-week intervals. Cages and housing can be treated with carbamate dust topically. Application should ensure it is up to the base of the feathers.
- Only approved insecticides should be applied to poultry or used in the vicinity of housing to avoid contamination of the food chain. Insecticides should be used in accordance with manufacturers' label instructions

#### Prevention and Control:

• Ectoparasites are also found in the poultry premises like wall cracks, perch joints, rough

floor, in litter and in nest boxes. These surfaces should also be dusted with the acaricide powder.

- Isolation of all new birds and application of acaricide powder before allowing them to mix with the flock should be done
- Regular spraying of the chicken house with acaricide applications recommended for use in chicken such as Ectomin 100 EC (cypermethrin) at a ratio of 1:1.
- Birds can also be dipped to allow better penetration of the chemical. Immerse the chicken one by one into the Ectomin solution. Always follow manufacturer's instructions on use to ensure birds are not poisoned.



### Figure 138

Deposit of louse eggs ("nits") on the shafts of feathers (Left picture) and right picture is a comparison of the normal leg compared to scaly leg of a chicken infested with scaly leg mite

#### References

- Apopo AA, Kariithi HM, Ateya LO, Binepal YS, Sirya JH, Dulu TD, Welch CN, Hernandez SM, Afonso CL (2020). A retrospective study of Newcastle disease in Kenya. Tropical Animal Health Production. 2020 52(2):699-710. https://doi.org/10.1007/s11250-019-02059-x
- 2. MSD Veterinary Manual 2022. https://www.msdvetmanual.com/
- 3. WOAH Terrestrial Animal Health Code (2021). VOLUME II: Recommendations applicable to WOAH Listed diseases and other diseases of importance to international trade. https://www.WOAH.int/en/what-we-do/standards/codes-and-manuals/terrestrial-code-online-access/
- 4. Vegad J.L. (2007). A colour atlas of poultry diseases: an aid to farmers and poultry professionals. published by international book distributing co. (publishing division). ISBN: 978-81-8189-130-3.
- Picture Source: Picture book of infectious poultry diseases (2010). Produced by FAO ECTAD Southern Africa. https://www.itpnews.com/uploads/2017/03/color%20atlas%20of%20poultry%20disease%20-%20 01.pdf?direct\_access\_media=1

# Chapter 9: Donkey Diseases

Table 30: Local dialect name of priority donkey diseases in Marsabit county

Donkey Parasitic Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Mange	Chito	Chito	Haddo	Lpepedo	Emitina / Ekokoi	Nyaach
Helminths	Miini	Miini	Deyah	Ntumwa	Ngipelei	Chade
Donkey Bacterial Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Wounds	Madha	Madha	Jiis	Ngoldonyot	Ngajemei	Lilo Dhuorich
Tetanus	Dukub Gola	Dukub Gola	Sugeri Gerii	Nyoole	Etergege Loteru	Lile
Strangles	Gororsa	Gororsa	Argabi Ehel	Lchama Loo Ngiron	Lowala	Iidho
Donkey Protozoal Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Equine Trypanosomiasis	Gandhi	Gandhi	Omar	Saar	Lokipi	Lesikonye
Babesiosis	Birte	Bira	Kati Ti Gudhudhan	Noonkula	Lodip	Sin Bura
Donkey Disease Due To Undetermined Causes	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Colic	Dukub Garaa	Dukub Gara	Ure Yubama	Nolmurui	Loding'achin	-
Donkey Viral Diseases	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
African Horse Sickness (AHS)	-	-	-	-	-	-

NB- AHS does not occur in Marsabit county. The disease will be described in the handbook as it is an important disease that is endemic in Kenya with new outbreaks being reported in areas that have no previous outbreak of the disease. Rabies is an important disease in donkeys and is covered in the Annex section under Zoonotic diseases.

Table 31: Medicinal plants and traditional practices used to treat priority donkey diseases

Ethnoveterinary Practice	Target donkey disease
Acacia Nubica	Rabies, Tetanus, Colic, Strangles, Trypanosomiasis
Euphorbia species	Rabies, Tetanus, Colic
Onions	Tetanus, Babesiosis, Colic, Strangles
Azadirachta indica Neem tree	Strangles
Traditional Practice	Target donkey disease
Salty water from saline wells, springs or Lake Turkana- Topical application	Colic
Old engine oil	Mange
Bleeding from cuts made at the base of the ears	Babesiosis, Trypanosomiasis
Tobacco snuff	Strangles
Hot Iron or stone cauterization	Tetanus, Strangles
Vegetable Cooking oil	Colic
Melted Fat from a Sheep's tail	Strangles, Trypanosomiasis
Local brew (Changaa)	Colic
Mane trimming	Strangles
Torch battery inner content (Carbon)- pounded and diluted in a little water	Wounds and Mange
Smoke smudging/ fumigation	Strangles
NB: Text in red highlights practices that go against animal	welfare principles

#### Table 32: Veterinary Medicinal Products (VMPs) used by livestock keepers to treat or prevent priority donkey diseases

Type of VMP	Active ingredient	Target diseases
Antibiotic	Oxytetracycline 5 o 10%	Strangles
Antibiotic	Penstrep® procaine penicillin G and dihydrostreptomycin	Colic, Strangles
Anthelmintic	Albendazole / Levamisole	Colic, Helminthiasis
Anthelmintic	Ivermectin oral	Colic, Helminthiasis,
Antibiotic	Diseptoprim ® boluses Sulphadiazine and Trimethoprim	Colic, Strangles
Laxative	Epsom salt	Colic
Acaricides	Amitraz, synthetic pyrethroids, organophosphate and carbamates	Mange
Antibiotic	Gentian violet spray with oxytetracycline	Wounds

Table 33: List of notifiable Equine (Donkey) diseases that should be reported to DVS and WOAH\*

- 1. African horse sickness
- 2. Anthrax
- 3. Contagious equine metritis
- 4. Dourine (T. equiperdum)
- 5. Equine encephalomyelitis (Eastern and Western)
- 6. Equine infectious anaemia
- 7. Equine influenza
- 8. Equine piroplasmosis
- 9. Equine rhinopneumonitis
- 10. Equine viral arteritis
- 11. Glanders (B. mallei)
- 12. Japanese encephalitis
- 13. Rabies
- 14. Vesicular stomatitis
- 15. Venezuelan equine encephalomyelitis
- 16. West Nile fever

\*World Organisation for Animal Health (WOAH)

# Donkey Diseases: Bacterial diseases

# 9.1 Strangles

#### Other names- Horse distemper

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Gororsa	Gororsa	Argabi Ehel	Lchama Loo	Lowala	Iidho
			Ngiron		

#### Case definition

- Strangles is a highly contagious Upper Respiratory tract (URT) disease of equines characterised by abrupt onset of fever, mucopurulent nasal discharges, coughing and abscesses of the submandibular and retropharyngeal lymph nodes.
- The disease is caused by the bacteria Streptococcus equi subsp. equi (S. equi) and affects horses and donkeys of all ages.
- The bacteria can persistently and asymptomatically colonize the upper respiratory tract of animals for years and this serves as a source of new infection. Carrier animals are important for maintenance of the bacteria between epizootics and initiation of outbreaks on premises previously free of disease

#### Epidemiology

- Occurs in sporadic and epidemic outbreaks.
- Strangles occurs in all equine rearing parts of the world.
- · Affects horses, donkeys and mules of all ages.
- In donkeys, it is common in donkeys that are less than two years old (except those under four months of age, which are protected by colostrum-derived passive immunity).
- Previous infection confers immunity; some animals may contract the disease for a second or third time. Especially if they have underlying chronic diseases or are

malnourished.

- In 2022, livestock keepers in Marsabit county estimated that strangles has a 17% morbidity rate with a 13% mortality rate and 77% case fatality rate.
- Strangles in Marsabit county occurs throughout the year but most cases are seen during the short rainy season that occurs between January and March.

#### Aetiology, Pathogenesis and Transmission

- Streptococcus equi subspecies equi which is a Gram-positive, beta-haemolytic coccobacillus.
- The bacterium is an obligate parasite of the URT that relies on its host for survival and inter epizootic maintenance.
- Survival requires moisture and protection from sunlight.
- Transmission is through direct or indirect contact with oral and nasal secretions. After entry through the oral or respiratory systems the bacteria attach to the cells of the tonsillar crypts and the ventral surface of the soft palate then moves to the lymph nodes.
- Subsequent migration of neutrophils into the lymph nodes causes their swelling and abscessation
- Communal drinking points and overcrowding are important risk factors.
- S. equi survives for several weeks in water troughs, but not in soil or pasture.
- · Recovered animals are a persistent source of

infection as they may infection in the pharynx and guttural pouches for months to years.

### **Clinical signs**

- Incubation period of 1 to 3 weeks.
- Suddenly development of complete anorexia and depression.
- High Fever (39.4°-41.1°C)
- Serous nasal discharge that rapidly becomes copious and mucopurulent.
- Dyspnoea and dysphagia due to enlargement of retropharyngeal lymph nodes.
- Death by asphyxiation due to swollen URT lymphnodes.

About 20% of clinical cases have a severe or complicated form that includes;

- Development of suppurative necrotic bronchopneumonia secondary to the aspiration of pus from internal ruptured abscesses or metastatic infection to the lungs.
- Guttural pouch infection with empyema due to rupture of abscesses in the retropharyngeal lymph nodes.
- Metastatic infection, also known as "bastard strangles", results in the formation of abscesses in the lungs, mesenteric lymph nodes, liver, spleen, kidneys and brain.
- Purpura haemorrhagica which is a rare complication caused by bleeding from capillaries which results in red spots on the skin and mucous membranes.

### Diagnosis

- Field based diagnosis using characteristic clinical signs
- Confirmed by bacterial culture of exudate from abscesses or nasal swab samples.
- Haematology analysis reveals leucocytosis due to neutrophilia.
- Nasopharyngeal swab samples for PCR are also used widely in strangles diagnosis.

#### Treatment

• Topical application of warm compresses (hot fomentation) with or without dissolving epsom salt in the warm water should be applied to sites of lymphadenopathy twice a

day to facilitate maturation of abscesses.

- Aseptic lancing of mature abscesses to facilitated drainage improves recovery.
- Ruptured abscesses should be flushed with dilute (3%–5%) povidone-iodine solution for several days until discharge ceases.
- Administration of non-steroid based pain killers like phenylbutazone (dose of 4.4 mg IV once) to reduce pain and fever in sever disease should be considered.

### Antimicrobial therapy is controversial.

- Initiation of antibiotic therapy after abscess formation may provide temporary clinical improvement in fever and depression, but it ultimately prolongs the course of disease by delaying maturation of abscesses. Administration of penicillin during the early stage of infection (less than 24 hours after fever onset) will arrest abscess formation.
- Antibiotic therapy is only indicated in life saving situation like when the animal develops dyspnoea, dysphagia, prolonged high fever, and severe lethargy and anorexia.
- The other disadvantage of early antimicrobial treatment is that the animal fails to mount a protective immune response, rendering the donkey susceptible to another bout of infection after recovery.
- If antimicrobial therapy is indicated, the combined antibiotic Penstrep® (procaine penicillin G and dihydrostreptomycin) should be the first choice drug. For the short acting preparation give 1 ml per 20 kg body weight (BWT) for 3 days and for the long acting preparation give once at 5 mls of Penstrep 20/20 LA per 50 kg BWT.

### Control and prevention

- Clinically affected animals should be separated from healthy ones and should not share watering troughs with the rest.
- Quarantine of sick animals should not be less than 14 days and where possible introduction to the herd should be after two negative nasal swabs. However, this is not possible in Marsabit. Livestock keepers should integrate animals into herd when the animal has stopped having nasal discharge and abscesses

have stopped leaking pus.

- Immunity after infection is prolonged and is associated with local mucosa antibody production.
- Several vaccines have been developed but protection is only 50% for those that are not administered through the intranasal route. The intranasal vaccines were associated with side effects including development of disease in animals.
- No vaccination is done in Kenya as there is no safe vaccine.

Zoonotic potential- None reported Notifiable disease- No Ethnoveterinary practice- Refer to table 31.



A donkey with typical Strangles clinical signs

Picture credit: The clinical companion of the donkey (2021). The Donkey Sanctuary. https://www. thedonkeysanctuary.org.uk/sites/uk/files/2018-03/ the-clinical-companion-of-the-donkey.pdf

# 9.2 Tetanus

### Other names- Lock jaw

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dukub Gola	Dukub Gola	Sugeri Gerii	Nyoole	Etergege, Loteru	Lile

### Case definition

- Tetanus is a toxic reaction to a specific poison (toxin) that blocks transmission of inhibitory nerve signals to muscles. This leads to severe muscle contraction and an exaggerated response to stimuli without a relaxation phase.
- The toxin is produced by the bacterium Clostridium tetani in dead tissue. Most mammals are susceptible, but horses and humans are the most sensitive of all species.

#### Actiology and Epidemiology

- Bacteria Clostridium tetani Clostridium tetani is found in soil and intestinal tracts.
- It is introduced into the body through wounds, particularly deep puncture wounds. Sometimes, the point of entry cannot be found.

- The bacteria remain in the dead tissue at the original site of infection and multiply. As bacterial cells die and disintegrate, the potent nerve toxin is released. The toxin spreads and causes spasms of the voluntary muscles.
- The livestock keepers in Marsabit county estimated an annual apparent morbidity and mortality rates of 4 %. Indicating that tetanus has a 100% case fatality rate. Tetanus in the county can occur all year round but most cases are reported during the long dry season between the months of July and September.

#### **Clinical signs**

- Incubation period varies from 1 to several weeks but usually takes an averages of 10 to 14 days.
- Clinical signs begin as localized stiffness, of

the muscles of the jaw, neck and hind limbs, near the region of the previous deep puncture wound.

- General stiffness becomes pronounced about later spasms and painful sensitivity to touch become evident. As the disease progresses, the reflexes increase in intensity and the animal is easily excited into more violent, general spasms by sudden movement or noise. The spasms may be so severe that they cause bone fractures. Spasms of head muscles cause difficulty in grasping and chewing of food, hence the common name, lockjaw.
- Donkeys also has flared nostrils, protrusion of the third eyelid, difficulty breathing and inability to lie down or stand.
- Donkey may have extensor rigor 'saw-horse stance' with stiff rigid legs and hind limbs stretched out behind.
- Collapse and death from respiratory arrest (usually within 3 to 10 days of the onset of
- clinical signs)

#### Diagnosis

• Field based diagnosis should be based on wound history and clinical signs.

#### **Treatment and Prevention**

- Treatment may be successful if done early but this is rarely the case as by the time the clinical signs start they toxin effect are irreversible.
- Treatment involves thorough cleaning and disinfection of the wound, the use of antibiotics, and tetanus antitoxin (which helps protect against the effects of additional toxin being released).
- Recovering animals as part of supportive nursing care should be kept in a dark, quiet area. This is not practical in Marsabit.
- Immunization can be accomplished with tetanus toxoid and is usually recommended for all horses and donkeys. Yearly booster injections of toxoid are advisable.
- Breeding females' animals should be vaccinated during the last 6 weeks of pregnancy and the foals vaccinated at 5 to 8 weeks of age. In high-risk areas, foals may be given tetanus antitoxin immediately after birth and every 2 to 3 weeks until they are 3 months old, at which time they can be given toxoid.

Zoonotic potential- None reported Notifiable disease- No Ethnoveterinary practice- Refer to table 31.



### Figure 140

A horse showing classic signs of Tetanus 'saw-horse stance'. Note the erect ears, elevated tail and stiff-legged rigid stance.

> Picture credit: The Working Equid Veterinary Manual (2016). A BROOK publication https://www.thebrooke. org/our-work/working-equidveterinary-manual

# 9.3 Wounds

Type of lesions	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Skin wound	Dukub Gola	Dukub Gola	Sugeri Gerii	Nyoole	Etergege / Loteru	Lile

#### Aetiology

- Most wounds result from repetitive trauma or predisposing factors like poor harnessing, and carrying of inappropriate loads. In Marsabit county donkeys also get wounds from predation due to hyena bites.
- Wounds and abscesses in donkeys when discovered by the owner or animal health service provider are always advanced and with possible secondarily infection. This is because the long coat of the donkey hides wounds and any grooming or examinations are done infrequently.
- When managing wounds, the cause of the wound has to be addressed in order to prevent re-occurrence. This may involve education and advice on alternative options for the owner or the community.
- The prevalence of skin wounds in Marsabit county was estimated by livestock keepers to be 4% with no reported fatalities.
- Wounds that become infected are swollen, hot and painful to touch. There may also be an unpleasant smell or pus oozing from the wound.

# Management of Saddle wounds and abscesses

• The Golden Period is a theoretical time frame of 8 hours after which a contaminated wound is described as infected. Within approximately 8 hours any bacteria present within the wound will adhere to the tissue surface and is unlikely to be removed by wound flushing and debridement. This has implications when determining a wound management protocol (e.g. open wound or closed wound). If more than 8 hours have passed it is preferable to allow a wound to heal by second intention. Suturing an infected wound will worsen the prognosis as the wound could develop deep sited infection and cause suture breakdown.

- Livestock keepers need to be trained on how to assess wounds severity so as to be able to seek veterinary intervention early. They also need to be taught basic first aid to manage severe wounds as they wait for the animal health practioner.
- Wounds that require urgent veterinary intervention include
- Wounds that have continuous or excessive bleeding (haemorrhage)- need immediate attention before the vet arrives. Ask the livestock keeper to keep the donkey calm and still as possible. If bleeding wound is on the leg they should take care not to get injured. They should take a clean cloth and apply pressure directly to the wound. If blood seeps through the cloth they should apply a second layer of cloth on top, without removing the first so as not to dislodge the clot forming.
- 2. Wounds that penetrate or puncture the entire skin thickness.
- 3. Skin wounds on or close to a joint especially the knee, hock and elbow joints.
- Avulsed wounds (partial or complete tearing away of skin and the tissue beneath) as seen in predation wounds.

5. Skin Wounds of the lower leg can be serious even if they don't seem deep.

Management of severe wounds involves identification of all involved structures (synovial cavities, bones, soft tissues), control of active haemorrhage and determining if there is need for surgical intervention. Surgical intervention should be conducted by a trained veterinary surgeon, surgical intervention is indicated if there is continuous and extensive blood loss that does not respond to pressure application, tendon injury, penetration of a synovial structure or body cavity like the thoracic or abdominal and extensive avulsion of the skin and underlying tissues.

### Steps of treating skin wounds;

- 1. Wash the wound by running cold, clean water over the wound to remove dirt and to allow visualisation of the wound depth and margins.
- 2. Clean the wound using a gauze swab and antiseptic solution that is diluted according to manufacturer's instructions.
- 3. Trim the hair around the wound to help keep the wound clean.
- 4. Infected wounds should be lavaged with 1 to 2% povidone Iodine. Current research indicates that hydrogen peroxide used to clean infected wounds actually works against wound healing and should not be used
- 5. In case of infected puncture wounds establish

proper drainage by lancing open the puncture wound area.

- 6. Give tetanus toxoid, analgesic such as phenyl butazone and appropriate antimicrobial therapy such as Penstrep®. For the short acting preparation give 1 ml per 20 kg body weight (BWT) for 3 days and for the long acting preparation give once at 5 mls of Penstrep 20/20 LA per 50 kg BWT. As a general rule do not give corticosteroids as they delay the inflammatory process of forming granulation tissue which is one of the processes of wound healing.
- 7. If skin wounds are due to rabid animal, ensure you administer a rabies vaccine to animal at a post exposure dosage on Day 0, Day 3, Day 7, Day 14 and Day 28. If one is not able to give all the days, the general practice is that the animal should receive not less than 3 doses with the first dose given within 3 days of the bite.
- 8. Repeat wound lavage with Povidone iodine every day with 1 to 2 % povidone iodine until the pus discharge clears.

Zoonotic potential- None reported (except if bite wounds are from rabies cases)
Notifiable disease- No
Ethnoveterinary practice- Refer to table 31.



### Figure 141 Open wounds over the carpus joint (left) and how to shave around a wound in a horse

# Pyoderma

- This is secondary bacterial infection common in all skin conditions regardless of the primary cause.
- It is caused by many bacteria which are part of the normal flora of the skin. The bacteria invade the broken skin and colonise in the damaged tissue.
- Common causal bacteria include; Staphylococcus species (predominant bacteria in pyoderma), Corynebacterium species and Dermatophilus congolensis that causes 'rain scald'.
- Predisposing factors include damp skin due to warm and wet weather, poor grooming, skin wounds, abrasion and pruritic conditions (mange) resulting in excoriation.
- Clinical signs Staphylococcus infection forms crusts in a circular pattern (similar to ringworm). The skin appears wet due to exudate. Encrusted papules and pustules are evident. In severe cases a deep pyoderma with ulceration develops. There are signs of pruritus and discomfort. Inflammation – causes redness, swelling and pain. (Staphylococcus lesions are usually very painful.)
- Treatment Clip the affected area and clean with dilute antiseptic. An application of topical anti-bacterial solution may be necessary; in severe or chronic cases systemic antibiotics may be indicated (trimethoprim sulfamethoxazole).
- Zoonotic potential- Yes, Consider the human health risks of Staphylococcus infection particularly antibiotic-resistant strains known as MRSA (Methicillin Resistant Staphylococcus Aureus). Always use gloves when managing wounds.

# Proud flesh (excessive granulation tissue)

- Wounds heal via granulation from the depth and epithelialisation from the edges. Usually these two act in synchrony resulting in a fast healing process.
- Proud flesh occurs when granulation exceeds the epithelialisation process and it occurs as an excessive pink tissue forming a large protruding growth which prevents epithelialisation. Proud flesh is common on the distal limbs of horses and donkeys but can occur anywhere.

#### Management of excessive granulation tissue

- Using a scalpel blade, trim the excess granulation tissue to just below the skin surface. Granulation tissue has an excellent blood supply and one should expect profuse bleeding. Be careful not to trim the newly laid epithelium around the edges of the wound. The aim is to encourage epithelialisation so the wound heals.
- 2. After trimming apply corticosteroid cream to the centre of the wound only so as to inhibit further granulation, make sure you the cream does not reach the skin edges.
- 3. Firmly bandage the wound the pressure will prevent further bleeding.
- 4. Apply the corticosteroid cream to the granulation tissue every third day for 3–5 applications after debridement. If exuberant granulation tissue reforms, repeat the process. Do not use other chemicals such as potassium permanganate or copper sulphate.
- 5. Prevention of excessive granulation is possible through early treatment of wounds and cleaning wounds thoroughly to prevent infection. Where possible bandaging severe distal limbs wounds and preventing excessive walking also limits the development of proud flesh.







Figure 143 Fibroblastic sarcoid (left) and Nodular sarcoid in donkeys

# 9.4 Dermatophilosis

- **Dermatophilosis ('rain scald') is caused** by Dermatophilus congolensis (bacteria) and is spread by carrier animals. Pre-disposing factors for infection include moisture and abrasion.
- Clinical signs- Appearance of small crusty lumps with hair standing up (paintbrush) with underlying skin moist and inflamed. Lesions are found mainly on the dorsal surfaces of the body, caudal pastern due to hobbles or a muddy environment and the skin under harness such as the flanks as well as on the face of the donkey.
- **Diagnosis** make Impression smear of the underside of crusts that are stained with Giemsa and look for 'railroad track' cocci (bacteria joined together in a line)
- **Treatment** Minimise exposure to wet, muddy environments. Rugs and saddle padding should be removed to avoid contact with sweaty skin. Shave or clip the affected area. Apply dilute antiseptic and leave open to the air to dry. Remove loose scabs and crusting which harbour the bacteria. Burn the removed scabs, as bacteria within this material can infect other donkeys and horses. Treat with penicillin or trimethoprim sulfamethoxazole for 7 days. Protect lesions on the lower limb by applying a waterproof barrier cream (petroleum jelly or hydrous wool fat).



Figure 144 Dermatophilosis on the face of a donkey

# Donkey Diseases: Protozoal diseases

# 9.5 Equine Trypanosomiasis

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Gandhi	Gandhi	Omar	Saar	Lokipi	Lesikonye

#### **Case definition**

- Trypanosomiasis are infectious diseases affecting both humans and animals.
- The causative organism is a protozoa haemoparasites called Trypanosoma that is transmitted by Tsetse fly (Glossina species) or mechanically by hematophagous biting flies of the species Tabanus and Stomoxys.

#### Actiology and Epidemiology

- Tsetse flies are distributed in a 'belt' across 37 countries in Africa; trypanosomiasis is endemic in these areas. For T. congolense and T. brucei, development within the tsetse is an essential stage in the life cycle. All domestic animals are at risk of infection in tsetse areas. T. brucei gambiense and rhodesiense cause disease in humans resulting in a fatal condition known as sleeping sickness. Horses cannot be kept in tsetse areas without trypanocidal prophylaxis; however, donkeys have been kept in these areas. Although resilient, donkeys are not resistant to tsetse transmitted trypanosomiasis.
- Human African trypanosomiasis (HAT), also known as sleeping sickness, is an important public health disease caused by Trypanosoma brucei gambiense and Trypanosoma brucei rhodesiense. T. brucei gambiense causes the chronic form of HAT in west and central Africa while T. rhodesiense induces an acute form which is found in eastern and southern Africa. The human-infective parasites can

be transmitted to livestock and wildlife; the animals also serve as reservoir host for HAT.

- During the last three decades, HAT has been controlled effectively and this has allowed the World Health Organisation (WHO) to include it in the WHO roadmap for eradication, elimination and control of neglected tropical diseases, with a target set to eliminate HAT as a public health problem by 2050. Achieving these goals requires investigation of animal reservoirs especially in equines and especially so in donkeys as they are often an ignored species when formulating control and eradication programmes.
- The disease in animals is known as African Animal Trypanosomiasis (AAT).
- Equine trypanosomiasis caused by species of the genus Trypanosoma is a complex of infectious diseases called dourine, nagana and surra. These diseases are characterized by overlapping clinical features that can be defined by their mode of transmission.
- Nagana is caused by T. vivax, T. congolense and/or T. brucei subspecies and is transmitted by tsetse flies; surra is caused by T. evansi and is mechanically transmitted by biting flies; while dourine is due to T. equiperdum and is sexually transmitted.
- Knowledge of the different transmission modes is important so as to design appropriate control measures. This can only be done if animal health service providers gain a better understanding of the epidemiology of equine

trypanosomiasis by identifying trypanosomes that naturally infect horses and donkeys.

- T. equiperdum that is transmitted mechanically when mating is the only trypanosome that is not transmitted by an invertebrate vector. The pathogenesis of dourine differs from other trypanosomes in that it is primarily a tissue parasite which rarely invades the blood. There is no known natural reservoir of the parasite other than infected equids. Dourine is confined to parts of Africa, Asia, Central and South America.
- A 2020 prevalence study in Chad that collected 286 blood samples from horses and donkeys found a 19% prevalence for Trypanosoma parasite on Rapid Diagnostic Test (RDT), a 13% prevalence on Capillary Tube Centrifugation (CTC) test and 35% prevalence on PCR revealed. Trypanosomes of the subgenus Trypanozoon (T. brucei, T. evansi and T. equiperdum) were the most prevalent (29%), followed by T. congolense forest (12%), T. congolense savannah (5%) and T. vivax (5%). Two donkeys and one horse were found with T. b. gambiense infections that causes HAT.
- In Kenya, tsetse flies are endemic in 38 out of 47 counties. Since 2009, Kenya has not reported a case of HAT apart from two cases detected in 2012 from European tourists returning from the Maasai-Mara National Reserve. The Kenya Tsetse and Trypanosomiasis Eradication Council (KENTTEC), established in 2012 has made significant progress in controlling animal trypanosomiasis (AAT). However, 'nagana' is still a major problem in Kenya. Several trypanosome species such as T. b. brucei, T. congolense, T. vivax, T. simiae, and T. suis still occur in many parts of the country partly due to the extra risk of transmission by other biting flies and the fact that most livestock are kept under the extensive production system that is characterised by highly mobile herds in search of pasture and water.

#### **Clinical signs**

#### Nagana- Tsetse transmitted Trypanosomiasis

- T. brucei clinical signs include anaemia, icterus (jaundice), enlarged lymph nodes and petechial haemorrhages of the mucous membranes (third eye lid and vulva mucosa are good sites to check for petechiation). The animals also have parasitaemia 'waves' of intermittent high fever (41°C) which occurs as host immunity responds to changes in the proteins on the parasite surface. T. brucei can cause a serious and acute disease in donkeys and horses with mortality occurring within 14–90 days if left untreated.
- T. congolense and T. vivax have a low prevalence and cause milder and more chronic clinical disease when compared to T. brucei. The main clinical signs are anorexia, wasting and generalised oedema around 14 days after infection. T. vivax can also be transmitted mechanically, and therefore can spread beyond the tsetse belt.

#### Surra- Mechanically transmitted by biting flies Trypanosomiasis

- T. evansi clinical signs include severe weight loss, progressive weakness, anaemia, haemoglobinuria, intermittent fever, petechial haemorrhage of mucous membranes, oedema of limbs, lower abdomen and thorax and severe neurological signs. The mortality rate in untreated horses is almost 100%. The severity of the disease depends on the parasite strain and factors including stress and the health of the equid. Animals subjected to stress, such as malnutrition and physical labour, are more susceptible to the disease.
- Chronic forms persist for several months up to 2 years, providing a reservoir of infection for other animals. There is considerable variation in host species susceptibility; severe rapidly fatal disease is common in horses, whereas donkeys and mules tend to develop chronic mild or subclinical infections.
- There are rare reports of T. evansi infection in humans, these is thought to be rare anomaly that occurred as the individuals were immuno-compromised.

### Dourine- Sexually transmitted Trypanosomiasis

- T. equiperdum clinical signs include- A mucopurulent discharge from the penis or vulva, and oedema around the genital area (is the most common initial presenting sign). Generalised clinical signs include fever, oedema, anaemia and wasting. Approximately a month following inoculation, urticarial reactions erupt all over the body. Progressive paralysis is a possible sequel and these cases are frequently fatal. Abortion is also common.
- The protozoa parasite may persist for years in donkeys and mules without showing clinical signs; mortality can be high in untreated horses.

#### Diagnosis

- Microscopic evaluation of centrifuged blood (CTC) test.
- Serologic tests (RDT and ELISA) and trypanosome antigen detecting tests (PCR).
- T. equiperdum rarely produces haemoparasites so blood smear detection is not usually effective. Microscopy of direct smears from the fluid of infected genitalia may detect parasites; however, serology is the most reliable test for dourine.

#### Treatment

- Treatment is not recommended for dourine as it is never 100% effective; recovered animals can still infect others. Castration may be an option in males; however, euthanasia should be discussed with the owner. It is important to reiterate that infected animals should not be used for breeding.
- Quinapyramine salts only for treatment do not use for prevention in endemic areas-The drug is used for T. evansi at a dose rate of 3 to 5 mg/kg of a 5% solution split between three injection sites. Quinapyramine dimethylsulphate and quinapyramine chloride (Triquin) is the most commonly available drug but it is poorly tolerated (particularly in horses). Only administer it deep IM injection using a long narrow-gauge needle, as SC

injection results in sloughing of skin and wounds that take months to heal.

- Homidium bromide (250 mg) dissolved in sterile water, prepared as 1–2.5% solution for SC or IM injection at 1 mg/kg. With both prophylactic and curative properties, this is most effective against T. vivax
- Do not use Diminazene aceturate- Just like in camels it causes severe side effects which can be fatal.

#### Control

- Attempts to develop a vaccine have been futile as the parasite overrides the immune defences by rapidly changing the surface proteins. Each time an antibody response is mounted, the coat changes and the defences become useless.
- Prophylactic treatment is not recommended. Although in the short term this may reduce the number of clinical cases, it has led to drug resistance and is no longer encouraged.
- Vector control through topical application of fly repellents (especially when biting fly population is high during the rainy season) and dung removal are important control strategies. Also avoidance of tsetse infested areas if possible is encouraged. and do not house equids close to stagnant water.
- Treat infections early so as to prevent spread to other animals.

#### Zoonotic potential- Yes Notifiable disease- Yes

Dourine is a WOAH listed disease. Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System. Ethnoveterinary practice- Refer to table 31.



Figure 145 Petechial haemorrhage of the third eyelid in a horse and Trypanosomes in a blood smear



# 9.6 Babesiosis- Equine Piroplasmosis (EP)

#### Case definition and Epidemiology

- Tick-borne infection caused by two haemoprotozoa parasites- Babesia caballi and Theileria equi. This disease affects horses, donkeys and mules
- B. caballi and T. equi are endemic in many tropical and subtropical countries in Mediterranean Europe, Africa, the Middle East, Asia, and Central and South America. Equine piroplasmosis is a notifiable disease in many countries and is reportable to the WOAH by National director of veterinary services.
- Twelve species of ixodid ticks of the genera Dermacentor spp. (central Asia), Hyalomma spp. (Middle East and Africa), and Rhipicephalus spp. (Africa and South America) are vectors for EP.
- T. equi is also iatrogenic transmitted by contaminated needles and syringes.
- Carrier equids remain sources of infection for tick vectors for up to 4 years after infection.

#### Pathogenesis and Clinical Signs

- The causative agents of EP are intracellular parasites which invade red blood cells inducing haemolysis, and development of haemolytic anaemia.
- The severity of clinical signs relates to the parasite burden, and disease can manifest as acute or chronic, mild to severe. Fatalities may occur in the first 48 hours of infection but chronic disease often develops. B. caballi is clinically milder than T. equi; severe anaemia is very rare with a B. caballi infection. Donkeys tend to develop the chronic form of the disease and the signs are often unspecific.
- Clinical signs of acute EP include -Intermittent fever (> 40°C), sudden sweating,

anaemia, icterus, petechial haemorrhage of third eyelid, rapid heart rate and respiratory rate, Oedema of muzzle, limbs, ventral abdomen and thorax, hind limb weakness, reluctance to move, tremors, haemoglobinuria and dry faeces. Death within a few days (mortality 5–10% in endemic areas and up to 50% in naïve horses)

• Clinical signs of chronic EP - Inappetance, weight loss and poor performance.

#### Diagnosis

• The number of parasites in the blood varies throughout the course of infection. In acute cases with clinical signs the haemoparasites are readily visible on a blood film, appearing as dark dots, rings or pear-shaped marks within red blood cells. It may be difficult to detect parasites in animals in the latent or chronic stages of disease, particularly with B. caballi. Laboratory techniques include PCR and antibody ELISA can be used for chronic cases.

#### Treatment

- Drugs used to treat EP are toxic, so administer treatment only to animals with severe clinical signs or if parasites are present in > 50% of RBCs.
- Imidocarb dipropionate (Imizol)- efficacy of this medication is variable and toxic side effects are common. Administration can cause severe colic and diarrhoea. Administer 2.4 mg/kg as a single deep IM injection; donkeys are more susceptible to toxic side effects so use a low dose (1–2 mg/kg). A treatment protocol of 4 injections at 4 mg/kg at 72hour intervals has been recommended for complete elimination of T. equi in endemic areas complete parasite elimination will reduce

endemic stability which is not advisable so only treat to alleviate acute severe symptoms.

• Diminazene aceturate- avoid this drug in equids unless other drugs are unavailable, as it has a low therapeutic index and toxic side effects are common. Administration of 3.5 mg/kg IM reduces the clinical signs within 24 hours. (In areas of resistance, horses require even higher doses; 2 doses 24 hours apart at 5 mg/kg for B. caballi, 6–12 mg/kg for T. equi.) However, this drug is associated with marked side effects even at the lower dose rate. At higher doses, the risk and severity of such side effects would be increased and is not recommended. Toxicity can be treated with calcium salts.

Endemic stability- 'Infected but not affected'this is an epidemiological state in which severe clinical disease is scarce despite high levels of infection in the population. In endemic areas where equids are faced with a low level of continuous challenge, immunity develops and reduces the severity of disease. Disruption of this low-level challenge, through control of ticks or constant use of treatment drugs might result in an increase in clinical disease incidence. Under conditions of epidemiological stability, when hosts are under frequent exposure to EP, foals are protected passively by maternal antibodies acquired via colostrum. This protection can last for up to 9 months. Infections of foals in this period will induce immunity without any overt signs of the disease.

#### Zoonotic potential- No

#### Notifiable disease- Yes

WOAH listed disease. Report to county and national DVS by entering the outbreak event in the KABS mobile surveillance app. KABS stands for Kenya Animal Bio Surveillance System.

#### Ethnoveterinary practice- Refer to table 31.



Figure 147 Blood smear showing Theileria equi (A) and Babesia caballi (B) in erythrocytes.



Picture credit: Kumar, Sanjay & Kumar, Rajender & Sugimoto, Chihiro. (2009). A Perspective on Theileria Equi Infections in Donkeys. The Japanese journal of veterinary research. 56. 171-80. https://www.researchgate.net/ deref/http%3A%2F%2Fwww.ncbi.nlm.nih.gov%2Fpubmed%2F19358444

# Donkey Diseases: Parasitic diseases

# 9.7 Mange

#### Aetiology and Clinical Signs

- Mange is caused by microscopic parasites called mites that burrow into the skin causing skin irritation that manifests as continuous licking or scratching. Mange can be contributing cause of pastern dermatitis.
- The most common mites found infesting equids are Chorioptic sp. Other genera of mange mites, such as Sarcoptes sp., Psoroptes sp., and Demodectic sp., are rarely diagnosed due to their susceptibility to macrocyclic lactone dewormers (ivermectin and moxidectin) which are mostly used in equine husbandry.
- Sarcoptes scabiei var equi is rare but is the most severe type of mange. The first sign of infestation is intense pruritus due to hypersensitivity to mite products. Early lesions appear on the head, neck, and shoulders. Regions protected by long hair and lower parts of the extremities are usually not involved. Lesions start as small papules and vesicles that later develop into crusts. The skin becomes lichenified, forming folds. If infestations are not treated, lesions may extend over the whole body, leading to emaciation, general weakness, and anorexia.
- Chorioptic mange is caused by infestation with Chorioptes bovis (formerly C. equi) and is the most common form of mange. Lesions caused by C bovis start as a pruritic dermatitis affecting the distal limbs around the foot and fetlock. Papules are seen first, followed by alopecia, crusting, and thickening of the skin.

A moist dermatitis of the fetlock develops in chronic cases. Infested animals may stamp their feet or rub one foot against the opposite leg or object. Chorioptic mange is a differential diagnosis for "greasy heel" in working equids. The signs subside in hot weather but recur with the return of cold weather.

 Demodectic mange is caused by infestation with Demodex equi or D.caballi. Demodex mites infest hair follicles and sebaceous glands. D equi lives on the body, and D caballi on the eyelids and muzzle. Demodectic mange is rare but can manifest as patchy alopecia and scaling or as nodules. Pruritus is absent; therefore, secondary infections due to excoriation are rare. Therapy is rarely done, although there is limited evidence that the macrocyclic lactones may be effective. Lesions have also been reported to resolve without treatment.

#### Diagnosis

- Mites burrow in the skin and therefore are not visible to the naked eye.
- Skin scraping of affected area or margin of affected area should be done.
- See Chapter 1 for more details on mite diagnosis.

#### Treatment

- Hot lime sulphur spray or dip is labelled for use against sarcoptic, psoroptic, and chorioptic mites in equids. Treatment should be repeated every 12 days if needed, following the speciesspecific dilution on the label.
- Although not labelled for treatment of mange,

two doses of oral ivermectin at 200 mcg/kg given 14 days apart (field studies), or a single treatment of oral moxidectin at 400 mcg/kg can be used.

#### Control

- Affected animals should be separated from those that appear to have no active signs of the disease.
- Insecticide application (pyrethrins, carbamates) may be of value in reducing reinfestation of recently treated animals, and the

spread of the condition.

- Clipping of excess hair in affected regions, such as the pasterns, may aid in the management of mites.
- Some animals with few or no clinical signs may harbour chorioptic mites, and serve as carrier animals.

## 9.8 Dermatophytosis ('ringworm')

- Common in young or immune-compromised animals.
- Lesions are caused by fungal species that utilise keratin, Trichophyton and Microsporum.
- The incubation period is 1–4 weeks and the infection spreads slowly amongst a group of equids.
- Ringworm is a zoonotic skin condition and one should wear gloves when treating affected animals.
- Clinical signs Crusting and scaling of skin that has multifocal, sharply demarcated areas of hair loss, classically a coin-shaped appearance. Initial lesions appearing as raised, swollen lumps (urticaria) with variable pruritus
- Treatment Generally equids recover over the course of several months. Affected equids are a source of infection to other animals

and humans throughout recovery. If severe, topical antifungals are indicated. Follow the instructions as these medications can be toxic. Wash daily with dilute iodine (10% solution). Systemic antifungals (griseofulvin) require a prolonged treatment period for efficacy, and compliance can be problematic. Griseofulvin should not be used in pregnant mares as the medication is teratogenic. Isolate the affected animal and monitor animals in contact for signs of similar skin lesions.

Zoonotic potential - Yes for Sarcoptic mange – always use gloves Notifiable disease- No Ethnoveterinary practice- Refer to table 31.





Picture source: https://twitter.com/4helpinganimals/ status/1242755217952624641



Figure 149 Clinical appearance of dermatophytosis in a horse

# 9.9 Helminthiasis

Type of parasite	Predilection Sites
Large strongyles (Strongylus spp.particularly vulgaris)	Adults Large Intestines Larvae Intestinal Wall
Small strongyles (cyathostomes)	Adults Small Intestine Larvae Liver And Lungs
Roundworms (parascaris equorum)	Adults Large Intestine Larvae Intestinal Wall
Pinworms (Oxyuris equi)	Adults Large Intestines Larvae Intestinal Wall
Tapeworms (Anaplocephala spp.)	Adults Large And Small Intestine Larvae Forage Mites
Thread worms (stronglylooides westeri)	Adults Small Intestine Larvae Lungs And Other Tissues
Liver fluke (rarely causes diseas in equids) Fasciola hepatica - temperate Fasciola gigantica - tropical)	Adults Liver Larvae Snail

#### **Clinical signs**

- Clinical signs are varied, including poor coat health, anorexia, weight loss, diarrhoea, colic, rectal prolapse, lack of vigour, poor work performance, and productive loss.
- Large strongyle- S.vulgaris larval stages migrate through the cranial mesenteric arteries and adjacent branches. Migration induces thrombus formation and thickening of arterial walls. Blockage of the blood supply to the intestine results in infarction and severe colic. If the cranial mesenteric artery is completely occluded the intestines become necrotic; this condition is fatal. S. edentus. larvae causes liver damage, and large numbers of adults in the intestine result in unthriftiness, weight loss and anaemia.
- Small strongyle- Cyathostome infection are more moderate than that of large strongyles

but include loss of condition, peripheral oedema, malaise, weight loss, poor appetite, lethargy and disrupted intestinal motility. Mass emergence of larvae results in a marked pathology, 'verminous enteritis', with profuse diarrhoea, colic, weight loss, dehydration, inappetance, dullness depression and proteinlosing enteropathy. Mortality rates can be as high as 40–70%.

• Roundworms - Parascaris equorum is one of the largest endoparasites in livestock. Clinical signs include - Mild coughing and nasal discharge in the migratory phase. Affected equids remain bright and alert. Light intestinal infections are tolerated well with no clinical signs. Heavy infections result in unthriftiness, a dull coat, poor growth in young stock, and lethargy. Younger animals can show signs of colic if a heavy roundworm burden causes a blockage of those parts of the digestive tract with a particularly narrow lumen. Clinical signs mainly in foals as adults generally develop resistance.

- Pinworm- Oxyuris equi are a common parasite in equids but are often non-pathogenic. Adult worms live in the lumen of the colon. After fertilisation the female migrates to the anus and deposits eggs in yellowish white streaks on the perineal skin. Pinworms rarely cause gastro intestinal signs other than Intense pruritus around the anus that can lead to self-trauma.
- Tapeworms (Anoplocephala spp.)- cause mild infestations are considered non-pathogenic; however, moderate to severe tapeworm burdens have been associated with spasmodic colic, intussusception and intestinal rupture.
- Liver fluke infestation is low in equines.
- Stomach bot Gasterophilus spp. the larvae of the bot fly. Presence of larvae in the buccal cavity may cause inflammation, although this is rare. Attachment. to the rectal mucosa has been associated with rectal prolapse

### Diagnosis

• The McMaster technique is a widely accepted protocol for the detection of nematode eggs in equine faeces. Refer to chapter 1 for more details.

### Treatment

- Ivermectin Used in treatment of all major helminth parasites at a dosage rate of 0.2 mg/ kg PO
- Benzimidazoles like fenbendazole are used for all helminth but action is slow - at a dosage of 7.5 mg/kg PO
- Pyrantel emboate Indicated for treatment of large and small strongyles, Oxyuris, Parascaris equi and Anoplocephala spp. At a dose rate of 19 mg/kg PO.

#### Control

 Grazing management practices can be combined with drug intervention to minimise the spread of helminths between animals. Rest heavily grazed ground, and limit density of numbers to reduce pasture contamination.

# 9.10 Rectal prolapse

Is defined as protrusion of one or more layers of the rectum through the anus. causes of rectal prolapse include heavy worm infestation, malnutrition, feeding of dry feed (bran) without adequate water, overloading or overworking. Treatment is aimed to reduce the prolapsed mass as well as correcting the spasmodic colic and tenesmus. To reduce intestinal motility, administer hyosine at the dose rate of 0.5 mg/kg body weight intravenously. Administer flunixine meglumine to reduce the colic at the dose rate of 1.1 mg/kg body weight intravenously. Ensure the donkey is properly restrained by administering low dose of xylazine hydrochloride at the dose rate 0.5 mg/kg intravenously for sedation and 5 ml of 2% lignocaine hydrochloride solution in the first intercoccygeal space. Confirm the effect of analgesia on the perineum by pricking the area with a sterile needle. Then proceed to manually reduce the prolapsed mass by applying sugar granules to reduce oedema and apply liberal lubrication like glycerine. Reinforce with a pursestring suture if there is a tendency for recurrence.





Figure 150 A donkey with rectal prolapse as a result of helminth infestation

# Donkey Diseases: Viral diseases

# 9.11 African Horse Sickness

### Actiology and Epidemiology

- Causative agent is an Orbivirus of which there are nine serotypes.
- Virus is spread by arthropod vectors, usually a Culicoides midge. It is important to realise that African Horse Sickness (AHS) is not contagious between individual equids but will spread in a population from the biting vectors, hence the importance of vector control.
- AHS is a seasonal disease endemic to Sub-Saharan Africa and parts of Southern Africa and mainly occurs during the warm rainy season when Culicoides midges' population is high.

## Clinical signs

- All equids of all ages are affected (mortality rate 70–90%).
- The disease has several manifestations disease depending on which form is present.
- Acute lung form occurs in horses- it has a short incubation period of 3-5 days, high fever, laboured breathing, coughing and profuse frothy nasal discharge. Mortality rate is high. Up to 95% of horses die within a week, often from 'drowning' in their own pulmonary secretions.
- Cardiac form or subacute occurs in donkeys-Has an incubation period of 7–14 days, Fever followed by swelling over the head, eyelids, lips, cheeks and under the jaw, Conjunctival swelling which can be significant. Mortality rate is about 60%, death results from heart failure.

- Mixed form a combination of the above two types- The incubation period is between 5–7 days and is indicated initially by mild respiratory signs which progress to the typical swellings of the cardiac form.
- Mild or Horse Sickness Fever form- Seen in zebras and African donkeys, this form occurs with mucosal congestion and conjunctivitis. Animals usually recover.

### Diagnosis

- A presumptive diagnosis can be made based on clinical signs or post-mortem lesions in areas with a known vector presence.
- Laboratory confirmation through ELISAs or PCR.

### Treatment

- Viral infection- there is no treatment give supportive care
- Extreme acute form may have to be euthanised.

### Prevention

- AHS is notifiable in many countries; report suspected cases to the relevant governing bodies. AHS is a WOAH listed disease.
- Live attenuated vaccines are available commercially in South Africa; these vaccines are polyvalent but may not contain the serotype causing a local outbreak. It is essential that the vaccines are administered annually.
- Vector control using repellents or other locally available substances that are not harmful to equids.



# Donkey Diseases: Undetermined Causes

# 9.12 Colic

- Colic' is a general term meaning abdominal pain. There are a large number of known causes of colic in equids, so a thorough work-up is necessary. Colic is a symptom, not a diagnosis.
- Most, 80% of colic cases resolve spontaneously or with simple medical treatment, with a further 10% requiring intensive medical treatment.
- Inflammation of the stomach or intestinal walls This could be due to parasitic worm damage, sand ingestion, bacterial infection or ingested foreign bodies (e.g. plastic bags). It can lead to altered motility and diarrhoea.
- Stretching of the intestinal wall This is due to distension with gas (tympany) or food material (grain overload, or colonic impaction).
- Altered intestinal motility Peristaltic movements may be abnormally increased (spasm), decreased or absent (ileus). Changes in motility may be caused by dehydration, dietary changes, parasitic worms, toxins, or obstruction from inappropriate ingested materials. They often lead either to diarrhoea or to constipation.
- Loss of blood supply to an area of intestine (ischemia) - caused by intestinal torsion, strangulation, intussusception or total blockage. It leads to the release of toxins into the blood, inflammation and severe pain followed by necrosis and death of the affected section of intestine.
- Unlike in horses that have dramatic colic signs. Donkeys show subtler pain symptoms which may be overlooked. They must be examined thoroughly in order to assess the severity of

colic. Dullness and depression are signs of colic as well as subtle changes in behaviour.

### Approach to a colic case

Take a detailed history of the case by asking the following questions

- 1. When did the colic signs start?
- 2. Has the animal had colic before? If so, when was the last time?
- 3. When did it last pass faeces? How much? What consistency?
- 4. Does it have diarrhoea?
- 5. When was the last de-worming treatment?
- 6. When did the animal last eat? What does it eat? Has the diet changed recently?
- 7. When did it last drink and how much?
- 8. When did it last pass urine?
- 9. Is the animal pregnant?
- 10. History of teeth problems?

### Diagnosis

- · Nasogastric intubation- one must be trained
- Rectal examination- refer to the reference section of this chapter to refer to the book -The Working Equid Veterinary Manual for a detailed guide on rectal examination in equine
- Faecal sampling- to rule out helminthiasis

### Management of a colic case

- · Analgesics to control the pain
- If gastric reflux is not present, and there is evidence of gut sounds, give oral fluids by nasogastric tube. 3–6 litres can be given, depending on the size of the animal, and electrolytes can be added. This can be

repeated every 2–4 hours; if the impaction is large this may be necessary. Because foreign body impactions are difficult to distinguish from food impactions, laxatives such as mineral oil are often used. However, frequent administration of fluids per nasogastric tube has been shown to be as effective, if not more so. Refer to Section 6.2 on fluid therapy for information on how to make an isotonic solution suitable for nasogastric intubation.

- Food should be withheld and the treatment repeated until the animal starts to pass faeces again.
- Recent research on treatment of large colon impactions shows that oral fluid hourly is the most effective way to treat an impaction.



down and stretching out.



# 9.13 Laminitis

- Laminitis is inflammation of the 'laminae', the sensitive soft tissue structure inside the hoof that joins the pedal bone to the hoof wall. As a result, the pedal bone loses its support and becomes unstable.
- The pedal bone then rotates or move

downwards within the hoof, causing pain and irreversible damage.

• Laminitis is an very painful condition that is seldom reversible and can be fatal.



Figure 154 X-ray of a normal donkey foot (left) vs one with laminitis (right)

Picture source: https://www.thedonkeysanctuary.org. uk/what-we-do/knowledge-and-advice/for-owners/ laminitis-indonkeys#:~:text=Laminitis%20is%20 inflammation%20of%20the,causing%20pain%20 and%20irreversible%20damage.

- Potential causes of laminitis are: a) An infection in any part of the body, e.g., uterus or chest infections; b) Excess weight being placed on one foot because the donkey has pain in the opposite foot; c) Access to too much grass; d) Access to cereal or other sugar rich feed;
  e) Hormonal imbalances/disturbances and Equine Metabolic Syndrome (similar to Type 2 Diabetes in humans).
- Donkeys do not behave in the same way as

Note how the pedal bone has lost its parallel relationship with the front part of the hoof wall.

horses when they have severe foot pain. A donkey will tend to lie down more than usual or adopt subtle weight shifting as it stands.

- Evidence of bouts of laminitis is revealed by 'laminitic rings' on the hoof wall. These can be distinguished from other 'event rings' (e.g. sudden diet change) because they are not parallel with each other but diverge towards the heels.
- Main treatment is to give analgesics like phenyl butazone (avoid steroids as they worsen the

laminitis). Hoof trimming may also be useful. Refer to the reference section of this chapter to refer to the book - The Working Equid Veterinary Manual for a detailed guide on rectal examination in equine for more detailed information on laminitis.

• Severe long standing cases cannot be treated and donkey should be euthanised.



Picture source: https://www.littlelongears.org/wpcontent/uploads/2017/04/Laminitis-In-Donkeysfrom-The-Donkey-Sanctuary.pdf

#### References

- 1. Adungo, F., Mokaya, T., Makwaga, O. et al. Tsetse distribution, trypanosome infection rates, and smallholder livestock producers' capacity enhancement for sustainable tsetse and trypanosomiasis control in Busia, Kenya. Trop Med Health 48, 62 (2020). https://doi.org/10.1186/s41182-020-00249-0
- 2. MSD Veterinary Manual https://www.msdvetmanual.com/
- 3. The Working Equid Veterinary Manual (2016). A BROOK publication https://www.thebrooke.org/our-work/ working-equid-veterinary-manual
- Vourchakbé, J., Tiofack, A.A.Z., Mbida, M. et al. Trypanosome infections in naturally infected horses and donkeys of three active sleeping sickness foci in the south of Chad. Parasites Vectors 13, 323 (2020). https:// doi.org/10.1186/s13071-020-04192-1
- Zhu Y, Chen S, Yi Z, Holyoak R,Wang T, Ding Z and Li J (2021). Nasopharyngeal Microbiomes in Donkeys Shedding Streptococcus equi Subspecies equi in Comparison to Healthy Donkeys. Front. Vet. Sci. 8:645627. https://doi.org/10.3389/fvets.2021.645627

# Annex 1: Livestock Vaccination Schedule for Marsabit County

MONTHS/SEASONS	VACCINATION	TREATMENT	OTHER ACTIVITY
JANUARY - MARCH	Routine vaccination of sheep and goats against: PPR CCPP SGP Emergency vaccination against: LSD Enterotoxaemia	Vector control: Ticks Fleas Lice Gnats Mosquitoes Bot fly Horse fly Stable fly Face fly Camel fly Sarcoptic mite Sheep ked	Disease surveillance Outbreak investigation Extension services
APRIL - JUNE	PPR CCPP FMD LSD SGP Enterotoxaemia	Deworming, foot rot management	Disease surveillance, Extension services Response to emergencies
JULY - SEPTEMBER	Vaccination campaigns suspended	Deworming, Vector control Supportive treatment	Disease surveillance Extension services
OCTOBER - DECEMBER	PPR CCPP FMD LSD RVF Enterotoxaemia SGP	Deworming	Disease surveillance Extension services Response to emergencies

# Annex 2: Zoonotic diseases

- A zoonosis (zoonotic disease or zoonoses -plural) is an infectious disease that is transmitted between animals to humans (or from humans to animals).
- About 60% of human infections have an animal origin and 75% of all new and emerging human infections have zoonotic origin. In addition, 80% of pathogens infecting animals are "multi-host," meaning that they move among different specie hosts including humans.
- Zoonotic diseases emergence is not only about the relationship between domestic animals or wildlife and people, but it is also about the complexity of the system as a whole and the interactions between biotic and abiotic components. Biodiversity is a key factor that supports healthy ecosystems thus reduces zoonoses risk.
- All zoonotic diseases mentioned below are notifiable and should be reported to the county and national director of veterinary services.
- Management of zoonotic diseases require a One Health approach this means there is
  need for a multi-disciplinary team that investigates the links between the environment
  (weather, climate, soil, vectors and vegetation factors), susceptible animal and
  human host and behaviour in at-risk populations. The one health approach improves
  information sharing and leverages on discipline expertise that translates to early
  detection, early response thus preventing large scale outbreaks.

# Anthrax

Other names: Splenic fever, Siberian ulcer, Charbon, Milzbrand and Wool sorters disease

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Chit / Chilmale	Chit /Chilmale	Sugeri Harra	Lokuchum / Nkeya E	Lokichum	Gumudich Fas
			Nkulupo		

### Causative agent:

- Gram positive, Spore-forming bacteria called Bacillus anthracis that exists in two forms vegetative form when in the ruminant body and spore form in the environment.
- The bacteria produce extremely potent toxins, which are responsible for illness.
- Anthrax spores are resistant and can survive in the environment for decades, making the control or eradication of the disease difficult.

### **Transmission and Predisposing factors**

- Soil-borne transmission
- Anthrax does not spread from animal to animal or from person to person.
- Bacteria produce spores on contact with oxygen. These spores are extremely resistant and survive for years in soil, or on wool or hair of infected animals.

#### Main route of transmission

• Ingestion or inhalation of spores or through entry of spores through broken skin especially in humans.

#### Occurrence

• Anthrax is mainly a disease of ruminants and is found all over the world on all continents

except Antarctica. Each country has endemic areas with frequent outbreaks. Outbreaks are sporadic in response to unusual weather patterns which can cause spores that were dormant in the soil to come to the surface where they are ingested by domestic and wild ruminants that germinate and cause disease.

### Main clinical signs

Clinical signs are rarely seen;

- Ruminant animals often found dead with no indication that they had been ill.
- Blood that does not clot is seen exuding from body openings.
- The carcass undergoes rapid decomposition and will not have rigor mortis (stiffen after death).
- In ruminants there may be digestive upsets, fever, depression and sometimes swellings of the body. These symptoms last four days before death.
- In carnivores, if animal feeds on infected ruminant carcass there may be an intestinal form of the disease with presents with fever and cramps from which animals sometimes recover.

### Humans

More than 95% of human anthrax cases take the cutaneous form and result from handling infected carcasses or hides, hair, meat or bones.

- In humans, anthrax manifests in three distinct patterns (cutaneous, gastrointestinal and pulmonary). The cutaneous form is the most common (95%) form and is characterised by a boil-like skin lesion that eventually forms an ulcer with a black center (eschar).
- The most susceptible group that contracts anthrax are veterinarians, agricultural workers, livestock producers or butchers.
- The spores enter the body through cuts or scratches in the skin and cause a local infection that if not controlled may spread throughout the body. The digestive form occurs when the spores are eaten. Tragically people who lose their animals may also lose their lives when they consume meat from an animal that has died of anthrax.
- The deadliest form of the diseases is the pulmonary form also known as 'wool sorters disease'. The pulmonary form is rare under natural circumstances. However, it is the form that occurs when anthrax spores are laboratory engineered to act as biological weapon.

#### **Differential Diagnosis**

- Black quarter (Clostridium chauvoei)
- Peracute bovine Babesiosis
- Chemical poisoning (heavy metals, other poisons)
- Plant poisoning
- Snake bite,
- lightning strike
- Metabolic disorders such as lactic acidosis, magnesium deficiency and bloat.

#### Post mortem lesions

- Do Not Open Carcass with dark blood that does not clot oozing from all orifices and with rigor mortis absent or incomplete, has marked bloating and rapid decomposition.
- If the carcass is inadvertently opened, septicaemic lesions are common and include blood that is dark and thickened and fails

to clot readily. Haemorrhages of various sizes are common on the serosal surfaces of the abdomen and thorax as well as on the epicardium and endocardium. Oedematous, red-tinged effusions commonly are present under the serosa of various organs, between skeletal muscle groups, and in the subcutis.

• The most characteristic necropsy finding is a markedly enlarged black to dark red spleen

#### Diagnosis

- Clinical signs of sudden death with blood oozing from all orifices, rapid decomposition of carcass that lacks rigor mortis development.
   Sample Collection
- Anthrax is diagnosed by examining blood smears for the presence of the bacteria. Samples must be collected carefully to avoid contamination of the environment and to prevent human exposure to the bacteria.
- Giemsa stained blood smears made from a cut surface of the base of the ear of a fresh carcasses contains large numbers of B. anthracis vegetative forms which can be seen under a microscope.
- Cultured and isolation of bacteria is prohibited.
- Blood smear is the main confirmatory diagnostic test for laboratories.

#### **Treatment and Control**

- Anthrax is susceptible to most antibiotics especially penicillin group. Prompt treatment of animals at the earliest signs can be effective. Treatment with antibiotics counteracts the vaccine though, so antibiotics should not be given to animals that are recently vaccinated.
- Vaccination is the main control strategy and is done once a year. The vaccine Blanthrax<sup>™</sup> that combines anthrax and black quarter is the most common in Kenya and is available from KEVEVAPI.
- Proper disposal of dead animals is criticalcarcass should not be opened, since exposure to oxygen will allow the vegetative form of the bacteria to form spores, infected areas should be quarantined until all susceptible animals
are vaccinated and all carcasses disposed of preferably by incineration or alternatively, by deep burying with quick lime poured over the carcass.



Anthrax Transmission cycle in cattle Picture source: https://infonet-biovision.org/AnimalHealth/Diseases-Killing-very-fast-Killer-diseases



Blood smear stained with Giemsa showing characteristic Bacillus anthracis bacteria Picture source: https://repository.up.ac.za/handle/2263/32656

# Rabies

## Other names: Mad dog disease, Hydrophobia

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Nyanye	Nyanye	Sugeri Kare	Nkwaang	Nkwaang	Sigire

#### Definition:

• Rabies is an acute, progressive viral encephalomyelitis that principally affects carnivores and bats, although any mammal can be affected. The disease is fatal once clinical signs appear.

#### Causative agent

- Virus- RNA virus of the genus Lyssavirus in the family Rhabdoviridae
- Lyssaviruses are highly neurotropic and have a long incubation period
- There are over 16 different lyssaviruses
- Rabies virus can be transmitted between mammals, whether they belong to the same or different species

#### **Transmission and Predisposing factors**

- All mammals are thought to be susceptible to the rabies virus the disease is a fatal zoonosis that causes encephalitis. It is 100% fatal.
- Following the bite of an infected animal, the virus is placed under the skin surface. Here the virus multiplies for a period of days or weeks. It then travels along the peripheral nerves and via the axons to the dorsal root ganglia. Here further multiplication of the virus occurs. The virus passes to the central nervous system and is capable of passing from cell to cell. The final path of its journey takes it back along axons to the salivary glands and many other deep organs like the heart, skeletal tissue, kidney and cornea.
- The virus is then transmissible through saliva after 3 days to up to years. Most commonly between 1 to 3 months. The incubation

period is directly related to the extent of the inoculation and the closeness of the brain. Thus bites on the face and neck are particularly significant and will, in general, be associated with a short incubation period. The incubation period is both prolonged and variable. Typically, rabies virus remains at the inoculation site for a considerable time. The unusual length of the incubation period helps to explain the effective action of local infiltration of rabies immune globulin during human post exposure prophylaxis, even days after exposure. Most rabies cases in dogs develop within 21–80 days after exposure.

- There reports of infection in animals (carnivores) due to Ingestion of infected meat but this has not been reported in humans.
- Human to human spread is possible in theory but has not been reported to date. All ages are at equal risk.
- Transmission is mainly through the saliva of an infected animal. Saliva becomes infectious a few days prior to the onset of clinical signs. Infection occurs primarily via bite wounds, or infected saliva entering an open cut or wound or mucous membrane, such as those in the mouth, nasal cavity or eyes.
- Occasional, though rare, transmission by inhalation of infected aerosol has been described. This is common when humans enter caves or houses with bat faeces (Guano). There are reports of laboratory workers getting infected by brain tissue (hence virus isolation or brain tissue handling should not be attempted unless in a Biosecurity level 3 or 4 laboratories.

#### Occurrence

- Rabies is found throughout the world, with the exception of Antarctica
- Rabies is a zoonotic disease that can affect all mammals. Carnivores circulate different rabies virus (RABV) variants and act as a reservoir for rabies.
- Rabies infection is maintained in two epidemiological cycles, one urban and one sylvatic. In the urban rabies cycle, dogs are the main reservoir host. This cycle predominates in areas of Africa, Asia, and Central and South America. Canine-mediated rabies is one of the most important zoonosis and is estimated to cause up to 70,000 human deaths per year mostly affecting people in rural areas and especially children.
- The sylvatic (or wildlife) cycle is the predominant cycle in the northern hemisphere.

#### Main clinical signs

- On set of clinical signs vary with the distance of infecting bite to the nervous tissue it may take a few days to more than a year.
- Clinical course may be divided into three phases—prodromal, furious and paralytic. However, this division is of limited practical value because of the variability of signs and the irregular lengths of the phases. During the prodromal period, which lasts approximately 1 to 3 days, animals show only vague nonspecific signs, which intensify rapidly. The disease progresses rapidly after the onset of paralysis, and death is virtually certain a few days thereafter. Some animals die rapidly without marked clinical signs.
- Furious Rabies: This is the most common form (80%).In this form animals are aggressive is pronounced. This is the classic "mad-dog syndrome," although it may be seen in all species. There is rarely evidence of paralysis during this stage. The animal becomes irritable and, with the slightest provocation, viciously and aggressively uses its teeth, claws, horns, or hooves. The posture and expression is one of alertness and anxiety, with pupils dilated. Noise may invite attack. Such animals lose

caution and fear of people and other animals.

- Carnivores with the furious form of rabies frequently roam extensively, attacking other animals, including people, and any moving object. They commonly swallow foreign objects. As the disease progresses, muscular incoordination and seizures are common. Death results from progressive paralysis.
- Paralytic Rabies: manifest by ataxia and paralysis of the throat and masseter muscles, often with profuse salivation and the inability to swallow. Dropping of the lower jaw is common in dogs. Owners frequently examine the mouth of dogs and livestock searching for a foreign body or administer medication with their bare hands, thereby exposing themselves to rabies. These animals may not be vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, and coma and death follow in a few hours.
- Species Variations- Cattle with furious rabies can be dangerous, attacking and pursuing people and other animals. Lactation ceases abruptly in dairy cattle. The usual placid expression is replaced by one of alertness. The eyes and ears follow sounds and movement. A common clinical sign is a characteristic abnormal bellowing (sounds like the animal has a hoarse voice), which may continue intermittently until shortly before death. Horses and donkey's mules frequently show evidence of distress and extreme agitation. These signs, especially when accompanied by rolling, may be interpreted as evidence of colic. As in other species, horses may bite or strike viciously and, because of their size and strength, become unmanageable. Rabies should be suspected in terrestrial wildlife acting abnormally. The same is true of bats that can be seen flying in the daytime, resting on the ground, paralyzed and unable to fly, attacking people or other animals, or fighting.

#### Differential Diagnosis

- Heart water
- · Chemical toxicity
- Acute lead poisoning,
- · Acute salt (sodium toxicosis) and water

deprivation

(Cerebrocortical necrosis)
 Polioencephalomalacia (PEM) is an important
 neurologic disease of ruminants that is
 seen worldwide in Cattle, sheep, goats, and
 camelids. PEM associated with two types of
 dietary risks: altered thiamine status and high
 sulphur intake.

#### Diagnosis

- Immunofluorescence microscopy (direct fluorescent antibody test) on fresh brain tissue is the test of choice. Brain tissues examined must include the medulla oblongata and cerebellum (and should be preserved by refrigeration with wet ice or cold packs). Immunofluorescence microscopy on fresh brain tissue allows direct visual observation of a specific antigen-antibody reaction.
- Diagnosis should only be done by a qualified laboratory, designated by the local or state health department in accordance with established standardized national protocols for such viral testing.
- Confirmatory tests include PCR and histology of brain tissue.
- A definitive pathologic diagnosis of rabies can be based on the findings of Negri bodies in the brain or the spinal cord. Negri bodies contain

rabies virus antigens and can be demonstrated by immunofluorescence. Both Negri bodies and rabies antigens can usually be found in animals or humans infected with rabies, but they are rarely found in bats.

#### **Treatment and Control**

- Vaccination and registration of dogs, with control of stray populations
- Oral vaccination of wildlife can reduce rabies in reservoir populations
- Notification of suspected cases, and euthanasia of dogs with clinical signs and dogs bitten by a suspected rabid animal
- Reduction of contact rates between susceptible dogs by leash laws, dog movement control, and quarantine
- Mass immunization of dogs by campaigns and by continuing vaccination of young dogs
- Stray dog control and euthanasia of unvaccinated dogs
- Available animal vaccines use killed vaccines. A rabies control programme should consider: Socio-cultural framework, including public awareness, the promotion of responsible pet ownership and animal welfare. This includes annual rabies vaccination and neuter of animals.



Pathogenesis and spread of rabies virus in animals from the bite site to the central nervous system

Picture source: https://www. intechopen.com/online-first/77118





The brain tissue is tested for the presence of Negri bodies at the cerebellum or brain stem (brain stem is made up of the midbrain, pons and medulla)

Direct fluorescent antibody test that is negative (Left) and positive (Right ) for the rabies virus.

Histology brain tissue with neuron without Negri bodies (right) and with Negri bodies (left) Picture source: http:// uwrfbio353koob.blogspot. com/p/histology.html

A cow bitten by a Rabid donkey in Kajiado county, 2021

Photo credit: Dr. Pauline Gitonga



# **Rift Valley Fever**

#### Definition

 Rift Valley fever (RVF) is a peracute or acute, mosquito-borne, zoonotic disease of domestic and wild ruminants in Africa, Madagascar, and the Arabian Peninsula. Large outbreaks of clinical disease are usually associated with heavy rainfall and localized flooding. During epidemics, the occurrence of abortions in livestock and deaths among young animals, particularly lambs, together with an influenzalike disease in people, is characteristic. However, infections are frequently subclinical or mild.

#### Causative agent:

- RVF virus (RFV)belongs to the genus Phlebovirus family Bunyavirus.
- The RNA virus has little genetic diversity, but has different pathogenicity.
- The virus is transmitted by mosquitoes and occurs in inter epizootic intervals (10 years). RVF has caused serious disease in laboratory workers and infected samples should be handled at biosecurity level 2 and 3 laboratories.

#### Occurrence

• Endemic in many tropical and subtropical regions of Africa, Madagascar, and the Arabian Peninsula. RVF was originally confined to the Rift Valley region of eastern and southern Africa, the virus recently expanded its range, with major outbreaks seen in Egypt since 1977, West Africa since 1987, Madagascar since 1990, and the Arabian Peninsula in

2000. Particularly large epidemics with large numbers of human cases occurred in Egypt in 1977–1978 and in Kenya in 2006–2007.

- RVF is considered a threat to regions where competent mosquito vectors are present.
- Sporadic, large epidemics have occurred at 5–10 year intervals in drier areas of eastern Africa, and less frequently in southern Africa. Outbreaks are usually associated with periods of abnormally heavy rainfall or, in some cases, with localized flooding due to dam building or flood irrigation.
- During inter-epidemic periods, the virus remains dormant in eggs of floodwaterbreeding Aedes mosquitoes in the dry soil of small dambos or pans. This transovarial transmission is believed to be the most important inter-epidemic survival strategy for the virus.
- However, inapparent cycling of the virus between vectors and wild or domestic mammalian hosts has been shown to occur in many areas. RVF virus may also spread by movement of viremic animals and possibly by wind-borne mosquitoes. When emergence of infected mosquitoes, or introduction of virus to an area, coincides with abnormally wet conditions and the presence of a highly susceptible host population, a large epidemic may ensue when the virus is amplified in ruminants and spread locally by many species of mosquitoes or mechanically by other insects.
- In Africa where insect vectors are present continuously, seasonality is not seen.

#### **Transmission and Predisposing factors**

- People are readily infected through blood aerosols from infected animals during slaughter, or by exposure to infected animal tissues, aborted foetuses, mosquito bites, and laboratory procedures.
- Therefore, veterinarians, farm labourers, and abattoir workers are particularly at risk.
- People can also act as amplifying hosts and introduce the disease (via mosquitoes) to animals in uninfected areas.
- Elnino climatic conditions favours breeding of mosquito vectors (Aedes, Anopheles, Culex, Eretmapodites and Mansonia). Mosquito species act as reservoirs for RVF virus during inter-epidemic periods and increased precipitation in dry areas leads to an explosive hatching of mosquito eggs; many of which harbour RVF virus.
- During inter epidemic period immuno-naïve domestic ruminants' animal populations are present and when Infected Aedes feed on domestic ruminants they act as biological amplifier of RVF virus.
- Satellite imaging has been used to confirm historic importance of precipitation in RVF outbreaks and in forecasting high-risk areas for future outbreaks.
- There are more human deaths from communities that consume raw infected meat during home slaughters during outbreaks.
- There is possible human transmission through consumption of raw milk
- Virus regularly circulates in endemic areas between wild ruminants and mosquitoes' disease is usually inapparent.

#### **Clinical signs**

- Storm of abortion in goats, sheep (mainly), cattle and camel,
- · High mortalities in young lambs
- In mature ruminants- bloody nasal discharges, haemorrhagic fetid diarrhoea, dysgalactia and jaundice during abnormally prolonged heavy rainfall.
- Mortality rate in lambs is high (90-100%), goat

kids and calves (70%), adult sheep and s 10-30%, adult goats, cattle, camels (5-10%).

#### Humans

 Influenza-like syndrome: fever (37.8–40°C), headache, muscular pain, weakness, nausea and epigastric discomfort, photophobia and recovery occurs within 4–7 days. Complications of retinopathy, blindness, meningo-encephalitis, haemorrhagic syndrome with jaundice, skin petechial spots and death.

#### Post mortem lesions

- Appropriate bio-security and biosafety must be exercised as RVF is a potent zoonotic pathogen.
- Focal or generalised hepatic necrosis (white necrotic foci of about 1 mm in diameter).
- Congestion, enlargement, and discoloration of liver with sub capsular haemorrhages.
- Brown-yellowish colour of liver in aborted foetuses.
- Widespread cutaneous haemorrhages, petechial to ecchymotic haemorrhages on parietal and visceral serosa membranes.
- Haemorrhagic enteritis and Icterus

#### **Differential Diagnosis**

- Bluetongue
- · Enterotoxaemia of sheep
- Ephemeral fever
- Brucellosis
- Leptospirosis
- Q-fever
- · Plant poisoning
- Bacterial septicaemias
- Anthrax

#### Diagnosis

Field based diagnosis

 Clinical signs- storm of abortion in goats, sheep (mainly), cattle and camel, high mortalities in young animals (lambs and kids), bloody nasal discharges in mature animals, haemorrhagic diarrhoea and jaundice during abnormally prolonged heavy rainfall.

#### Sample Collection

- Donning of full personal protective clothing is a must
- Most laboratories will require serum or blood to conduct competitive ELISA (antibody testing) or PCR tests (antigen identification). IgM antibodies signify a recent infection while IgG a past infection.

## **Treatment and Control**

- There is no treatment of sick animals, supportive therapy to animals with clinical signs. Care should be taken not to spread infection through re-use of hypodermic needles or self-infection during treatment of the sick animals.
- Conduct Participatory disease surveillance (PDS) Surveillance during inter-epizootic period. Targets the period prior to and during the early warning of heavy rain by international and national meteorological agencies. It will be crucial at this point to assess the capacity in critical areas such as coordination, sero-surveillance, laboratory diagnosis and response.
- Develop platforms for crowd sourcing of information from the community. Creation of public awareness on the risk factors and need to report the occurrence of risk factors such as

heavy rains, flooding, abortions in animals or mosquito build up should be encouraged.

- NB: Vaccination should not be done in areas with suspected clinical disease. This is because vaccination team have a high likelihood of spreading infection through equipment and have a high chance of self-infection as most animals having no clinical sign but are amplifying the virus.
- Quarantine and close of slaughter slabs and houses, livestock markets and movement restriction
- Scale up public health messages to ensure people stop home slaughter or touching aborted foetuses without protection
- During the inter-epizootic phase without predisposing factors, the following control measures may be applied:
- Vaccination in identified high risk areas-RIFTVAX TM®- is a freeze-dried live attenuated vaccine prepared from Smithburn's attenuated strain of RVF virus.
- Public education should inform livestock keepers that vaccination is associated sometimes with early embryonic death, congenital CNS anomalies and arthrogryposis, or abortion or stillbirths.



Rift Valley Fever (RVF) Transmission cycle Picture source: https://veterinaryresearch.biomedcentral.com/articles/10.1186/1297-9716-44-78#Fig2



RVF infection in a cow placenta with oedema and haemorrhages (hydramnion) and aborted emphysematous foetus with 'meconium staining (left)



Small intestine of a cow (Left) with marked mesenteric and serosal oedema and New-born Lamb intestines (Right) with haemorrhages in the intestine Picture source: https://journals.plos.org/plosntds/article?id=10.1371/journal.pntd.0006353



Rift Valley fever (RVF) markedly enlarged ruminant liver, yellowbrown discolouration of the organ, and congested patches scattered throughout the organ

Picture source: https://repository.up.ac.za/ handle/2263/32685

# Brucellosis

Other names: Contagious abortion, Bang's disease in livestock and "undulant fever", "Mediterranean fever" or "Malta fever" in humans

Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Dukub Anani	Dukub Anani	Sugeri Hanut	Nkeeya E Kule	Edeke-Angakile	Bilbilaam

# Definition

- Zoonosis transmitted by direct or indirect contact with infected animals or their products. It affects people of all age groups and of both sexes.
- Duration of human illness and its long convalescence means that brucellosis is an important economic problem for the patient because of time lost from normal activities. In many underserved regions the delay in diagnosis and endemic status of the disease in livestock means that the disease remains a constant threat to human welfare, particularly for those in the most vulnerable socioeconomic sections of the population.
- Brucellosis is primarily a disease of animals, especially domesticated livestock, with humans being accidental host.

# Causative agent:

- Brucella organisms are gram-negative coccobacilli and may be present in high concentration in blood cultures drawn early in the disease process.
- Six main specie": B. abortus, B. suis, B. melitensis, B. neotomae, B. ovis, B. canis. Brucella melitensis and Brucella abortus are the most important species in Kenya.

# Occurrence

• Disease occurs worldwide, except in countries where bovine brucellosis (B. abortus) has been eradicated. This is usually defined as the absence of any reported cases for at least five years. These countries include Australia, Canada, Cyprus, Denmark, Finland, the Netherlands, New Zealand, Norway, Sweden and the United Kingdom (WOAH Reports).

- Mediterranean countries of Europe, northern and eastern Africa, Near East countries, India, Central Asia, Mexico and Central and South America are especially affected. While B. melitensis has never been detected in some countries, there are no reliable reports that it has ever been eradicated from small ruminants.
- The sources of infection for humans and the species of Brucella spp. found vary according to geographical region. It is usually either an occupational or a food-borne infection. Both sporadic cases and epidemics occur in humans, but often the disease or infection is either unrecognized, or, if diagnosed, not reported to the public health authorities.
- Brucellosis is a notifiable disease and must be reported to the Kenya DVS who then reports to WOAH.

# Transmission and Predisposing factors

- Infection spreads rapidly and causes many abortions in unvaccinated herds especially in cattle.
- In endemic areas infected animals typically aborts only once after exposure; subsequent gestations and lactations appear normal. After exposure, animal become bacteraemic for a short period and spontaneously recover.
- The incubation period is variable and is inversely related to stage of gestation at time of exposure. Organisms are shed in milk and uterine discharges, and the cow may become temporarily infertile. Bacteria may be found in the uterus during pregnancy, uterine involution, and infrequently, for a prolonged time in the non-gravid uterus.
- Shedding from the vagina largely disappears with the cessation of fluids after parturition.

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- Some infected cows that previously aborted shed Brucella from the uterus at subsequent normal parturitions.
- Organisms are shed in milk for a variable length of time—in most cattle for life. B abortus can frequently be isolated from secretions of nonlactating udders.

#### Main clinical signs

- Abortion
- Stillborn
- Weak calf born
- Retention of foetal membranes
- Swollen testicles in bulls
- General health of animal is not impaired in uncomplicated abortions or orchitis
- Testicular abscesses may occur. Longstanding infections may result in arthritic joints in some cattle

#### **Differential Diagnosis**

- Rift valley Fever (RVF)- abortion in animals and fever in humans
- Q fever- abortion in animals and fever in humans
- Leptospirosis- abortion in livestock
- Listeriosis- abortion in livestock

- Campylobacteriosis and chlamydia infection -Infertility and orchitis in livestock
- Blue tongue for small ruminants- abortion

#### Diagnosis

- Culture is the hold standard for confirmatory diagnosis. Culture requires a BSL laboratory. I
- ELISA or PCR can also be used as confirmatory tests.

#### Control

- The search for ideal brucellosis vaccines remains active today. Currently, no licensed human or canine anti-brucellosis vaccines are available. In bovines, the most successful vaccine (S19) and is only used in calves, as adult vaccination results in orchitis in male, prolonged infection, and possible abortion complications in pregnant female cattle. Another widely deployed vaccine (RB51) has a low protective efficacy. In Kenya, there is no vaccine approved for use by the DVS.
- Methods of prevention include health education to reduce occupational and foodborne risks, including pasteurization of all dairy products and handling aborted foetuses with protective gloves.



Rose Bengal or card test showing negative (left) and positive (right) reactions



Rapid Brucella antibody chromatography test

#### Further reading

1. MSD Veterinary Manual https://www.msdvetmanual.com/

2. Peter J. Fernández & William R. White (2010). Atlas of Transboundary Animal Diseases https://WOAHbulletin.com/?panorama=atlas-of-transboundary-animal-diseases

3. United Nations Environment Programme and International Livestock Research Institute (2020). Preventing the Next Pandemic: Zoonotic diseases and how to break the chain of transmission. Nairobi, Kenya. https://www.unep.org/resources/report/preventing-future-zoonotic-disease-outbreaks-protecting-environment-animals-and

# Annex 3: Antimicrobial Resistance

#### Definition

- Antimicrobial resistance (AMR) is the ability of bacteria, fungi, viruses and parasites to develop biological and propagatable ability to resist the effects antimicrobial agents (antibiotics, antifungals, antivirals, antimalarial, and anthelmintics). As a result, the medicines become ineffective and infections persist in the body, increasing the risk of spread to others.
- Resistant diseases agents have the ability to multiply or persist in the presence of increased level of an antimicrobial agent relative to their susceptible counterpart of the same species.
- Antimicrobial resistance (AMR), is described as a silent pandemic at both national and global levels that is threatening the advancement of antibiotics use in managing infectious diseases in both humans and livestock.
- Emergence of antibiotic resistant bacterial strains are a product of both continuous evolution and un-checked antimicrobial usage (AMU).
- Significant emphasis has been given to AMR in humans, however, trends of AMU in livestock is often not emphasized.
- There is need for global co-operative efforts at individual, community, local, regional, national, and international level to address AMR.
- In order to meet this global goal, a Tripartite Alliance was formed between the WHO, FAO, and WOAH with One Health approach. The tripartite alliance published the Global Action Plan on AMR in 2015. Likewise, FAO also launched its AMR Strategy in 2016 to back the proper execution of the WHO Global Action Plan in food and agricultural sectors. The WHO Global Action Plan emphasizes on increased awareness and understanding on AMU and associated AMR; build up knowledge regarding AMR through proper surveillance and research; optimal and rational use of antibiotics; lowering the incidence of infectious diseases; and organizing resources, research, and development for proper integrated prevention and containment of antibiotic resistance.



# Factors leading to antimicrobial resistance (AMR) in livestock

Conceptual representation of possible movement of antibioticresistant bacterial strains/genes between different



Critically	Critically Important		Important	Currently not used in
Highest priority	High Priority			humans
• Cephalosporins (3rd, 4th,	<ul> <li>Aminoglycosides</li> </ul>	Amphenicals	Aminocyclitols	• Bambermycins
5th Gen.)	Ansamycins	• Cephalosporins (1st	Cyclic Polypeptides	• Quinoxalines
Fluro And Other	Carbapenems And Other	& 2nd Gen.)	Nitrofurantoin	<ul> <li>Ionophores</li> </ul>
Quinolones	Penems	• Lincosamides	<ul> <li>Nitromidazoles</li> </ul>	Orthosomycins
<ul> <li>Glycopeptides</li> </ul>	<ul> <li>Glycylcylines</li> </ul>	• Penicillins (Anti-	<ul> <li>Pleuromitilins</li> </ul>	
Macrolides And Ketolides	<ul> <li>Lipopeptides</li> </ul>	Staphylococcal)		
<ul> <li>Polymyxins</li> </ul>	Monobactams	Pseudomonic Acid		
	<ul> <li>Oxazolidinones</li> </ul>	Riminofenazines		
	• Penicillins (Natural	Steroids		
	Aminopenicillins &	Antibacterials		
	Antipseudomonal)	<ul> <li>Streptogrmins</li> </ul>		
	Phosphonic Acid	<ul> <li>Sulfonamides</li> </ul>		
	Derivertives	Sulfones		
	<ul> <li>Tuberculosis And Other</li> </ul>	<ul> <li>Tertacyclines</li> </ul>		
	Mycobacterial Drugs			

# Critically important antibiotics for human use based on WHO

Info source: https://www.frontiersin.org/articles/10.3389/fvets.2017.00237/full

#### Antibiotics Mode of Action

- Understanding the mechanism of action of antibiotics by animal health service providers is a first step in mitigating AMR.
- Ability of an antimicrobial drug to arrest the growth of or kill bacteria is dependent upon its mechanism of action and the concentration that the drug attains at the infection site. When a drug is introduced into the body, it is rapidly carried through the bloodstream to the liver, kidneys, and other organs that can chemically change or reduce its antibacterial activity and promote its excretion.
- The pharmacokinetic process (movement of drugs within the body) include;
- (1) absorption from its site of administration(2) distribution throughout the body and its

elimination by (3) biochemical metabolism, and (4) excretion through the urine, bile, or other routes are collectively given the acronym ADME.

- Bactericidal Vs. Bacteriostatic- Antibiotics can be divided to two groups on the basis of their effect on microbial cells through two main mechanisms, which are either bactericidal or bacteriostatic. Bactericidal antibiotics kill the bacteria and bacteriostatic antibiotics suppress the growth of bacteria (keep them in the stationary phase of growth).
- Bactericidal antibiotics cause cell death by preventing cell wall synthesis of the bacterial cell. This reduces the number of bacterial cells present.

- Bacteriostatic antibiotics prevent further cell growth. This is achieved by inhibiting protein synthesis, DNA replication or other cellular metabolic actions, although it does not cause bacterial cell death. This effect is reversible, unlike the action of bactericidal drugs. The amount of bacterial cells, therefore, remains the same, although in a static phase. The bacteriostatic drugs rely on the host immune system to overcome or clear the bacteria held in static phase.
- Clinical outcome for antibiotics is affected by factors that include infecting bacterial density, host immune response, underlying disease or the site of infection for example if infection is in the organs with physiological barriers like the brain or infection has caused formation of thick granulomatous capsule that is not penetrable.

- Efficacy- bactericidal antibiotics are effective against dividing cells its efficacy may be reduced if treatment is paired with the slower growth caused by bacteriostatic drugs. This is why practitioners should not mix bactericidal and bacteriostatic drugs.
- Side effects- bactericidal drugs have a rapid mode of action and can stimulate severe inflammation due to the release of bacterial cell contents upon cell death, which can result in toxic shock syndrome. This means that in certain diseases caused by bacteria that release toxins, bacteriostatic drugs are often preferred so as to prevent fatal toxaemia syndrome. Bacteriostatic drugs have no side effects other than preventing bacterial growth, although if removed from the system their effects can be reversed and the bacteria can start multiplying (Main reason why dosage regime and length should be followed).

#### **Further Reading**

- Sharma C, Rokana N, Chandra M, Singh BP, Gulhane RD, Gill JPS, Ray P, Puniya AK and Panwar H (2018) Antimicrobial Resistance: Its Surveillance, Impact, and Alternative Management Strategies in Dairy Animals. Front. Vet. Sci. 4:237. https://doi.org/10.3389/fvets.2017.00237
   Difference between bactericidal and bacteriostatic antibiotics http://www.differencebetween.net/science/difference-between-
- bactericidal-and-bacteriostatic/#ixzz74urGEVrM
- 3. WHO guidelines on use of medically important antimicrobials in food-producing animals. Geneva: World Health Organization; 2017. Licence: CC BY-NC-SA 3.0 IGO.
- https://apps.who.int/iris/bitstream/handle/10665/258970/9789241550130-eng.pdf

	Examples of common	y used veterinary	antibiotic and	mode of action
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Class of antibiotic	Mode of action	Spectrum of activity	Veterinary use in ruminants
Aminoglycosides Gentamicin, tobramycin, amikacin, streptomycin, kanamycin	Bactericidal – inhibit protein synthesis	Broad spectrum except anaerobic bacteria	Enterobacteriaceae. Infections and severe sepsis cause by Gram-negative aerobes.
β-Lactam Penicillins: penicillin G, ampicillin, amoxicillin, carbenicillin 1st, 2nd, 3rd, 4th Generation Cephalosporins*	Bactericidal- Inhibit of cell wall synthesis.	Broad spectrum for 2nd, 3rd, and 4th generation cephalosporins Narrow spectrum (Gram positive mainly) for penicillins and 1st generation cephalosporins	Anthrax, listeriosis, leptospirosis, clostridial and corynebacterial infections; streptococcal mastitis, keratoconjuntivitis
Fluoroquinolones Enrofloxacin, ciprofloxacin,	Bactericidal- Inhibition of nucleic acid synthesis	Broad spectrum: 3rd-generation fluoroquinolones Narrow spectrum: other fluoroquinolones	Acute respiratory disease, infections with E. coli, Salmonella, Mycoplasma, mastitis, metritis, conjunctivitis
Macrolides Erythromycin, Tylosin, Spiramycin, Tilmicosin, Tulathromycin	Bacteriostatic- inhibit protein synthesis- can be bactericidal at high concentrations or if there is low numbers of susceptible bacterial	Narrow spectrum	Erythromycin: drug of choice against Campylobacter jejuni. alternative to penicillin in penicillin-allergic animals and second choice for anaerobic infections. Tylosin against Mannheimia, Actinobaciullus, Pasteurella, Mycoplasma.
Sulphonamides Sulfadiazine, sulfamethoxazole, sulfadoxine	Bacteriostatic- inhibit folic acid synthesis	Broad-spectrum: affects Gram- positive. Gram-negative bacteria, toxoplasma and protozoal agents	First line of drugs used in ruminants and poultry to manage diarrhoea and coccidiosis Also used for infections due to E. coli Haemophilus Salmonella Pasteurella Staphyloccocus Streptococcus
Diaminopyrimidines Trimethoprim,	Diaminopyrimidines Trimethoprim,	Broad spectrum: affects Gram-positive and many Gram- negative bacteria Act synergistically (and becomes bactericidal) in combination with sulphonamides	
Tetracyclines Chlortetracycline, oxytetracycline, doxycycline, minocycline	Bacteriostatic – Inhibits protein synthesis	Broad spectrum. Gram-positive, Gram-negative bacteria, atypical organisms such as chlamydiae, mycoplasmas, rickettsiae, and protozoan parasites.	Ehrlichiosis, leptospirosis, listeriosis, rickettsiosis, Anaplasmosis, Heart water, ECF vaccination

# Annex 4: Calculating drug dosage

The Volume of injectable veterinary drug to be administered can be calculated as follows (Nair & Jacob, 2016);

Injection Volume (ml) = Dosage (mg/kg) X Body weight (kg) Concentration (mg/ml)

#### Where;

Volume is measured in ml

Dosage is measured in mg/kg body weight- usually stated in the drug label or insert kit

Body weight is measured in kg

Concentration is measured in mg/ml- usually stated in the drug label or insert kit

Therefore: To calculate the volume to inject a 150 kg cow for the different oxytetracycline preparation

if given a recommended dosage of 10mg/kg will be;

- 5% preparation =  $(10 \times 150) \div 50 = 30$  mls
- 10% preparation=  $(10 \times 150) \div 100 = 15$  mls
- 20% preparation= (10× 150) ÷ 200 = 7.5 mls
- 30% preparation=  $(10 \times 150) \div 300 = 1$  ml

#### Estimating weight of animal



Weight measurement using heart girth in cattle (top) and goat (down)

#### Picture sources:

http://bairnsley.com/Weight%20by%20Girth.htm (Cattle) http://www.infovets.com/books/smrm/C/C098.htm (Goat)



Estimating live-weight of goats (top) and cattle (bottom) using weight estimation formul

- MEASURE: (A-B) body length measured from point of shoulder to point-of-rump or pin bone.
- MEASURE: (C) heart girth circumference, around the body behind the shoulder, over the heart area and very close to the forelegs. When measuring a sheep, compress the sheep's wool to obtain a circumference that reflects the body and not the body plus the wool.
- Using these 2 measurements, calculate body weight estimate with this formula:
- o (Heart girth (inches) X heart girth (inches) X body length (inches) ÷ 300 = approx. animal weight (pounds).
- Convert to kilograms using formula- 1 kilogram is equivalent to 2.205 so divide answer by 2.2

Example; if a sheep has a heart girth equal to 35 inches and a body length equal to 30 inches, the calculation would be (35 x 35 x 30) / 300 = 122 lbs

converted to kgs 122÷2.2= 55.45 kgs.

## DONKEY WEIGHT ESTIMATOR

To estimate a donkey's weight using the diagram below mark the height and heart girth measurements on the correct axis. Then draw a line between the two. The donkey's weight is indicated by where the line crosses the weight. For example, a donkey 104cm tall (a) and with a heart girth 122cm (b) should weigh 181kg (c).



Measuring height (cm)

Measuring girth (cm)



Picture source: http://bairnsley.com/Weight%20by%20Girth.htm

Whilst the weight estimator is an effective tool to estimate weight it's accuracy cannot be guaranteed.

Weight extinuation table for donkeys under 2 years

Number (1971) 75 77 75 77 76 77 86 75 87 88 87 87 87 88 87 88 87 98 75 76 76 76 89 100 Weige Syst. 48 47 48 51 51 51 57 10 41 51 61 67 68 75 78 76 78 78 78 68 68 68 50 50

# Annex 5: Photo Gallery of Medicinal and Poisonous plants in Marsabit County

Pictures credit:

African Plants a Photo Guide and World Agroforestry websites and website link to Marsabit county plants http://palkowitschia.cz/sukulenty/en/travelling.php?dest=kenya&cat=flora&page=7

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Zanthoxylum usambarense (Prickly ash)	GADDA	GADDA	GATHAHA	LOISUK	EUSUGU	-
		-	the of	A CAR		

Zanthoxylum usambarense bark, ripe fruit opening to release seed and leaves

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Acacia Nubica	Wanga	Wanga	Holiya	Ldepe	Eiyellel	Morte
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Acacia nubica leaves and pods

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Acacia Tortilis	Dadach	Dadach	Dahar	Ltepes	Ewoi	Seechgeebe
(Umbrella Thorn)						



Acacia tortilis whole tree, leaves, straight and curved thorns, flowers and pods

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Aloe Scabrifolia	Harges	Harges	Yaharr	Sukuroi	Echuchuka	Dhelbo



Aloe scabrifolia, whole plant

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Moringa Stenopetala	Safara	Safara	Lorsanjo	Waddessa	Etebusoit	Hocholoch
(Cabbage Tree)				Lorsanjo		
				Chibulukwa		



Moringa stenopetala, whole tree, leaves, flowers, green pods and mature pods with seeds

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Ficus sur	ODHA	ODHA	ODHA	LNGABOLI	ECHOKE	-
(Bush fig, Cape fig						
Broom cluster fig)						



Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach		
Azadirachta indica	λαγά τι ττρά τητι							
(Neem tree)			WWAR	UDAINI				



Azadirachta indica (Neem tree), whole tree, branches, leaves, fruits and flowers

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Salvadora Passica	Huda	Huda	Hagay	Sakotei	Esekon	Nyedhe- Singular
(Toothbrush Tree/ Salt	Aadee	Aadee				Nyaa- Plural
Bush)						



Salvadora passica (Toothbrush tree/ Salt bush), whole tree leaves and fruits

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Euphobia Species	Adama	Adama	Lekule	Ltilemani	Emus	-



Euphorbia kalisina (Kali sana) in Marsabit County

Euphorbia marsabitensis in Marsabit County



Euphorbia Magnicapsula in Marsabit County

Botanical /Common name	Borana	Gabra	Rendille	Samburu	Turkana	Daasanach
Commiphora Species	Warra	Warra	Warra	Warra	Wuara	Kerech



Commiphora species in Marsabit County



Chewing Tobacco (Nicotiana tabacum) Photo credit: Gosh Kwanyang (CDR ILLeret, Marsabit County)



Solanum species (Poison apple) leaves, flowers and fruits Solanum mature fruits causes camel gastrointestinal tract disturbances.



Capparis tomentosa (woolly caper bush or African caper) flower, fruits and leaves The plant causes paralysis in camels.



Lantana camara (Lantana) is an invasive weed Long term consumption of Lantana causes photosensitisation in cattle and the berries are toxic, and can result in liver failure and death.



Prosopis consumption by goats and donkeys causes gastrointestinal impaction. The pods that have high sugar content are associated with rotting of teeth in goats.



#### Euphorbia species

Euphorbia plant species has Irritant toxins that have not been identified. In camel's long term consumption causes gastro intestinal tract irritation that manifests as diarrhoea. Some Euphorbia species have Cyanogenic glycosides that can cause sudden death in livestock.

#### Nitrate poisoning

Animal health staff in the county requested the handbook to include literature on discuss Nitrate poisoning. Nitrate poisoning is common in ruminants consuming certain forage or water that contains an excessive amount of nitrate. Donkeys are less sensitive to nitrate toxicity since they are hindgut ferments and do not have a rumen. In normal conditions, nitrate ingested by ruminant livestock, like cattle, sheep and goats, is converted to ammonia and finally to bacterial protein in the rumen. If high amounts of nitrate are consumed, accumulation of nitrite occurs in the rumen. Nitrite is then absorbed into the bloodstream and will convert haemoglobin to methaemoglobin, which is unable to transport oxygen. Blood that contains methaemoglobin has a chocolate-brown colour. Death of livestock is due to lack of oxygen. Occurrence of nitrate poisoning is difficult to predict because nitrate levels change rapidly in plants and the toxicity of nitrate varies greatly among livestock due to prior exposure, age, health status, and diets. Nitrates and nitrites are water soluble and are leached or washed away by moving water–either by surface run-off or ground water percolation. Nitrates are more concentrated below or near the area of waste accumulation. Excess nitrates are often found in ground water especially shallow dug wells have higher nitrate levels when compared to deeper drilled wells. Before using a well that has not been in use for some time it is good practice to test the water for high nitrate levels.

In January 2019 there were reports of mass death of 115 camels, 8 cattle and several sheep and goats in El hadi, North Horr Sub-county. A herd of 140 camels from Yaa Gaara, a community that recently migrated from Forole watered at Tula well which had been abandoned for some time. Two hours after drinking the water most camels died. According to the elders, thee camels on slaughter remained red and had erosions of the rumen pili. The lungs and heart were black and there was no frank blood in the carcass. There was no bloating or decomposition even after 5 days. The laboratory reports from indicated that the water from the well has a high levels of nitrate of between 230 and 780 mg/L above the normal 10 mg/L.



Mass death of camels in 2019 after drinking water from a well in Elhadi village, Marsabit county

#### Treatment of nitrate and nitrite poisoning

Slow intravenous (IV) injection of 1%–2% methylene blue in distilled water or isotonic (0.9% NaCl) saline solution. Administered at 4–15 mg/kg or more, depending on severity of exposure. Lower dosages may be repeated in 20–30 minutes if the initial response is not satisfactory. Lower dosages of methylene blue can be used in all species; only ruminants can safely tolerate higher dosages. If additional exposure or absorption occurs during therapy, retreatment with methylene blue every 6–8 hours should be done. Affected animals should be handled to minimize stress, which can exacerbate effects of hypoxia. Animals should be removed from the source of poisoning.

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# marsabit county VETERINARY HANDBOOK







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# FOR PRIORITY CAMEL, CATTLE, Sheep, Goat Donkey & Chicken Diseases

Common Diagnosis, Treatment and Control Guidelines for Animal Health Service Providers