Afrivet
Animal health is in our DNA
TRAINING SERVICES

1.4

LIVESTOCK HANDLER TRAINING MANUALS

MODULE 1: EARLY DISEASE IDENTIFICATION

The most common disease processes

Understanding the disease development process for the most common diseases.

English

ANIMAL HEALTH IS IN OUR DNA

LIVESTOCK HANDLER TRAINING MANUALS

MODULE 1: EARLY DISEASE IDENTIFICATION

The most common disease processes

Understanding the disease development process for the most common diseases.

CONTENTS

Developed by Dr Danie Odendaal

Disclaimer

Although care has been taken to be as accurate as possible, neither the author nor the publisher makes any express or implied representation as to the accuracy of the information contained in this book and cannot be held legally responsible or accept liability for any errors or omissions .

Afrivet Animal health is in our DNA

RPO

V-NET

UNIVERSITEIT VAN PRETORIA
UNIVERSITY OF PRETORIA
YUNIBESITHI YA PRETORIA
Faculty of Veterinary Science

AECF Funding Innovation for Business in Africa

INTRODUCTION

This manual forms part of Afrivet's series on primary animal health care (PAHC), and has been developed to help the veterinarian, animal health technician, livestock owner and livestock handler to understand the methodology used when implementing PAHC and production management.

These manuals are ideally suited as practical training aids for training livestock handlers in the principles of planned production management, disease prevention and early disease identification.

Information contained in this manual is a summary of the material used by Afrivet Training Services for formal training of animal health technicians, extension officers, livestock farmers and livestock handlers.

Developed by Dr Danie Odendaal

The disease process

Disease development happens over a period of time. If this is understood it creates an understanding of the window of opportunity in which the different diseases can be prevented or treated before they cause major losses.

The difficult concept is that the disease transmission and development process differs for each disease condition and therefore the time over which each disease develops also differs.

In order to understand the urgency of the disease management actions to be taken, the person who takes care of the animals on a daily basis must understand the timeline over which diseases develop.

The livestock handler needs to understand the disease processes of the 10 most important diseases on his/her farm in order to manage them effectively.

The disease developm of important diseases is ill understand the disease prev

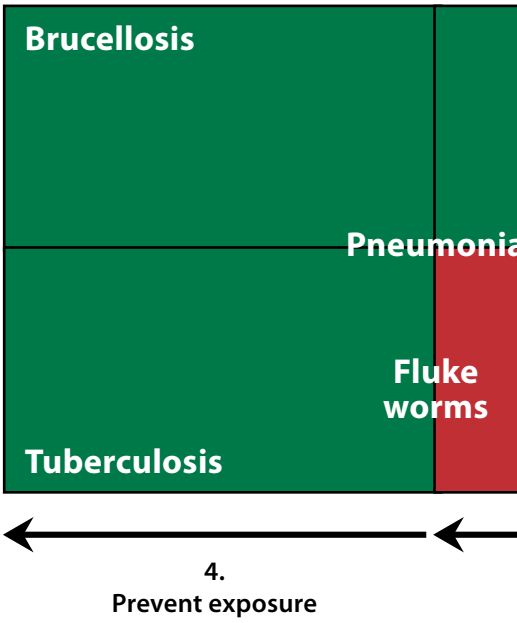
Disease prevention options:

1. Increase general resistance
2. Increase specific resistance (vaccination)
3. Decrease exposure
4. Prevent exposure

Every disease needs to be managed differe development process and the limited options ava

Prevention

Some diseases must be prevented because they cannot be treated effectively.

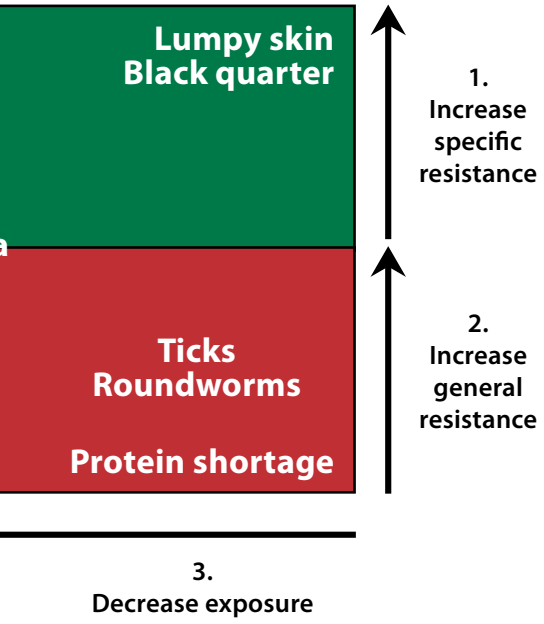


ent process (timeline)
ustrated in order to better
ention or treatment options.

Disease treatment options:

1. Treatments against external parasites
2. Treatments against internal parasites
3. Antibiotic treatment against bacterial diseases
4. Combination and other treatments

ntly owing to the differences in the disease
ailable for prevention or early treatment thereof.

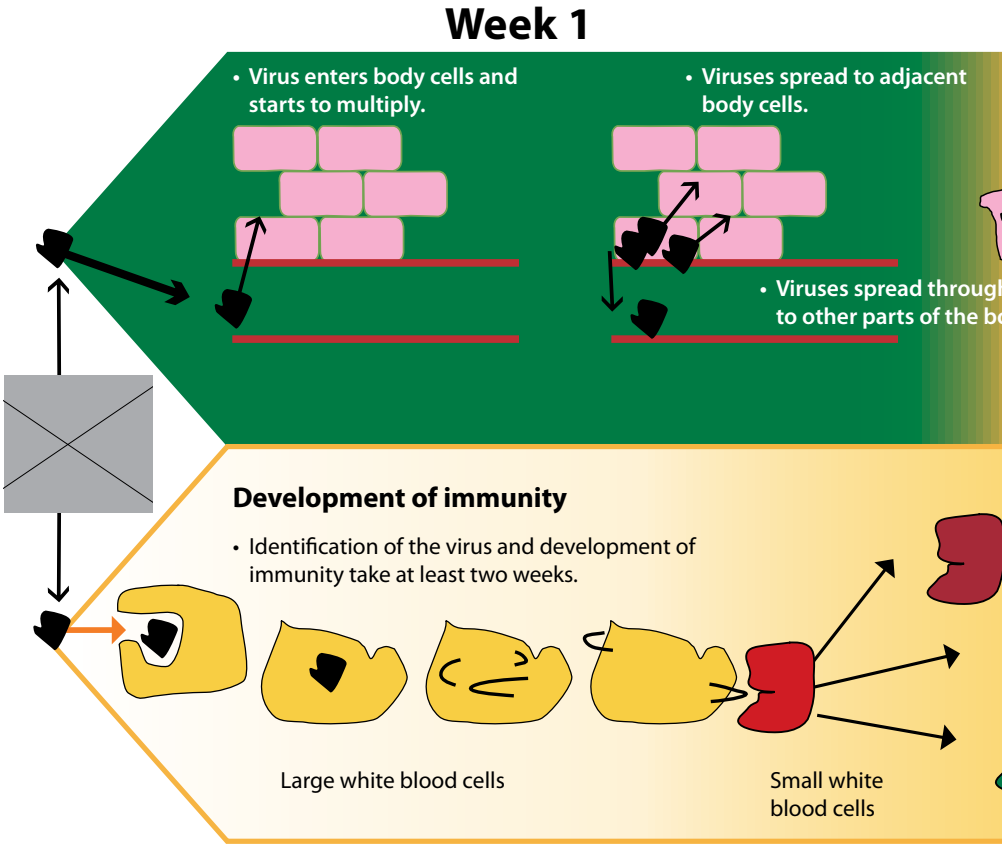


Treatment

Some disease conditions must be identified and treated on an on-going basis or with the first signs of disease because they cannot be prevented effectively.

Lumpy skin disease in cattle – th

Graphical illustration of the disease development process for lumpy skin until the first signs of disease are visible in the animal.



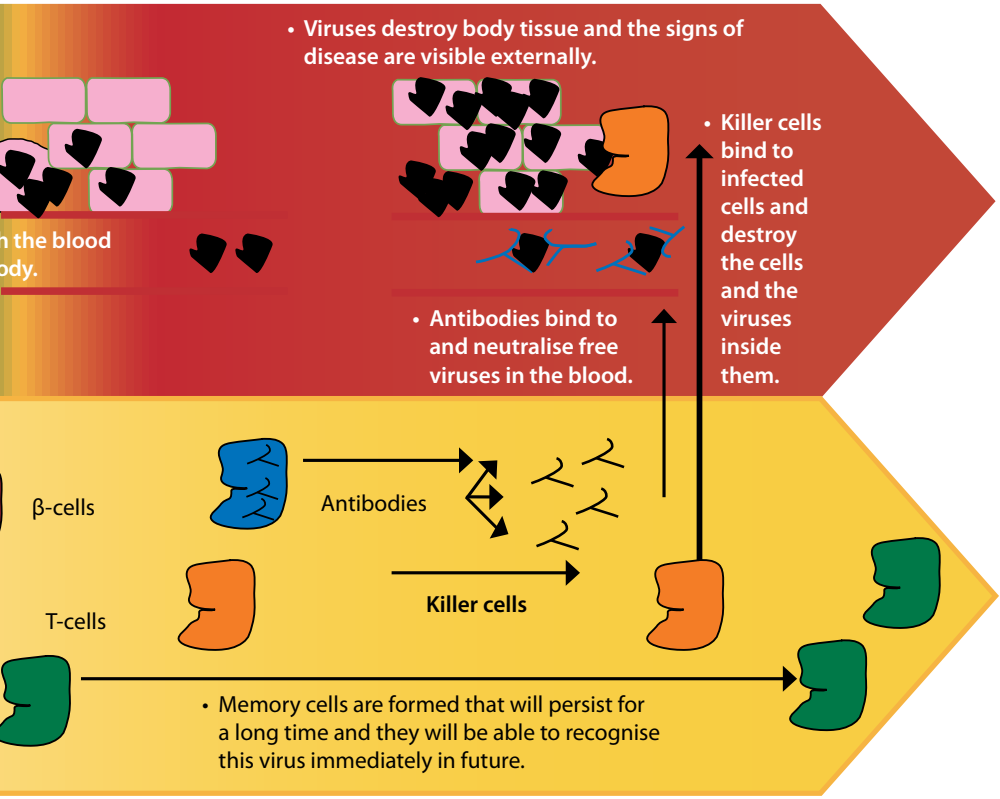
After infection, the body develops immunity against the virus disease, but this takes **at least two weeks.**

he disease development process

n disease from transmission of the virus (infection) by the biting insect,

Remember, that the development process and the type of body cells infected differ for each viral disease.

Week 2



The limitation is that the development of protective immunity takes longer than disease development during which the disease causes damage to the body.

Pneumonia – the disea

Every type of disease has a different development process, which can take f

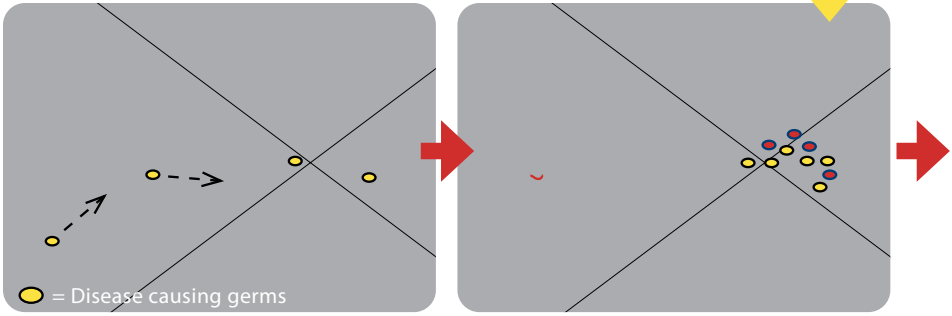
Start of infection

No signs of disease can be observed and the animal will look healthy and is still eating.

One to two days later

The first signs of disease are coughing and a watery discharge from the nose. The ears are now hanging and the animal starts to eat less.

First signs of disease



Development of pneumonia

24-hour early

Germs (bacteria and viruses) from the air are inhaled. Normally, the body will trap these germs in the slime covering the smooth lining of the nose and windpipe. The slime will then be coughed out to get rid of the germs. Yet because of cold, wind or dust, the body sometimes cannot get rid of the germs and they begin to multiply in the lungs.

Because of the favourable growth environment in the lungs, the germs multiply fast and is now 10 times more. The multiplying germs start to damage the lungs. The animal's own defence system now starts to react and this defence reaction causes a fever.

This part of the disease process is not easily visible.

se development process

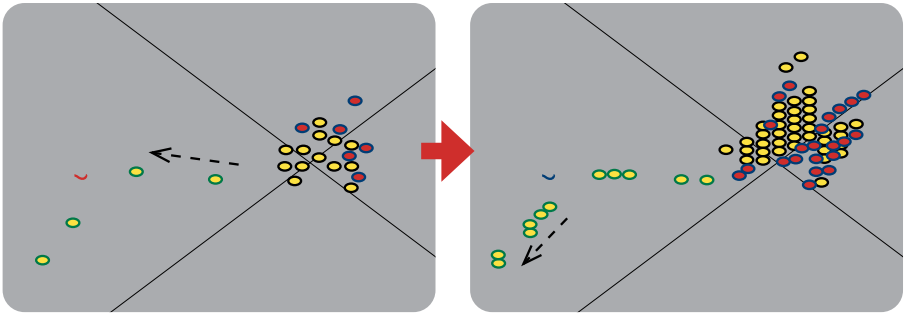
rom a few seconds to a few months.

Three to four days later

The nasal discharge becomes yellow and thick. The animal stands with a lowered head and falls behind the herd when driven.

Five to six days later

The animal is struggling to breathe through its nose, and straining the belly and ribcage to breathe out. It lies down and is too weak to get up. The animal dies.



window for treatment

Bacterial infection of the lungs

The number of germs is now 1 000 times more. The germs and the body's own defence reaction cause damage to large parts of the lungs – normally a very soft and delicate organ. The transfer of oxygen to the blood is severely reduced. The animal has a fever and a temperature above 40°C.

The millions of germs and puss produced by the body's defence reaction have permanently damaged and destroyed most of the lungs. The lungs cannot function anymore and the animal dies, owing to a lack of oxygen. When the dead animal's organs are examined, the lungs are very hard, dark red and sometimes filled with yellow pus.

This part of the disease process is now easily visible.

Black quarter in cattle – the

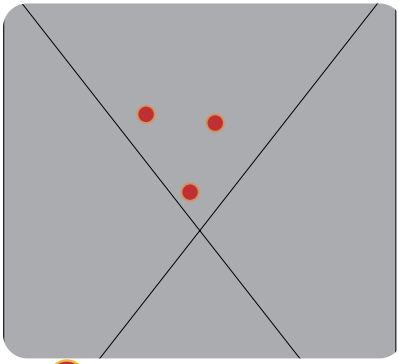
Low-grade infection

No signs of disease can be observed and the animal will look healthy and eat and produce normally.

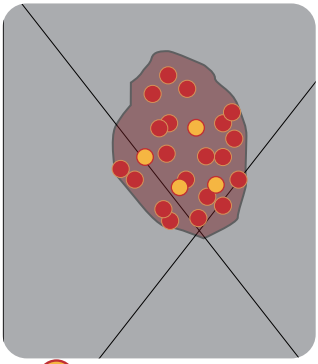
One to two days after bruising

There are no clearly visible first signs of disease, other than lameness and swelling of one leg if the cattle are continuously observed.

First signs of disease



= Bacteria



= Poison

Development of black quarter

Normally there is no chance for treatment

The bacteria that cause black quarter enter the body through small wounds. These **germs will migrate to the large muscles and will remain (resting)** there without multiplying or causing damage. These germs will start to multiply only when these muscles are bruised.

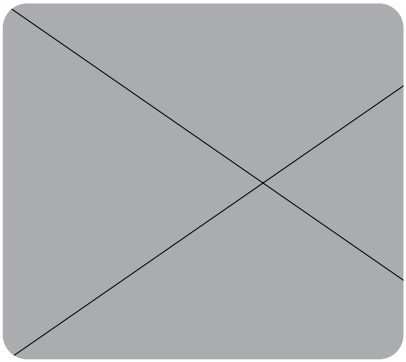
Muscles are bruised, for example when animals are beaten or handled roughly in a badly constructed crush pen. This causes internal **bleeding and the accumulation of dead blood** forms the right condition for the **germs to start multiplying fast** and produce a **dangerous poison (toxin)** in the muscles.

This part of the disease process is not easily visible.

disease development process

Three to four days after bruising

Sudden death of animals – mostly calves or young cattle but adult cattle can also die.



Examination of dead cattle

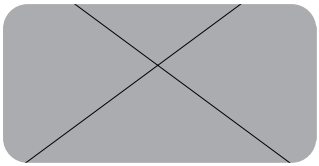
When a veterinarian cuts the dead cattle open he/she will look for typical signs of the disease in the muscles, under the skin and in other organs.

Photo's: University of Pretoria

Poison that forms in the muscles will kill cattle

This poison is now taken up into the bloodstream and circulated throughout the body. This poison causes immediate and severe damage to all the vital organs in the body including the kidneys, heart, lungs and brain.

The veterinarian will take some samples to confirm the cause of the disease (diagnosis).



This part of the disease process is now easily visible.

Blue ticks on cattle – the d

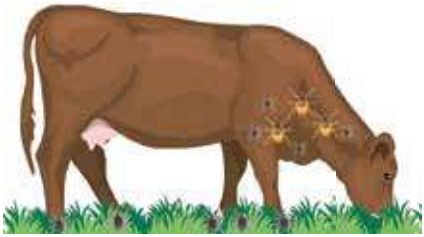
Week 1

Start of infection. The very small ticks (larvae) are not easy to see because they are as big as the head of a pin. The animal's coat (hair) still looks smooth.

= Real size of larva

Week 2

The small ticks (nymphs) are still not easy to see because they are now as big as the head of a match. During closer inspection nymphs can be seen in areas where the hair is short, like on the neck fold (dewlap) and backside of the upper back legs.



= Real size of nymph

Length of development = three weeks (21 days)

The tick larvae hatch from the eggs on the ground when the weather is hot enough. These larvae climb onto grass leaves and seed heads and wait for cattle to brush past when grazing. They then climb onto the cattle and attach by making small holes in the skin with their mouths. Then they start sucking blood from the small blood vessels in the skin and grow.

After one week, the larvae moult (change their skin) to become nymphs, which will attach again, suck blood and grow further. It must be taken into account that new infections with larvae will still take place continuously, adding to the total number of ticks on the cattle.

This part of the disease process is not easily visible.

isease development process

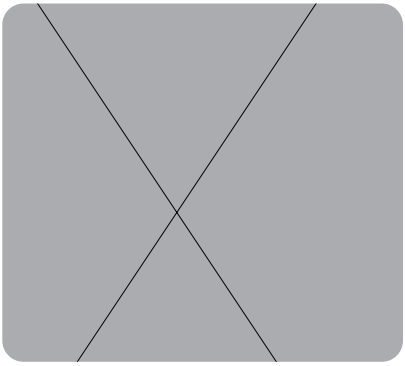
Week 3

Flat adult ticks are still not clearly visible. The first adult engorged blue tick females will become visible three weeks after having climbed on the cattle as small ticks (larvae). Hereafter large numbers of newly engorged blue ticks will be seen on a daily basis until animals are treated.

= Real size of an adult engorged female

Inspection after treatment with a dipping compound

If the treatment was successful, no engorged female blue ticks should be visible one week after treatment.



until engorged female blue ticks are visible.

After the second week, the nymphs will now moult to become adult ticks, which will attach and feed. At this stage the females are still flat and they will find a male to mate with. After mating at the end of the third week, the female ticks will quickly suck blood and become engorged overnight. The next day they will detach, fall off and produce up to 2 500 eggs.

Effective treatment

A dipping compound must kill all the ticks from very small to adult, present on the cattle at the time of treatment, and must further protect against new infections for up to one week after treatment.

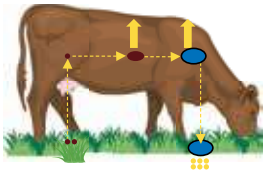
This part of the disease process is easily visible by weekly inspection of the cattle early in the morning.

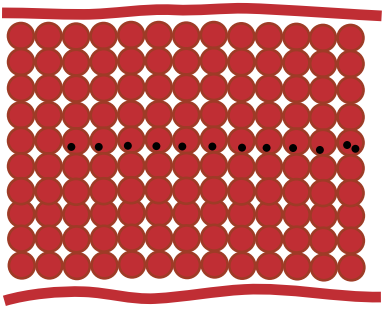
African redwater – the dise

An example of the disease development process of a tick-borne blood paras

Start of infection

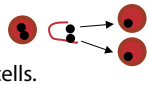
The blood parasite (*Babesia bigemina*) is transmitted by the nymph and adult stages of the blue tick when it sucks blood from the animal.





The blood parasite will enter the small blood vessels in the skin with the spit (saliva) of the tick when it sucks blood. It will then enter a red blood cell where it will grow and divide.

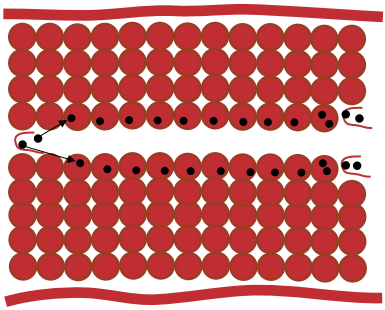
In this process, it will destroy (burst) the red blood cell and the two parasites formed will now enter two new red blood cells.



Initially, only very few red blood cells will be infected (fewer than 1 in 100).

Week 1 – 2

For the next two weeks, the animals will show no sign of disease and will behave, eat and walk normally.



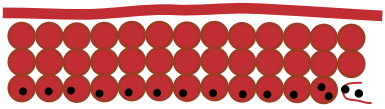
Over these two weeks, the blood parasites will multiply and infect more and more red blood cells. Infected red blood cells burst, leading to blood loss (anaemia). The liver or kidneys must now disperse of the red content of the burst red blood. The animal's own immune system now starts to react against the infection, giving rise to a fever.

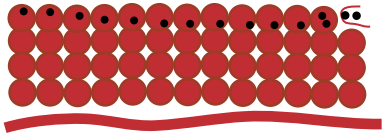
ease development process

site causing destruction of red blood cells leading to blood loss.

>2 Weeks

Infected cattle suddenly develop general signs of disease: they stop eating and chewing the cud, heads and ears hang and they become reluctant to move. More specific signs such as difficult or faster breathing and **red-brown urine** now become apparent.



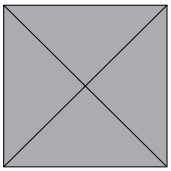


The animal's fever will rise to over 40°C at least two days before visible signs of disease appear.

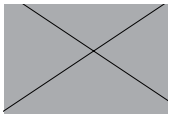
The animal copes with the blood loss up to a certain point when there are just too few red blood cells left for the animal to carry enough oxygen to the rest of the body cells. The animal will show sudden weakness and when examined, the inner eyelid will be white. The animal normally dies within 24 hours after advanced signs of disease.

Diagnosis of infection in the live animals

The veterinarian examines the animal by taking the temperature, listening to and determining the heart and respiration rate, and looking at the inside eyelid. He/she will make a blood smear for examination under the microscope to identify the parasites in the red blood cells.



Examination of dead cattle



The veterinarian will look for many signs including signs of blood loss, liver and kidney damage and the colour of the urine in the bladder. A blood smear will also be examined to confirm a final diagnosis.

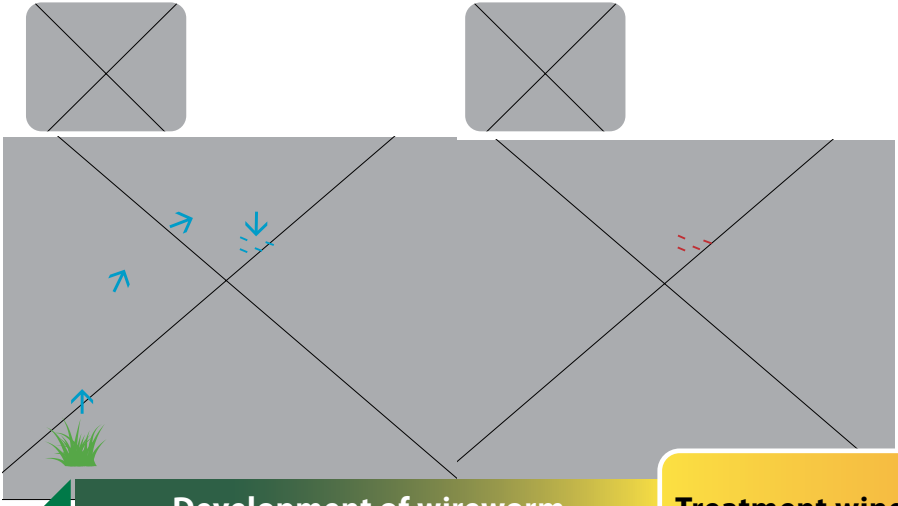
Wireworm in sheep – the di

Week 1

Start of infection. No signs of disease can be observed and the animal looks healthy and eats and produces normally.

Week 2

Usually, no signs of disease can be observed, as wireworm doesn't usually affect the desire for food like some other worms.



Development of wireworm – three weeks (21 days)

Treatment window for signs of blood loss –

Wireworm eggs in the dung (on the pasture) hatch when the average daily temperature is above 15°C and the rainfall higher than 50 mm. The small worms then feed and develop in the dung until it is ready to infect sheep. The small worms creep out of the dung onto the wet pasture and **are then taken in by the grazing sheep.**

After infection, the small worms attach to the wall of the fourth stomach called the abomasum and they start growing. **During this growth phase they suck a lot of blood on a continuous basis**, which produces the nutrition needed for their fast growth. Every drop of blood lost in this way will decrease the animal's production.

This part of the disease process is not easily visible.

sease development process

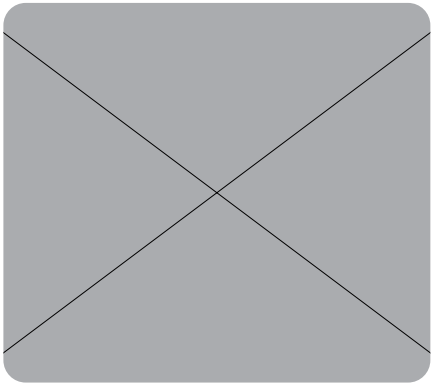
First signs of disease

Week 3

Individual sheep can fall behind when herded, or develop bottle jaw, which is a sign of slow blood loss. If the infection is advanced, the animal suddenly shows weakness and dies shortly afterwards, without showing other signs of disease.

Examination of the dead sheep

When a veterinarian cuts open the dead sheep, he/she will look for signs of blood loss and masses of adult worms in the fourth stomach.



sheep that show pale inner eyelid

Sheep can die because of blood loss

The adult worms reach maturity from three weeks after they have infected the animal. They will now mate and produce a large number of eggs on a continuous basis. The eggs mix with the dung, which then becomes the dung pellets excreted by the sheep onto the grazing. If it is warm and wet, the eggs will hatch and start the next cycle of producing infective small worms on the grazing.

Fresh dung pellets can also be collected from sheep and sent to the veterinarian for examination and determination of the number of worm eggs. This gives an indication of the number of worms in the sheep and is also used to determine if treatment is necessary.

This part of the disease process can only be identified by weekly inspection of the inside lining of the eyelid of animals.

Liver fluke in animals – the d

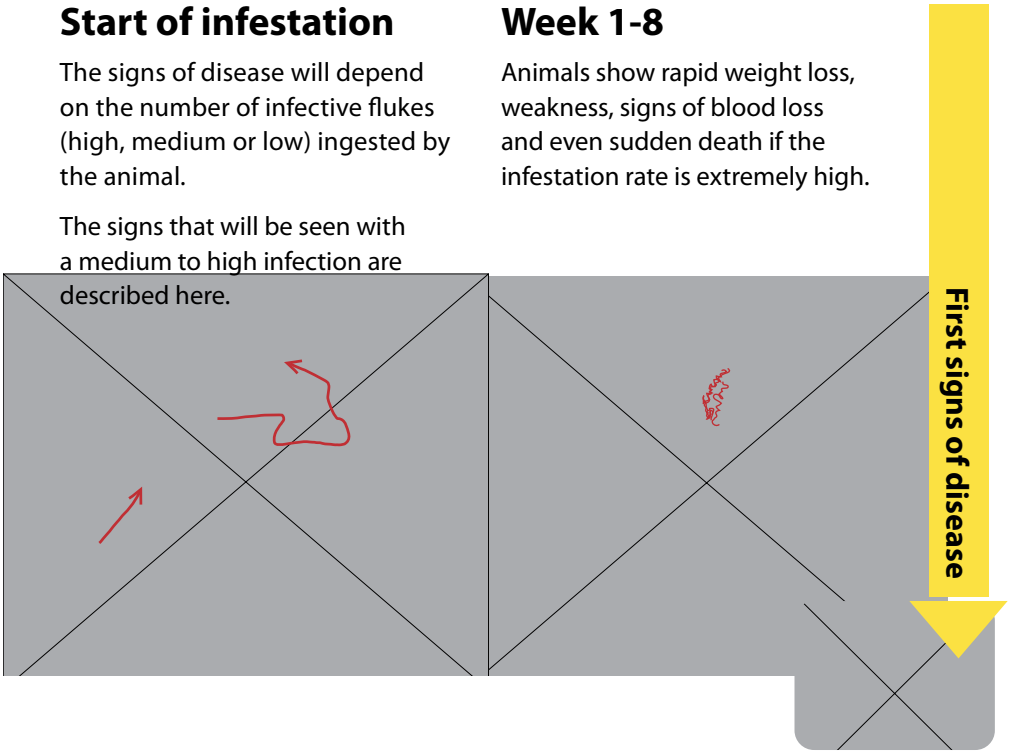
Start of infestation

The signs of disease will depend on the number of infective flukes (high, medium or low) ingested by the animal.

The signs that will be seen with a medium to high infection are described here.

Week 1-8

Animals show rapid weight loss, weakness, signs of blood loss and even sudden death if the infestation rate is extremely high.



Development of liver fluke inside

When the animals graze in wet areas they take in the immature flukes which are attached to plants.

Inside the animal, these immature flukes move through the wall of the small intestine, migrate to the liver and penetrate the liver.

The immature flukes will then start to eat liver tissue, forming small tunnels in the process.

Immature flukes feed on the liver for the next six to eight weeks while growing, causing severe damage to the liver.

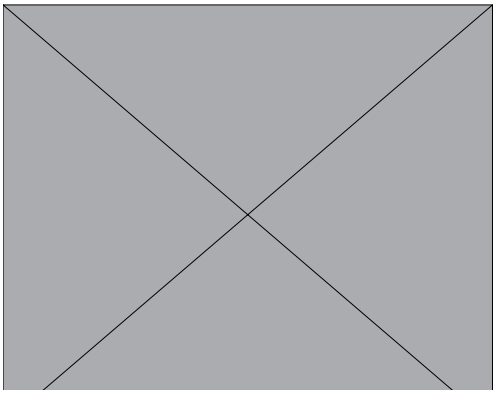
Liver damage depends on the number of immature flukes feeding on the liver tissue.

By week eight, they become adult flukes that enter the small bile ducts (tubes) and migrate to the larger bile ducts.

isease development process

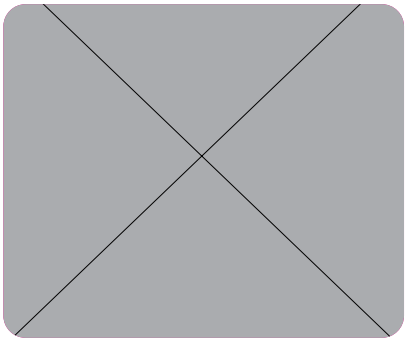
>12 Weeks

Affected sheep can fall behind when herded or develop bottle jaw – a sign of slow blood and protein loss. Continued weight loss especially during the period when the grazing is poor.



Examination of dead sheep

When a veterinarian cuts open the dead sheep he/she will look for signs of liver damage (thickened gall tubes) and flukes in the gall tubes.



cattle, sheep and goats

The adult flukes attach to the wall of the bile ducts and feed by drinking blood. After month, each starts to produce eggs (20 000 per day) which go with the bile into the intestine and out with the dung.

The thin walls of the bile ducts now become thickened and white, and whole liver can become hard (fibrotic), owing to the body's reaction to this infestation.

Diagnosis of infestation in the live animals

To confirm infestation in the live animal, dung must be collected and sent to the veterinarian for tests to determine if there is a liver fluke infestation. The newest tests can identify the infestation early (from four weeks after infestation), which will be at the time when the first signs of disease are observed in the case of a very severe infestation.

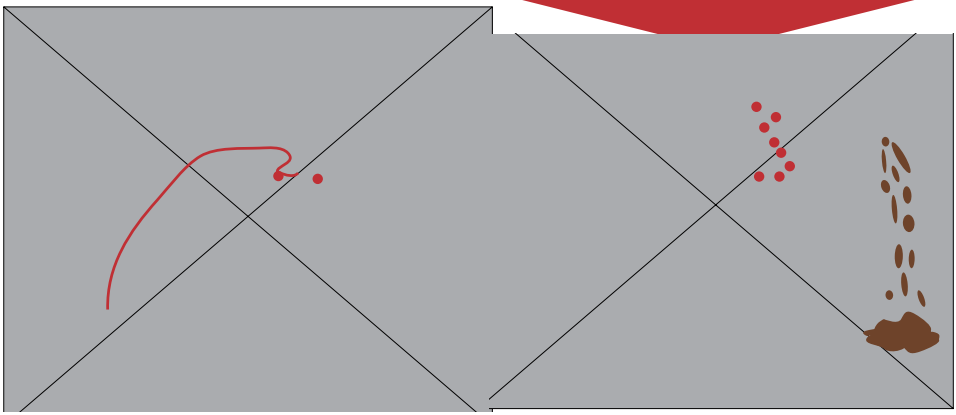
Stomach (conical) fluke in animals -

Start of infestation

No signs of disease can be observed and the animal will look healthy and will eat and produce normally.

Week 1-8

The first signs of disease are that the animals stop eating, develop severe foul-smelling and watery diarrhoea, and condition rapidly. Many animals can die when there is a severe infestation.



Development of stomach flukes inside

When the animals graze in wet areas they ingest the immature flukes attached to plants.

Inside the animal, these immature flukes go to the small intestine where they lose the protective capsule that was protecting them in the environment.

They now attach to the wall of the small intestine with very strong suckers to drink blood.

The immature flukes suck a piece of the lining of the intestinal wall into their body opening, which causes damage o, and holes in, the lining of the intestine. This causes blood components to leak into the intestine.

The damage depends on the number of immature flukes, which will become adults after 6–8 weeks.

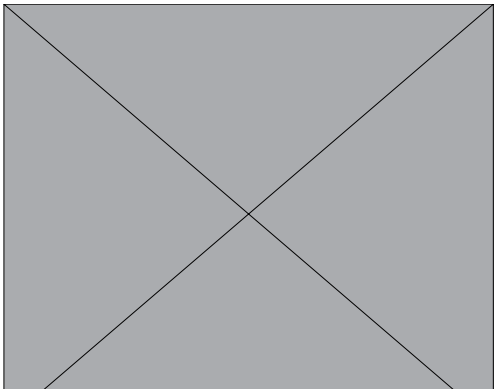
– the disease development process

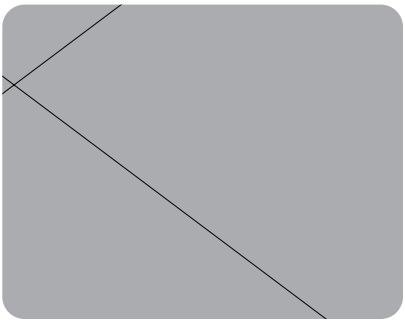
>12 Weeks

The adult stomach flukes cause no harm to the animal and no signs of disease are apparent.

Examination of dead sheep

When a veterinarian cuts the dead sheep open he/she will look for the presence of immature flukes in the small intestine and signs of damage of the lining of the small intestine.





cattle, sheep and goats

The adult flukes migrate to the big stomach (rumen) where they will attach without causing damage to the animal.

After four weeks, they will start to produce eggs that will pass out with the dung onto the grazing.

Under the right conditions, these eggs will hatch to infest the freshwater snails again.

Diagnosis of infestation in the live animals

To confirm infestation with immature stomach flukes in the live animal, at least a dessertspoon full of the watery dung must be collected when the first signs of diarrhoea are observed. This must be sent to a veterinarian for microscopic tests to determine if there is an immature stomach fluke infestation.

The development of dry gallsic

The amount of protein (protein percentage) in the grass is the first limiting fac
longer during the year, a disease condition called dry gallsickness can develop

Starts as a result of low-quality grazing

At the start of the dry season, the crude protein percentage in the grass will start to fall below 10%.

BCS – 2.5

The eye muscle is half full and the bone ends feel well rounded. 2 to 5 mm of fat can be felt under the skin over the pin bone.

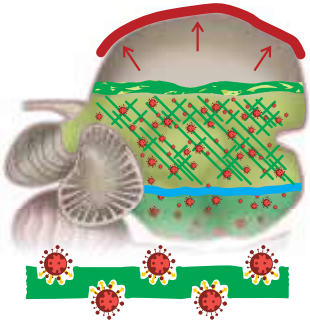
1 to 3 months later

The crude protein percentage in the grass is now 7% and below.

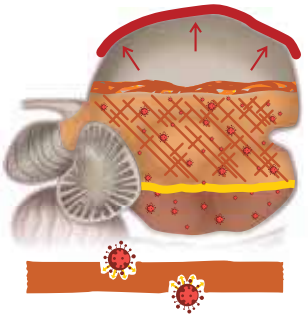
BCS - 2

Eye muscle is very indented but the bone ends just feel rounded. 1 mm of fat can be felt under the skin over the pin bone.

Development of dry gallsickness caused



Protein is the first limiting nutrient in natural grazing. The protein needs of the organisms are not provided by the grazing. The organisms in the rumen become less because they do not have enough protein to multiply at a fast rate. This directly reduces the food supply of the cow because there are less organisms to digest. Owing to the fact that cattle are pregnant but still producing milk for the calf, they will start to lose weight.



When the protein in the grass falls to 7% and below the number of organisms that must digest the grass becomes even less. These organisms need enough protein otherwise they can't multiply. Now the grass will be digested even slower, and the cattle will eat much less (1.5% of body weight) because undigested grass remains for a longer time in the rumen. Even if the calves are weaned, the cows will still lose condition because of the very slow rate of digestion.

ckness due to protein shortage

ctor in dry grass. Because there is only dry grass available for six months or p if the nutritional shortage is not supplemented by providing a protein lick.

4 to 5 months later

The crude protein percentage in the grass is now 4% and below.

BCS - 1

No eye muscle can be felt and the bone ends are very sharp. No fat can be felt under the skin and the skin is stuck to the pin bone.

by a protein shortage

When it is completely dry and very cold, the grass becomes very hard and the protein can fall to 4% and even lower. By now, the organisms in the rumen stop multiplying and die. The grass in the rumen can't be digested any more.

Cattle will rapidly lose weight when this starts to happen. They will get to the point that they are too weak to walk and will lie down. Once cattle lie down they will die in a few days and in most cases they can't be rescued, even if they are provided with good feed at this stage.

Examination of dead cattle

When a veterinarian cuts open the dead animal, he/she will find that the different stomachs of the cow contains very dry grass and the gallbladder is enlarged. In cattle that haven't eaten for a days, the gallbladder will be bigger so it is not a sign of a specific disease.

The animal will also not have any fat reserves specifically around the kidney, which is a sign of starvation.

Diagnosis of protein shortage in the live animals.

There is no specific test to make a diagnosis of a long-term protein shortage. The veterinarian will base their findings on the time of year, available grazing, pregnancy status and condition of the cattle.

This condition will occur in most areas of South Africa where a protein supplement is not provided during the dry season.

The digestibility of dry grass becomes too low to maintain good production and the goal of producing one calf every 12 months cannot be obtained without a protein supplement.

TREATMENT OF BACTERIAL INFECTIONS USING ANTIBIOTICS

ULTRATET 200 LA

Reg No. G2857 (Act 36/1947)
NS0 V01/18.1.2/307

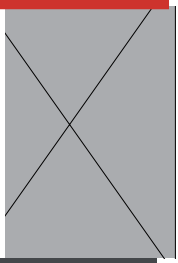
Oxytetracycline 230 mg/ml

Treatment of tick-borne gallsickness (*anaplasmosis*), heartwater, pneumonia, footrot, joint-ill and navel-ill in cattle, sheep, goats and pigs.

Withdrawal period:
Meat: 28 Days
Milk: 5 Days

Administration: IM or SC

Registration holder: ECO Animal Health Southern-Africa (Pty) Ltd Co.
Reg. No.1992/000835/07



Packaging available
100 ml, 500 ml

1 ml / 10 kg | 1 ml / 10 kg | 1 ml / 10 kg

TREATMENT OF EXTERNAL PARASITES WITH PLUNGE DIP/SPRAY

ERADITICK 250

Reg No. G4047 (Act 36/1947)

Amitraz 25 % m/v

Controls ticks and kills lice and mange mites on cattle. Controls ticks, controls sheep scab mites and kill itch mites and goat mange mites on sheep and goats. Has a detaching effect on ticks. OXPECKER COMPATIBLE.

Spray: 1 L / 1 000 L water
Boost with 100 ml / 200 head after 400 head sprayed
Plunge (total replenishment): 1 L / 5 000 L water
Plunge (lime stabilised): 1 L / 1 000 L water

Plunge (fresh fill):
1 L / 750 L water

Plunge (fresh fill):
1 L / 750 L water

Withdrawal period:
Meat: 7 Days
Milk: None

Packaging available
500 ml, 1 L, 5 L, 10 L

Registration holder: ECO Animal Health Southern-Africa (Pty) Ltd Co. Reg. No.1992/000835/07

TREATMENT OF ROUNDWORMS IN SHEEP AND CATTLE

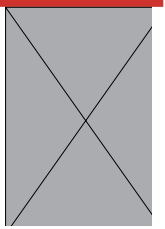
ECOMECTIN 1 %

RSA Reg. No. G2275 (Act 36/1947)
Namibia S0 V02/18.1.2/1145

Ivermectin 1 % m/v

Treatment of roundworms in sheep and cattle and *Parafilaria bovicola* (false bruising) and eye worm (*Thelazia*) in cattle. Kills sucking and biting lice, mange mites, cattle screw-worms and controls blue ticks (*Boophilus* spp) and sand tampans on cattle. Kills sheep scab mites, controls Australian itch mites and nasal worm (all stages) in sheep. Kills mange mites (*Sarcoptes scabiei* var *suis*) on pigs.

Withdrawal period:
Meat (Cattle & Sheep): 21 Days



Packaging available
20 ml, 50 ml, 500 ml, 1 L

1 ml / 50 kg | 1 ml / 50 kg | 1 ml / 33 kg

Registration Holder: ECO Animal Health Southern-Africa (Pty) Ltd Co. Reg.