Helminth infections of ruminants

Author: Prof. Joop Boomker
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INFECTIONS OF THE LUNGS AND TRACHEA

Dictyocaulus filaria is the lungworm that occurs in the trachea and bronchi of sheep and goats world-wide. The life cycle is direct and the developmental period is 28-30 days. Most eggs hatch in the bronchi and are coughed up and swallowed. Those not hatched do so in the intestine. L1 is present in the faeces and will only migrate from the dung pad when sufficient moisture is present. Larvae do not feed but live off the granular material present in the intestinal cells. They can easily overwinter for periods up to 100 days, provided there is enough moisture. Infection takes place per os and larvae move to the mesenteric lymph nodes. The worms migrate through the lymphatic ducts to the thoracic duct, from there to the heart and the lungs and will eventually burst through the lung alveoli to reach the bronchioli and bronchi, where they mature. Males are present from day 6 and females from day 8 after infection.

The pathogenesis can be divided into 4 phases:

a) The **penetration phase**, during which the larvae migrate via the lymphatics and lymph nodes to the lungs and lesions are not yet apparent.

b) The **pre-patent phase**, when the larvae arrive in the lungs. They cause alveolitis, followed by bronchiolitis and finally bronchitis as they become mature and move to the bronchi. Cellular infiltrates (neutrophils, eosinophils and macrophages) temporarily plug the bronchioli, causing the collapse of groups of alveoli. This is responsible for the first clinical signs.

c) The **patent phase** is associated with 2 main lesions, namely a parasitic bronchitis, characterized by the presence of many adult worms embedded in white frothy mucus. Secondly, a parasitic pneumonia occurs, characterized by collapsed areas around infected bronchi. The pneumonia is the result of aspirated eggs and L1 which act as foreign bodies and provoke pronounced polymorph, macrophage and multinucleated giant cell infiltrations. Varying degrees of oedema and emphysema may also be seen.

d) The **post patent phase** is normally the recovery phase after the adult lungworms have been expelled. The lung tissue organizes and clinical signs abate. In a small percentage of animals, however, a flare-up of clinical signs may occur during this time. This is due to one of 2 causes, epithelialization of the lungs that hinders gaseous exchange and may result in interstitial emphysema and lung oedema, and secondary bacterial infection, which results in an acute interstitial pneumonia.

Within 2 - 3 weeks after infection the animals’ breathing becomes shallow and the head raised; a violent cough, and a profuse discharge of mucus from the nostrils is also present. Affected animals lag behind the rest of the flock. Emaciation and prostration may set in before death.

There are small foci of pneumonia, resulting from the 5th stage worms breaking through the alveoli. Frothy fluid is present in the alveoli and terminal bronchioles and there may be oedema and emphysema of the interlobular septa. Most of the bronchioles contain plugs of exudate. As the worms mature and move to the bronchi these plugs of exudate resolve. The mature worms in the bronchi are
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easy to see. They are surrounded by a frothy bronchial exudate and they cause atelectasis and emphysema secondary to the bronchitis, and which is provoked by the eggs and larvae.

The lung lesions appear as fairly large wedge-shaped areas, dark red or grey in colour, usually situated at the posterior border of the diaphragmatic lobes and are slightly sunken below the surface of the surrounding tissue. In cases where bacterial infections took place, the lesions show all the changes associated with a purulent pneumonia. There is no pleuritis.

Sheep become resistant after the initial infection and larvae of subsequent infections become arrested and are destroyed in the lymph nodes. They do not reach the lungs. Animals are equally resistant if they are treated at different stages of the development of the worms. This is one of the few nematodes against which a good vaccine has been developed.

The larvae are extremely sensitive to heat and desiccation but are resistant to cold. In southern Africa these worms are therefore usually seen in isolated areas with generally low temperatures and adequate moisture. Irrigated pastures may provide a potential focus of infection in areas free of *D. filaria*.

**Dictyocaulus viviparus** is the lungworm of cattle. It is present worldwide and is a problem in especially Europe and North America, where cattle are housed in barns during winter. The life cycle is direct and the developmental period is 24 days or more. Most eggs hatch in the bronchi and are coughed up and swallowed. Those not hatched do so in the intestine. L1 is present in the faeces. The infective larvae are relatively inactive and there is little movement / migration from faecal pads onto herbage. During heavy rainfall or irrigation, larvae will move onto herbage but only for a limited distance.

The pathogenesis of the disease in cattle is the same as that for sheep and goats. Few other changes are noted, but in severe cases calves may die during the 3rd week after infestation. This is accompanied by moist rales, and rapid respiration (up to 200/min). Depending on the size of the infection, coughing may persist for weeks or months.

The epidemiology is largely unknown. The parasite is present in isolated foci in the mist belt of the Drakensberg in Natal and Transvaal, and is rife on irrigated pastures. Optimum conditions for larval survival are cool moist conditions and development can take place at temperatures as low as 5°C.

**Muellerius capillaris** is found in the lungs of sheep and goats, and snails and slugs are the intermediate hosts. They are common in the winter rainfall areas of the Cape and cause greyish nodules, 2-3 mm to 2 cm in diameter, to form in the parenchyma of the lung.
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*Dictyocaulus* in the trachea of a bovine (top left), and at the bifurcation of the trachea in a sheep. Note the large amount of foam (top right). Fairly large wedge-shaped areas, dark red in colour develop as a result of the aspiration of eggs and larvae (bottom left) and oedema and emphysema often occurs (bottom right)

Nodules caused by *Muellerius capillaris* in the lungs of a sheep
INFECTIONS OF THE GASTRO-INTESTINAL TRACT

Introduction

In South Africa it is generally accepted that the helminths in the gastro-intestinal tract of cattle do not to cause the same problems to the same extent as those of sheep and goats. Parasites are usually encountered in animals under 18 months of age, especially when large numbers of infective stages of the helminths are available on pastures and also when susceptible cattle are grazing these pastures. The infections that affect cattle mostly are Fasciola and Calicophoron, both in wet areas with standing water and where the snail intermediate hosts are present. In the Eastern and Western Cape Provinces, calves on irrigated pastures may contract Ostertagia sometimes with severe losses.

THE TREMATODES

Calicophoron microbothrium

This species was previously known as Paramphistomum microbothrium. It is the conical fluke that is found in the small intestines (immatures) and the rumen (mature flukes) of cattle, sheep, goats and most wild ruminants world-wide.

The life cycle is similar to that of Fasciola spp. and the only differences are the predilection site of the parasites and the intermediate hosts. Eggs are passed with the faeces and hatch in water 12-26 days later. Miracidia enter the young of the aquatic snail, Bulinus tropicus, at birth and up to the age of 3 weeks to form sporocysts. Older snails are not infected. Sporocysts give rise to rediae that are present 14 days after the snail has been infected and daughter rediae occur after 20-28 days. Cercariae are present after 30 days and start emerging from the snail by the 43rd day. Snails may remain infected and shed cercariae for up to 1 year. The cercariae encyst on vegetation to form the metacercariae (Figure 1 top left). They will die if desiccated or completely submerged, but remain viable for 2 months under cool moist conditions.

Once ingested, the metacercariae excyst in the first 3 metres of the small intestine and the young flukes attach to the mucosa (Fig 1, top right, and bottom left). After about 15-56 days in the small intestine, the young flukes start migrating to the rumen. The entire life cycle from the time that eggs are laid until the next generation eggs are laid, takes a minimum of 110 days in sheep and 132 days in cattle.

The immatures are the most pathogenic stage. They cause strangulation and necrosis of the intestinal villi. This leads to oedema, haemorrhage and ulceration of especially the duodenum (occasionally the jejunum) and cause discomfort, anorexia and loss of body mass. The absorption of food is impaired by the damaged intestine. Water intake remains high and the combination of high water intake, low food intake and degeneration of protein in the intestine produces a foul-smelling, fetid diarrhoea. Loss of plasma protein results in generalized oedema, which leads to haemoconcentration, retarded flow and hypoxia. The cause of death is lung oedema together with exhaustion and starvation. Anorexia,
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Fetid diarrhoea and mass loss, occasionally with bottle jaw may be present. In severe cases, death occurs 5-9 days after the onset of diarrhoea. Haemorrhage from the rectum due to straining may be seen.

Although heavy infections of adult worms may occur, they are relatively harmless and only occasionally petechiae or ecchymoses will be seen on the rumen wall.

*Bulinus tropicus* together with metacercariae (top left). Immature flukes in the small intestine. Note the oedema (top right). Immature flukes washed from the small intestine (bottom right) and mature flukes in the rumen (bottom right)

Sheep are not resistant and may be infected repeatedly. Cattle develop a strong immunity which is dependent on the presence of adult flukes. A single dose of 40 000 normal or irradiated metacercariae is sufficient to immunize cattle.

Outbreaks are common near permanent water supplies and are often associated with forced grazing in swampy or marshy areas. Outbreaks may occur from as early as April and may continue throughout the winter. Amphistomosis can occur throughout the year on irrigated pastures, since the moisture is adequate for the snails and the metacercariae. Please refer to ‘Infections of the liver’ where the ideal habitat for the snails is illustrated.

Clinical signs may give an indication. Eggs are absent from the faeces of acute cases, but large numbers of immature flukes in the small intestine are diagnostic. Collect diarrhoeic faeces and wash them over a 150 µm sieve, examine for the presence of immatures under a stereoscopic microscope. Variable numbers of eggs are present in the faeces of subacute and chronic cases. Previous outbreaks and a history of grazing of marshy areas and/or irrigated pastures may also be indicative.
THE NEMATODES

The term ‘Parasitic Gastro-enteritis’ is currently associated with the presence of large numbers of NEMATODES in the abomasum and/or intestine, rather than any other parasite. The worms in the abomasum are regarded as the primary pathogens, with those in the intestine playing a lesser but synergistic role. There are a number of nematodes that are important pathogens in sheep and goats, but the two nematode species in cattle that are of major importance are *Haemonchus placei* and *Ostertagia ostertagi*.

*Haemonchus contortus* in sheep

The worms are known as Barber’s pole worms or wireworms, or Public Enemy number One in the sheep industry, and occur in the abomasum. The adults are easily identified because of their specific location in the abomasum and their large size. Fresh female specimens are conspicuous, the white ovaries twisting spirally around the blood-filled intestine, giving them the barber's pole appearance and in having a large vulvar flap.

![Ventral aspect of the male bursa, showing the spicules with the barbs on different heights, the gubernaculum and the asymmetrical dorsal ray (top left). Female, with the large vulvar flap (top right). Masses of *Haemonchus* in the abomasum (bottom left) and oedema of the folds of the abomasum (bottom right).](image)

The life cycle is direct and females produce about 10 000 eggs per day. The L1 hatch on the pastures and infective stages can occur in as short a period as 5 days during warm, moist weather. However, development may be retarded for weeks or even months under cool conditions. Adults move freely across the abomasal mucosa and suck blood wherever they happen to be at the time. The developmental period is 18-21 days.
The pathogenesis of haemonchosis is essentially that of haemorrhagic anaemia due to the blood-sucking habits of the worms. Thus, each worm can remove up to 0.05 ml of blood per day by ingestion and seepage from the lesion.

**Peracute:** After infection with 20 000 to 35 000 L3, the resulting L4 cause petechiae while the 5th stages and adults cause frank haemorrhage and erosions at their attachment sites. Sheep can loose 1 000 – 1 750 ml blood per day. Death in apparently healthy sheep occurs suddenly as a result of severe haemorrhagic gastritis.

**Acute:** A burden of 2 000 – 20 000 adult worms cause a daily blood loss of 100 – 1 000 ml. Anaemia becomes apparent from about two weeks after infection and is accompanied by a progressive and dramatic fall in the PCV. Subsequently, the haematocrit stabilizes and intense compensatory erythropoiesis (visible as hyperplasia of bone marrow from white to red) at the expense of the iron reserves occurs. Together with the continual loss of iron and protein (albumin) into the gastrointestinal tract, the bone marrow eventually becomes exhausted and shortly before death, the PCV falls even further.

**Chronic:** About 100 to 2 000 adult worms can cause blood loss of about 5 – 100 ml per day. Chronic haemonchosis usually develops during winter when re-infection is negligible, but the pastures become deficient in nutrients, notably protein and iron. The continual blood loss depletes the iron reserves completely, with the result that a marked anaemia develops shortly before death.

The clinical signs associated with peracute infection are that animals die suddenly with few signs except anaemia and dark-brown to black faeces. It is rarely seen in South Africa.

In acute infection there is anaemia, bottle jaw, weight loss despite increased food intake, and dry dark brown to black faeces. Ewes stop producing milk and suckling lambs die of starvation. Lethargy and a break in the wool set in before death.

Chronic infection is often seen. Animals lose weight progressively over several months, but show neither severe anaemia nor submandibular oedema. Finally the animal becomes weaker and anorexia sets in. Anaemia is present shortly before death, as is submandibular oedema.

On autopsy of peracute cases few changes are seen, since death occurs too rapidly for lesions to become established. The carcass may be pale as result of anaemia, and there are many petechiae and small erosions in the abomasal mucosa. Coagulated blood may be present in the abomasal contents, the latter often being brown due to blood seepage. Masses of worms are usually seen squirming in the abomasal contents.

Acute cases are the ones most frequently encountered and the outstanding clinical feature is the extreme pallor of anaemia. The carcass is emaciated and generally oedematous. Compensatory erythropoiesis is seen as hyperplasia of the red bone marrow. The abomasal mucosa is hyperaemic and many petechiae and focal erosions are present. The submucosa is thickened and oedematous and few small ulcers may occur. The abomasal contents are scant and watery, slightly brown in colour and semi-digested blood clots are sometimes present. The worms are easily seen when the abomasal contents is gently poured off. If the autopsy has been delayed for 24 hours or longer the
worms may no longer be visible. However, upwards of 10 000 epg are usually encountered if a faecal examination is done.

In chronic cases the carcasses are generally emaciated and pale. The abomasal mucosa shows hyperplastic metaplasia and the rugae are opaque and thickened. There is evidence of chronic red bone marrow hyperplasia combined with reversion to white bone marrow, the latter being the result of iron depletion.

The two helminthological factors affecting the epidemiology are firstly, retarded larval development (hypobiosis): On the Gauteng and Mpumalanga Highveld L4 are retarded from March to October, reaching a peak from May to June. In the eastern Karoo and the non-seasonal rainfall areas, L4 are retarded from March to September. Normal development of these larvae continues as soon as the environmental conditions become favourable and this is responsible for the spring rise of adult worms. In areas with a mild climate during winter, development, although longer, is continuous.

Secondly, the spring rise is associated with pregnant ewes especially when they are about to lamb or shortly thereafter, but is not limited to female animals. The spring rise is also referred to as the periparturient relaxation of resistance (PPRR) and has an immunological basis. The spring rise is the result of retarded (hypobiotic) L4 reaching adulthood and may persist for a long time as evidenced by the faecal egg count.

Diagnosis by faecal egg examination is of limited value, since the eggs are difficult to differentiate from those of the other trichostrongylids. Larval culture is a better method of identifying the nematodes and the larvae can be identified by the excellent article published by Van Wyk, & Mayhew.
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in 2013 in the Onderstepoort Journal of Veterinary Research. Clinical signs, especially the colour of the mucous membranes of the eyes are almost pathognomonic, since there are few helminthoses that cause such a severe anaemia in sheep. Charts correlating the colour of the mucous membranes with the probable infection rate are available. Bottle jaw is indicative, as are black faeces. Autopsy is the only reliable method. In peracute and acute cases, masses of the worms can be seen squirming the abomasal ingesta. In chronic cases, the presence of few worms, as well as the emaciated, anaemic and oedematous carcass and red bone marrow are diagnostic.

![A larval culture showing the masses of third stage larvae crawling up the sides of the bottle](image)

The diagnosis of chronic haemonchosis may be difficult, because of concurrent poor nutrition. Confirmation may have to depend on gradual disappearance of the syndrome after anthelmintic treatment. In this regard, management of haemonchosis by several means has been extensively documented by Dr. J.A. van Wyk and co-workers.

**Haemonchus placei** of cattle

These nematodes are virtually indistinguishable from *H. contortus*, but their ecological requirements, epidemiology and general behaviour so different that, according to many authors, a separate species is warranted. This is, however, not upheld by all helminthologists and the name *H. contortus* of cattle may still be found in the literature. The worm is also known as the Barber’s pole worm, wireworm, and “haarwurm” and is found in the abomasa of cattle. They are widely spread throughout the world. In South Africa it is common in the summer rainfall area. The life cycle is direct and the developmental period is 23-28 days.

The pathogenesis of bovine haemonchosis is essentially that of haemorrhagic anaemia due to the blood-sucking habits of the worms. Depending on the number of infective larvae ingested, haemonchosis can be peracute, acute or chronic. In the acute cases, death results from the rapid loss of blood (per rhexis), while in the acute cases death results from a combination of blood loss, anorexia and lack of erythropoiesis. Chronic cases are seen when small numbers of helminths are present and there is a chronic loss of a small amount of blood. Erythropoiesis cannot take place as the iron intake is usually lower during winter.
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As opposed to *H. contortus*, *H. placei* elicits a strong and lasting immunity. The immunity is produced by the M4 and early 5th stages, while functional antibody is produced by the 5th stage and adult worms. After initial infection, calves develop a marked immunity and subsequent infestations are retarded in the late 4th stage or as stunted 5th stage worms.

After the age of about 2 years, cattle are relatively immune although this may be broken down by massive infestations often acquired around waterholes during drought conditions, or by poor nutrition. Only about 8% of calves fail to develop the immunity.

Self-cure has not been shown to occur and treatment with anthelmintics (or the removal of adult worms) may interfere with the development of the immunity.

**Teladorsagia circumcincta** of sheep and goats

The brown stomach worm occurs world-wide in the abomasum of sheep and goats: The life cycle is direct. Infective larvae enter the gastric pits, where they develop and moult to the 5th stage worms. Adult worms start emerging from the gastric glands after 18 - 21 days (developmental period). L4 may still be found in the mucosa 8-12 weeks after infection; this is the prolonged histotropic phase and is not to be confused with hypobiosis

The pathogenesis is largely brought about by the L3 and L4 that cause enough pressure necrosis in the glandular epithelium to destroy the function of the parietal and zymogen cells. The result is that the pH rises from 2 to 7, in which environment pepsinogen is not activated to pepsin (above pH 5 pepsin activity is negligible), and that digestion of food cannot take place. In addition, protein is not denatured and bacteriostatic activity is lost, resulting in an increase in the number of bacteria in the abomasum. The pepsinogen output is further reduced, again resulting in reduced pepsin activity. Adult worms suck a lot of blood, as evidenced by the fall in the haematocrit and the haemoglobin values 3-4 weeks after infection, but haemorrhage into the abomasal lumen does not occur.

The clinical signs in sheep are anorexia, weight loss, anaemia, submandibular oedema, occasional diarrhoea and death. Angora goats show marked oedematous swelling of the abdomen and limbs ("waterpens").

Anaemia, submandibular and sternal oedema, emaciated and dehydrated carcasses, evidence of diarrhoea, hyperaemic abomasal mucosa, sometimes small abscesses in the gastric pits where the larvae live, sometimes, depending on the duration, scar tissue where the gastric pits used to be. The entire mucosa has an 'ostrich leather’ appearance, each nodule having a small central opening through which the worms can be seen. The picture is one of severe diffuse hyperplastic nodular abomasitis which may be confluent and covered with tenacious mucus,

Once adult worms are present, L4 are retarded and once a sheep has been infected, the challenge infection moves from the pyloric to the fundic region of the abomasum, possibly indicating a degree of local immunity. Apparently, if the adult worms are removed, sheep show a good degree of immunity against subsequent challenge infections, but not against maturing L4 and fifth stages.
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In the winter and non-seasonal rainfall areas, larvae are common on pastures from about April to September if the temperature varies from 14-18°C and there is adequate moisture. The relative abundance of L4 in the respective hosts increases markedly from May to September.

*Teladorsagia circumcincta* is very prevalent in sheep in the areas adjacent to Lesotho and less so in sheep in the Eastern Province and the eastern Karoo. On irrigated pastures worm burdens vary from April to October, most of the worms being L4 (retarded).

The epidemiology in goats is unknown, but the parasite is of importance in Angora goats in the Eastern Province. The goats are always more heavily infected than the sheep when the two species graze together on the same pastures.

The autopsy is diagnostic.

![Spicules of a *Teladorsagia circumcincta* male (left) and the lesions in the abomasum produced by the worms (right). These lesions are mild but in severe infections the nodules coalesce](image)

**Ostertagia ostertagi** of cattle

This parasite is known as the brown stomach worm is found in the abomasum of cattle. These worms occur world-wide and in South Africa they are common in the Eastern and Western Cape Provinces. The life cycle is direct and the developmental period is 18-24 days. Infective larvae enter the gastric pits, where they moult to the 5th stage after 9-11 days. However, L4 may still be found in the mucosa 8-12 weeks after infection. This in known as the prolonged histotropic phase and is not to be confused with retardation or hypobiosis.

The pathogenesis is similar to that of *T. circumcincta* in sheep. In light infestations, the lesions are visible as a few raised nodules with central openings and the rugae may be thickened. In massive infestations the nodules are tightly packed together and are often confluent to form large, uneven greyish-white patches or plaques.

From 18-21 days after infection (developmental period) the worms are liberated from the tissues and the nodules undergo necrosis. Small ulcers may form and these may extend to the muscularis mucosa. During the developmental period a number of changes as result of the tissue destruction may set in.
Immunity takes a long time to develop, because adult worms are constantly replaced by emerging larvae, inhibited in the 4th stage. The presence of adults inhibits larval development, and if adults are removed, larvae will continue to develop. With re-infection adult worms are stunted, and after prolonged exposure there is a resistance to the establishment of the infection (population limitation).

*Ostertagia ostertagi* occurs in the Eastern and Western Cape Provinces but the epidemiology is largely unknown. However, peak adult worm burdens are seen in spring and inhibited L4 increase at the same time. These L4 remain dormant through the summer and develop into adults the following winter. The parasite is essentially a winter parasite, adults being scarce or absent during summer. Heavy infections on especially irrigated pastures can cause severe losses of calves. As can be seen from the foregoing, there is little difference in the pathogenesis and pathology of ovine teladorsagiosis and bovine ostertagiosis.

![Spicules of an *Ostertagia ostertagi* male (left) and the lesions in the abomasum produced by the worms (right)](image)

**Trichostrongylus axei** of sheep, goats and cattle

Stomach bankrupt worm: It is found in all the domestic ruminants, pigs, horses and occasionally, man. It occur world-wide wherever ruminants are kept and is present in the abomasum, stomach and duodenum. The life cycle is direct, and the developmental period in ruminants is 24 days and in horses 25 days.

Fair numbers of infective larvae are necessary to cause clinical disease, e.g. 40 000 larvae cause death in Dorper sheep, but not in Merinos. The worms cause an increase in the pH, the abomasal and serum pepsinogen, and a decrease in the available nitrogen. In sheep the carcass is emaciated, the abomasal ingesta are foul-smelling and there is oedema of the abomasal mucosa. Catarrhal gastritis due to larval action occurs. Mature worms cause thickening of the mucosa, which resemble wart-like plaques or "ringworm-like" lesions. If these thickenings are removed, erosion with an intensely hyperaemic base remains. With heavy infections, the thickened areas coalesce to produce a diffuse hypertrophic gastritis.

Immunity develops in sheep after the primary infection and it is possible to protect animals by giving them small numbers of infective larvae (approximately 1/10th of a lethal dose). The worms can then be eliminated by anthelmintics and a residual immunity should result. Infection with *T. axei* has an adverse effect on simultaneous infection with *Haemonchus* spp., *T. circumcincta* and *O. ostertagi*.
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Very little is known about this worm in cattle in South Africa.

Lesions in the abomasum (left) and anterior part of the duodenum (right) caused by Trichostrongylus axei

**Bunostomum trigonocephalum in sheep and goats**

This worm is known as the grassveld hookworm that affects sheep, goats and certain antelope. They are found in the small intestine. The life cycle is direct and the developmental period is in the region of 30-60 days. Infection occurs either percutaneously or per os. In the former way method of infection, the larvae are transported via the blood to the lungs where they break through the alveoli and are coughed up to eventually end up in the small intestine.

Three syndromes can be recognized: Skin penetration, where initial infection causes swelling and within 24 hours the formation of small isolated scabs. Repeated infections cause severe swelling that may persist for several days; intestinal lesions caused by the large mouths of the adults that cut the intestinal villi at their bases. Exposed, haemorrhaging ulcers remain when the worms move to a new feeding site and anaemia that develops gradually with haemoglobin levels dropping to as low as 0.35 g/l. The anaemia is of the progressive aplastic type with no regenerative changes occurring in circulating red cells.

Clinical signs include itching of the skin, particularly that of the limbs, and wet eczema. Rapid mass loss, emaciation, anaemia, submandibular oedema and constipation followed by diarrhoea, the faeces being fetid and tarry may occur. Animals lie down for a few days before they die.

A massive dose can kill adult sheep without any antemortem signs (4 000 larvae) being evident. However, more commonly, as few as 200 - 300 adults can cause severe signs of anaemia causing a fall in the haematocrit levels as well as hypoalbuminaemia.

Because of the marked reactions in the skin after re-infection, infective larvae are trapped and are unable to enter the host (see also Strongyloides papillosus). An inflammatory reaction occurs in the intestine after re-infection but is limited to the mucosa. This prevents the worms from attaching properly, and they may make several attempts to do so, damaging the mucosa even further.
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The parasite occurs in areas with a rainfall of 500 mm or more. Kraaling facilitates the spread of the infection. It is rife in Lesotho and the eastern Free State and older animals carry heavier burdens than young ones.

**Gaigeria pachyscelis in sheep and goats**

These are the sandveld hookworms of sheep, goats, impala and wildebeest. They occur in the small intestine and are distributed world-wide. The life cycle is direct. Only percutaneous infection takes place and the developmental period is 70 days.

The worms are virulent blood suckers of which 100 can cause the death of a sheep in less than 100 days. As few as 44 worms can cause severe anaemia and generally 50 worms are considered a severe infection. In addition, the larvae cause allergic skin reactions, and the lesions in the small intestine caused by the adults are similar to those caused by *Bunostomum*.

Since the only route of infection is through the skin, the same reaction as seen in infections caused by *Bunostomum* species occurs. The immunity develops quickly and adult animals have a good immunity against re-infection.

The parasites are common in the western parts of the Northern Cape Province and Namibia. The free-living stages are very susceptible to desiccation, heat and cold and yet they occur in the arid and semi-arid regions of the country. Water is scarce in these areas and the only supplies are derived from bore-holes which invariably lead to leaking water troughs. This apparently provides adequate moisture for the infective larvae to survive.

**Bunostomum phlebotomum in cattle**

This is the hookworm of cattle that occurs in the small intestine: The life cycle is direct. The developmental period is 52-56 days and infection takes place percutaneously or per os.

The three syndromes as seen in sheep and goats also occur in cattle, i.e. skin penetration, intestinal lesions, and anaemia. The clinical signs are as described for sheep, although not so pronounced. As few as 2 000 adults can cause severe signs of anaemia and death.

Cattle apparently become resistant at about 5-9 months of age, which results in the elimination of the entire infection.

Eggs hatch and develop in the dung pad but good rain is needed to liberate the larvae. In the Northern Cape Province the infection is essentially a kraal infection and infective larvae are usually present only near leaking drinking troughs during the dry season. When good rains fall, larvae are present throughout the kraal and calves are infested from about December. The infection disappears when rains cease in April and May. Peak worm burdens are reached during July and August, but only in young calves. Yearlings have little, if any, hookworms, indicating the development of a good immunity.
The Cooperia species

The species involved in both sheep and cattle are *C. pectinata*, *C. punctata*, *C. spatulata*, *C. oncophora*, *C. mcmasteri* and *C. curticei* occur together with the others listed above. They occur in the small intestines of a wide range of ruminant species world-wide. Unless infected by massive doses (300 000 L3 or more during a ten-day period) clinical signs are seldom seen. They seem to be more important in cattle.

They are known as cattle bankrupt worm and occur in the small intestine. In severe infestations, there is a fluid foetid diarrhoea, selective anorexia, bottle jaw and eventually death which results from starvation, dehydration and exhaustion. Necrotic enteritis with parasites penetrating the mucosa, haemorrhages in the first 3 metres of the small intestine and catarrhal exudate in the posterior half of the small intestine are seen on autopsy.

Calves are markedly resistant to reinfection 3-4 months after the initial infection. The parasites are only of importance in adult cattle that are stressed, e.g. with repeated pregnancies, poor quality food or during and immediately after long periods of drought. The infective larvae are extremely resistant to desiccation and, particularly in areas with long winters, they can survive for months. When good rains fall, the larvae are released from the dung pads and, where calves are crowded in kraals or pens, it can be particularly dangerous in summer. The infection is essentially a summer one with minor differences, depending on rainfall patterns.
Helminth infections of ruminants

Two Cooperia spp., *C. oncophora* (left) and *C. pectinata* (right) that occur in a wide range of ruminant species

**The Trichostrongylus species**

Bankrupt worm: The following species are found mostly in the intestine: *Trichostrongylus colubriformis, Trichostrongylus rugatus, Trichostrongylus calcatus* and *Trichostrongylus vitrinus*. All of these species are known by the same common name and occur world-wide. They tend to occur in the first 7 metres of the small intestine, less often in the abomasum. Almost any animal species can be infected but dealt with here are the ones most often found in ruminants. The life cycle is direct with a developmental period 18-20 days.

In the acute disease, the L3 penetrate between the epithelial glands of the mucosa and form tunnels beneath the epithelium. When these tunnels rupture to release the young adult worms, there is considerable haemorrhage and oedema, and plasma proteins are lost into the lumen on the gut. The chronic disease is much the same as the acute disease, but less noticeable.

In acute cases, the pain caused by the parasite causes anorexia, closure of the pyloric sphincter, and retention of food in the abomasum and rumen. Sheep become listless, signs of submandibular oedema develop and there is yellow foetid diarrhoea. Sheep die 16-17 days after infection. Acute disease is rarely seen.

Anorexia gets progressively worse in chronic cases, with concomitant loss of body mass. Anaemia is caused by a lack of available protein to form haemoglobin. The overall result is emaciation, atrophy of muscles, hydrothorax, hydropericardium and ascites. The clinical signs may be aggravated by poor grazing especially during winter. This form of the disease is the more commonly one seen. Dorper sheep seem to be more susceptible to trichostrongylosis, and they develop a slight transient bottle jaw. Faeces become putty-like but not fluid and animals become weak and listless. Mucous
membranes become pale. Merino sheep show slightly pale mucous membranes and putty-like faeces. On lush green pastures the sheep may have dark diarrhoea. The chronic form of disease develops when 100 000 larvae are ingested. Since these changes occur over a period of time and the effects are not immediately visible, the name ‘bankrupt worm’ is apt, i.e. the worms bankrupts one before one realizes it.

The carcass of the acute case is emaciated and there is atrophy of the fatty tissues. The intestines show catarrhal inflammation with numerous small petechiae in the first few metres. The intestinal walls are thickened and the mesenteric lymph nodes are enlarged. Adult parasites are found beneath the greyish white film that covers the mucosa, but their presence can only be determined by examining a scraping of the mucosa against the light. Changes such as fluid accumulations in the serous cavities, ruminal atony and food retention in the rumen and abomasum, dry ingesta and distension of the small intestine by fluid may also occur. Carcasses are markedly emaciated in chronic cases, and there is muscular and myocardial atrophy. Mucous membranes are generally pale and the intestinal walls may be thickened.

Existing infections are expelled when sheep acquire a new infection, indicating that some form of immunity exists. Occasionally self-cure occurs. Retarded larvae and females that are not laying eggs are frequently expelled and the sheep’s condition improves after self-cure has taken place. Once sheep have acquired immunity to T. colubriformis, subsequent infections are retarded in the 5th stage.

Eggs that contain fully formed embryos can withstand desiccation for 15 months, to emerge when the first good rain falls. Infective larvae (L3) can withstand desiccation for a few months, provided the temperatures are low. Infective larvae are most numerous in late winter and outbreaks are usually seen in spring. The larvae almost disappear in summer.

In the winter rainfall areas T. vitrinus is the most important parasite, increasing in numbers from June to September, and diminishing markedly thereafter.

All the Trichostrongylus species mentioned occur in non-seasonal rainfall areas. Pastures are heavily infected from February to September and peak worm burdens are reached at the end of winter, i.e. August or September.

In the summer rainfall areas Trichostrongylus rugatus and T. colubriformis are important in the Eastern Cape Province. On the Gauteng and Mpumalanga Highveld, T. colubriformis and T. falculatus are present in moderate numbers. Irrigation on Highveld farms can lead to a tenfold increase in worm burdens when compared with sheep grazing on dry land pastures.

The dominant worm in the semi-arid areas area is T. falculatus and the other species are seldom encountered. Trichostrongylus falculatus differs from the other Trichostrongylus species in that the usually low burdens can increase markedly from October to March if preceded by at least 25 mm rain during the warmer months.

Faecal nematode egg counts are of little value. Faecal cultures are essential for generic identification of the larvae (L3). Clinical signs are diagnostic provided that malnutrition and poor management can
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be excluded. Consider the age of the animals and the season. The presences of large numbers of worms at autopsy as well as the fairly typical lesions are diagnostic.

The parasites behave similarly in cattle as in sheep, but in cattle, the resulting syndrome caused is considerably less severe and is hardly noticeable. It is seen mostly in the young animals.

Spicules of *Trichostrongylus colubriformis* (left) and *Trichostrongylus falcatus* (right)

**Strongyloides papillosus**

White bankrupt worm of sheep, goats, cattle and wild ruminants occur all over the world. The life cycle may be homo- or heterogonic. Eggs are characteristic in that they contain a fully developed, unsheathed larva by the time they are excreted from the host. Infection takes place percutaneously, or, in new-born animals, through the milk. Those larvae that have entered percutaneously migrate via the venous system to the lungs while those that are ingested in the milk first penetrate the intestinal wall and then move to the lungs via the venous system. In the lungs they moult, migrate to the bronchi and are swallowed. Adult females develop in the small intestine. The developmental period is from 8 - 14 days.

Skin penetration causes an erythematous reaction, which can be secondarily infected. Animals become sensitized to the larvae after the first infection and upon reinfection a marked allergic reaction at the entry site occurs. The L3 cause erosive enteritis with only the muscularis remaining. Acute diarrhoea and secondary bacterial infections are common. The haematocrit and the haemoglobin levels fall due to interference with digestion and protein absorption. Anorexia is seen and the animals starve to death. Kids (goats) are more susceptible than lambs; approximately 11 000 L3 can be lethal.

Marked urticaria, caused by the larvae, occurs at the site of infection. Adult worms cause anorexia, diarrhoea or constipation, sunken eyes with a purulent discharge, a frothy mucous discharge from the nose, muscle atrophy and paresis just before death.

Young animals are severely affected and as they grow older, resistance develops. The resistance is mostly due to a hypersensitivity reaction in the skin of previously infected animals that traps the penetrating L3. Older animals seldom die of the infection, unless they had no previous exposure.
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The larvae are not sheathed and are susceptible to adverse climatic conditions. However, warmth and moisture favour their development and survival and allows the accumulation of large numbers of infective larvae. It is for this reason that lambs or kids in small kraals (pens) may become heavily infected in the summer months.

Another major source of infection for the very young lambs and kids is the reservoir of larvae in the tissues of their dams. These larvae can cause clinical *strongyloidosis* in animals during the first few weeks of life.

Faecal egg counts; the eggs are the only ones that have a thin shell and contain the L1 at the time they leave the host. Remember that apparently healthy animals may have high egg counts. Clinical signs may be indicative. The finding of the worms at autopsy is diagnostic. In heavy infections they appear as threads of cotton wool clumped together.

The reader is referred to the publication ‘Experimental studies on *Strongyloides papillosus* in goats’, authored by Pienaar, J.G. *et al.* in 1999, and published in the *Onderstepoort Journal of Veterinary Research*, volume 66, pages 191-235, for a comprehensive overview, including illustrations, of the condition caused by this nematode.

**Nematodirus spathiger**

This worm is known as the long-necked bankrupt worm and is found in the small intestines of sheep, goats and various antelope. It occurs world-wide wherever cattle and sheep are kept. The life cycle is direct. The first 3 stages of this worm develop inside the egg, which make it one of the characteristic nematode eggs, and the infective L3 hatches. Developmental period is 14-21 days.

![Spicules of the *Nematodirus* spp., *N. spathiger* on the left and *N. helvetianus* on the right (Left figure). The female tail typically bears a terminal spine (Right figure)](image_url)

The L4 remove the epithelial cells of the villi and superficial necrosis of the lamina propria occurs. Extensive necrosis develops when M4 and the early 5th stages are present, due to the worms feeding on the living cells. This impairs the ability of the intestine to exchange fluids and nutrients and diarrhoea develops. The worms return to the lumen, allowing the mucosa to heal.
Clinical signs include depression, listlessness, anorexia, fluid faeces and death within 10-14 days after infection. The clinical signs are caused by the L4 rather than the adults.

Usually only suckling lambs are susceptible; lambs older than 3 months are highly resistant. In such lambs L3 are eliminated, the development of L4 is retarded, there is reduced egg production by the females, and adult worms are eliminated. More female L4 are retarded and more adult females are expelled. Stress, and other diseases can break down the resistance.

The eggs and larvae are highly resistant both to desiccation and temperature and may survive for a year. A temperature of 5°C kills them after 16 days.

In semi-arid areas, if the monthly rainfall exceeds 25 mm, infection increases, even in midsummer. This is because all the larvae that have developed to L3 in the eggs hatch at the same time. The first growth of grass after rain usually takes place in the heavily manured areas, such as around drinking troughs. Rain causes the infective larvae to hatch and stimulates their migration. The first green grass is grazed immediately by the young lambs, which then acquire the infection that in some cases may be considerable. With heavy infections, the young lambs die, while the older animals show transient diarrhoea and recover. With continuous rain, even the older animals die because of the combined effect of stress and continuous intake of large numbers of infective larvae. Similar outbreaks occur on irrigated pastures for the same reasons given above.

In winter rainfall areas: Larvae are always present, but are more numerous in winter, and in the non-seasonal rainfall areas peak numbers of infective larvae can be expected after rains, whatever the season.

Faecal nematode egg counts are of little value because the L4 cause the clinical signs. However, the egg is characteristically large, much larger than those of the other intestinal nematodes and can be easily identified. Clinical signs, the age of the animals and the season. The presence of large numbers of L4 rather than adult worms at autopsy is diagnostic.

**Nematodirus helvetianus**

Also known as long-necked bankrupt worm that occurs in the small intestine of cattle world-wide. The life cycle is like that of *N. spathiger*. Larval stages and adult worms tunnel into the mucosa, causing desquamation. Animals loose mass and lymphocytes increase after 24 days. Lymphocyte levels drop and eosinophil levels rise, the latter coinciding with a drop in the haemoglobin. The animal's temperature rises about 14 days after infection and the faeces become soft. Persistent diarrhoea is seen when the worms are adult and is at its worst 3-4 weeks after infection. The autopsy, histopathology and immunity are similar to that of the other species.

The parasite is rarely diagnosed in cattle in South Africa but occurs on isolated farms in KwaZulu-Natal and the Eastern Cape Province. Once calves reach the age of 5 months, faecal egg counts rarely show the presence of *Nematodirus* eggs.
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**Toxocara vitulorum**

This worm is known as the ascarid of cattle and is only seen in suckling calves. It is morphologically very similar to *A. suum*, but the eggs have a thick wall that is finely pitted. Infection takes place via the milk of the cow and adult worms are present in the calves after 33 days. Calves lose weight as a result of the diarrhoea and colic that accompanies the infection. This is one of the emerging helminthoses and is on the increase in recent years.

**Oesophagostomum columbianum nodular worm of sheep**

Adult worms occur in the colon, and immature stages in the wall of the small intestine of sheep and goats world-wide and in some antelope in southern Africa. The life cycle is direct. The development period is 35-39 days for the first infection, increasing to 46-47 days to many months for subsequent infections.

L3 enter the wall of the small intestine, undergoes the M1 and the L4 migrate to the lumen, then to the colon and some enter a second histotropic phase. The remaining L4 develop to the 5th and adult stages in the colon. Oesophagostomosis is usually seen in warm, damp conditions following good rains on natural pastures during summer and autumn.

Anorexia is caused by the intestinal discomfort starting with the larvae's migration to the gut lumen and it persists until death or recovery. A mild mucoid to projectile fetid diarrhoea sets in and this may lead to intussusception. Hypoproteinaemia develops which is probably the result of reduced food intake. Death may occur from the 18th to the 22nd day and is caused by starvation, dehydration and exhaustion.

Acute disease is seldom seen. It is caused by larvae that have not yet reached patency and is characterized by a rise in the temperature, diarrhoea, and rapid dehydration.

**Chronic disease**: Food and water consumption decrease only to improve again. Diarrhoea starts from the 10th day onwards and persists until death. The faeces vary in consistency from putty-like to mucopurulent, green and evil-smelling. Intussusception may occur. This form of oesophagostomosis used to be quite common, but due to intensive treatment, aimed mainly at *H. contortus*, it has become scarce.
Clinical signs of 'reksiekte' or oesophagostomosis (top left), the typical appearance of the small intestine with numerous nodules (top right). The opened intestine showing the nature of the nodules (middle left) and several adult worms (middle right). An abscess in a nodule as a result of bacterial infection (bottom left) and haemorrhagic nodules as seen in *Oesophagostomum radiatum* infection.

Thickened patches are noted on the intestinal mucosa, and as the larvae leave the mucosa, ecchymoses appear. Extensive nodule formation as well as thickening of the mucosa of the colon occurs, together with peritonitis and adhesions. There may be a diphtheritic jejunitis, typhlitis and colitis, with numerous perforations and adhesions. At a later stage, the nodules may calcify. Once the worms have reached patency, only nodules and a thickened intestinal wall with adhesions may be visible.

The immunity against *O. columbianum* is local, mainly in the large intestine. At first there is a marked local eosinophilia and oedema of the gut wall, and increased mucus secretion expels the new infection. Some of the larvae migrate into the lumen, causing diarrhoea which expels the existing adult worms and also some of the newly developed L4. The process is repeated and only ceases when environmental conditions reduce the challenge. Higher antibody titres are present in the mucus of the gut, and acquired immunity is manifested in the proliferation of lymphoid tissue in the large intestine. Larvae (L3) are smothered by the antibodies and the excess mucus production is also possibly a non-specific result of the antigen-antibody reaction. The prolonged histotropic phase of the L4 in the wall of the intestine seems to be a natural part of the life cycle and is not necessarily associated with an immune reaction.

The free-living stages are susceptible to desiccation and require hot, moist conditions for optimal development to the infective stages. The species is present in the summer rainfall areas where rainfall is in excess of 360 mm per annum. It is absent in the semi-arid, non-seasonal rainfall and winter rainfall areas.
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Massive infections take place in summer after good rains. Sheep quickly expel the worms on improved pastures, and the parasite is absent on irrigated pastures, with the exception of March and April.

**Chabertia ovina**

These are the large-mouthed bowel worms of sheep, goats and rarely cattle, and also occur in some antelope. Their habitat is the colon and they are found wherever sheep are kept and the climate is suitable. The life cycle is direct and the developmental period 49 days or more.

These worms browse on the mucosa causing haemorrhage from the 4th week onwards and faeces become flecked with blood. There is mass loss, diarrhoea and faeces may be flecked with blood.

The mucosa of the caecum and colon contain haemorrhages and inflammatory areas, produced by the adult worms. The adults cause rather extensive damage, since they frequently move to new sites to feed. Only in heavy infections do the immature stages cause tissue damage, and then throughout the entire intestine, from the pylorus to the ileo-caecal valve.

*C. ovina* is confined to winter and non-seasonal rainfall areas and is present throughout the year in moderate numbers. Eggs can survive cold, but are sensitive to desiccation, whereas the infective larvae can withstand cold and desiccation, surviving for a year or longer.

**Oesophagostomum radiatum** of cattle

Nodular worm of cattle occur in the colon and are distributed world-wide. The life cycle is direct. The developmental period is 32-34 days. Wet, warm weather and calves in overgrazed camps or in unhygienic kraal and pens are most susceptible and can be expected to show clinical signs.

Pain causes severe anorexia from the 4th week onwards, and blood is passed in the faeces from as early as the 16th day. This becomes more severe from the 19th and 20th day as the worms moult (M4) and haemoglobin levels fall from the 4th week onwards. As much as 9.5 liters of blood are lost during this period.

Pain, anorexia, loss in mass, hypoproteinaemia, anaemia and diarrhoea which is often foetid and blood-stained are present. Submandibular oedema and progressive cachexia occurs after 7 weeks. The first effects are noticed 3-5 weeks after infection; an improvement is seen after 10 weeks and animals may have recovered by 14 weeks.

On autopsy thickened patches are noted on the intestinal mucosa, and as the larvae leave the mucosa haemorrhages appear. Nodule formation as well as thickening of the mucosa of the colon occurs, together with peritonitis and adhesions. Once the worms have reached patency, only nodules and a thickened intestinal wall with adhesions may be visible.

Cattle in the field develop a strong immunity after 8-12 months of age. The 5th stage and adult worms provide the main stimulus to immunity but larval stages also do so. The immunity eliminates all or most of the L4 of the challenge infection shortly after they have returned to the gut lumen. This elimination is accompanied by diarrhoea. The immunity may also cause a great reduction in the
number of re-infecting larvae developing to adults during the normal prepatent period. An important point is that even if the adult worms are removed by anthelmintics, the resistance persists.

The parasite does not occur in the arid, non-seasonal and winter rainfall areas. In the semi-arid areas, *O. radiatum* is found in modest numbers in calves in May. In the summer rainfall areas, calves are infested throughout the year, burdens rising to a peak in August and declining in spring. Irrigated pastures are unsuitable for this parasite.

Faecal nematode egg counts are of little value. Clinical signs and the presence of haemorrhagic nodules or small abscesses in the wall of the small intestine and colon are diagnostic at autopsy.

**Trichuris spp.**

These are known as whipworms because of their long, thin anterior end and a short, thick posterior. They are found in the caecum and colon. The eggs are lemon-shaped and have a plug at each end. They contain an infective L1 and can survive for years in dry faeces. The eggs are also resistant to desiccation and survive temperatures of –20°C to 50°C.

Eggs are passed in the faeces and after 40 days to several months an infective L1 has developed inside the egg. The eggs are ingested by the host and hatch in the abomasum or intestines. L1 burrow into the wall of the intestines to undergo their moults, return to the lumen and move down to the caecum or colon where they will reach maturity. The developmental period is 53-57 days.

Larval stages cause haemorrhages and local oedema when they penetrate the intestinal wall. These injuries are considered to cause secondary bacterial infection. The adult worms are not pathogenic, unless present in large numbers when they may cause abdominal pains, mucoid diarrhoea, anaemia, loss of body mass and rarely, death.

![Typical morphological appearance of *Trichuris*. Note that the females’ posterior is slightly curved, while that of the male is spirally coiled (top left). *Trichuris in situ* in the caecum (top right) and a close-up of the same (bottom left). The egg is the third that can be identified to the genus level in a fresh faecal sample – the opercular plugs and lemon-shaped apperance are characteristic.](image)
THE CESTODES

The genus *Moniezia*

The two species that are involved are *Moniezia expansa* and *Moniezia benedeni*. The genus can be recognized by the double set of reproductive organs in each proglottid. Both species occur in the small intestine of sheep, goats, calves and wild ruminants and use oribatid mites as intermediate hosts.

The specific characteristics of *M. expansa* are the presence of interproglottid glands that extend right across the width of the proglottid and the triangular eggs. The parasites are found throughout southern Africa and are rife on irrigated pastures, where they occur from November to May. On dryland pastures they are present from October to April.

*Moniezia benedeni* differs from *M. expansa* in that the interproglottid glands are condensed to a short row in the middle of the proglottid and that the eggs are pyriform (diamond-shaped). This parasite is common throughout southern Africa on pastures, both in summer and winter rainfall areas.

The developmental period of both these tapeworms is approximately 6 weeks and the adult worms are short-lived, infections persisting only for about 3 months. Both tend to occur only in young animals and a good immunity develops after the first infection. They do not cause significant clinical effects, even in large burdens. However, clinical signs may include unthriftiness, diarrhoea, respiratory signs and even convulsions but then only with massive burdens. The benzimidazoles are erratic in their efficacy and resistance has been recorded from several strains of the parasites. Ploughing or avoiding the use of the same pastures for young animals for consecutive years may reduce the mite population.

*Moniezia expansa* (right) with the interproglottidal glands spread over the width of the proglottid and (arrow) *Moniezia benedeni* (left) with the interproglottidal glands concentrated in the middle (arrow)

The genera *Avitellina* and *Thysaniezia*

*Avitellina* is common in the semi-arid areas and occurs in the small intestine of a variety of ruminants. The intermediate hosts are thought to be psocids. There is only one set of reproductive organs per
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proglottid and the genital pores open dorsal in the mid-line. When held against the light there is a distinct white line formed by the paruterine organs down the middle of the body.

Although *Avitellina centripunctata* is probably the most commonly encountered one, there is some doubt as to the actual number of species that occur in the country. Reference to the genus rather than a species is preferable.

This tapeworm is considered apathogenic and occurs in young animals. They can be eliminated with the same compounds used for the *Moniezia* species. The efficacy is also the same as for the *Moniezia* species.

*Thysaniezia giardi* occurs in the small intestine of cattle and adult sheep. It uses oribatid mites as intermediate hosts. There is only one set of reproductive organs per proglottid and the genital pore alternates irregularly. This is the only tapeworm occurring in herbivores that has frills on the proglottids; these are the paruterine organs. The worms are distributed all over southern Africa. The tapeworms are considered apathogenic and can be eliminated with the same compounds used for the *Moniezia* species. The efficacy is also the same as for the *Moniezia* species.

*Thysaniezia giardi* (left) showing the irregularly alternating genital pores and the distinct paruterine organs, and *Avitellina* sp. (right) with the centrally situated paruterine organs and *Avitellina* sp. (right) with the centrally situated paruterine organs.
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INFECTIONS OF THE LIVER

Liver fluke may infect most mammals, including man. In South Africa the more common fluke is *Fasciola gigantica* with *Fasciola hepatica* occurring in a few localities only. The intermediate hosts are the semi-aquatic snails *Lymnaea natalensis* for *F. gigantica*, *L. truncatula* for *F. hepatica* and *L. columella* for both. (Figure 1) *Lymnaea natalensis* prefers warmer climates than the other two snails, and fasciolosis is not seen in semi-arid or arid parts of the country. The parasites occur in the bile ducts and the life cycle is indirect. The minimum period of development from miracidium to cercaria is 17 days and cercaria can be shed after 36 days (25 days for *F. hepatica*). The entire developmental period spans 13 - 16 weeks (8 – 12 weeks for *F. hepatica*).

(Fig. 1) Habitats where the intermediate hosts can survive. Top left, irrigated pasture, top right hoof prints of cattle, bottom left, panveld so typical of the Mpumalanga highveld, South Africa bottom right, even irrigation canals are utilized.
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Snail shells. Left, *Lymnaea columella*, middle, *Lymnaea truncatula* and right, *Lymnaea columella*. Note the opening of the body is on the right-hand side, when the tip of the shell points away.

The golden eggs of *Fasciola* get their colour from bile pigments (top left). A miracidium (top right) will enter a snail and from large redia (bottom left) that will form much smaller cercaria that have single tails (bottom left). The cercaria escape from the snail and encyst on solid objects to form the metacercaria (bottom right).

The pathogenesis in cattle and sheep is the same, but sheep react severely to the parasites. The young fluke grows fast and increases 8-fold in size in 15 days as it bores through the parenchyma of
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the liver. In massive infections the host usually dies of internal haemorrhage, while milder infections cause hepatic fibrosis and hyperplastic cholangitis.

During the initial 6-7 weeks of their migration, the flukes cause a mild anaemia due to the blood that seeps through into the migration tracts. The loss of plasma protein caused by the flukes is less important than the anaemia. When the worms arrive in the bile ducts, anaemia develops rapidly, as the worms are avid blood suckers.

The three factors that are basically responsible for the anaemia are an increased rate of destruction, a compensatory increase in haemopoiesis and continued draining of iron reserves, very similar to what happens in haemonchosis.

With peracute infections, animals die without showing any clinical signs. This is the result of massive internal haemorrhage that occurs with these infections.

Acute infections result in the death of animals while the flukes are still migrating in the parenchyma (after 6-7 weeks). If the animal can be examined, there is evidence of severe macrocytic anaemia and high AST levels. Fluke eggs are not present in the faeces.
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Subacute cases show a rapid loss of body mass, severe anaemia and high fluke egg counts. Death occurs from 12-20 weeks after infection.

Gradual wasting, severe anaemia with ascites, oedema, bottle jaw and high fluke egg counts are seen with chronic infections. Death may occur more than 20 weeks after infection.

![Severe anaemia as indicated by the metaplasia of yellow bone marrow to red (left) and distinct submandibular oedema (right)](image)

Usually there is no obvious reaction to the passage of the young flukes through the intestinal wall and on the peritoneum, except for small haemorrhagic foci where the flukes have temporarily attached. Parasites may sometimes be found in ascitic fluid. In heavy and repeated infections, peritonitis, either exudative (acute) or proliferative (chronic), occurs.

In acute cases of penetration of the liver, there are fibrino-haemorrhagic deposits on the serous surfaces and in chronic cases there are fibrous tags and adhesions (Figure). In the liver parenchyma itself there are numerous haemorrhagic migration tracts and in heavy infections the liver has a streaked appearance. Tracts from which the debris has been cleared appear yellowish as a result of eosinophil infiltration (Figure). Some flukes do not reach the bile ducts and form cysts in the parenchyma. These cysts consist of a connective tissue capsule and a dirty brown content of blood, debris and excrement from the fluke. Aberrant migration is uncommon, but flukes have been found in the lungs.

Mature flukes are present in the bile ducts where they obstruct the flow of bile, cause mechanical irritation, and produce toxins. Changes in the bile ducts are more severe on the left side. The bile ducts stand out as firm, whitish, branching cords. When these ducts are incised, flukes, detritus and clumps of eggs in a brown fluid may pour out.
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Fibrous tags on the liver surface (top left), migration tracts filled with eosinophils (top right), aberrant migration to the lung, where the parasite caused localized haemorrhage (bottom left) and flukes, and detritus expelled from a bile duct (bottom right)

The lesions are initially limited to the larger ducts, but with time and repeated infections the smaller portal units are also affected. This is due to direct irritation by the flukes, superimposed infections (bacterial) and biliary stasis. The lesions heal by fibrosis and large areas of the parenchyma may become completely fibrotic. With repeated infections, the left lobe of the liver may become atrophied, indurated and have an irregular surface. With light infections the lesions are milder and are sometimes not detectable.

Cattle are generally resistant and adult worms live only for 9-12 months before being expelled. Few worms survive for 3-5 years. The extensive fibrosis and pronounced tissue reaction apparently are responsible for the resistance. Treatment with anthelmintic that will kill both young and adult flukes prevents clinical signs and such cattle develop a strong resistance. If they graze on contaminated pastures, their resistance becomes even stronger.

Adult sheep appear to be more resistant to the infection but not to the effects of the flukes, while young animals are fully susceptible.

Clinical signs, seasonal occurrence, prevailing weather patterns and history of fasciolosis on the farm form part of establishing a diagnosis. The autopsy is pathognomonic. Faecal egg counts are significant as the size, shape and coloration of the eggs are diagnostic. The number of eggs per gram of faeces can give an indication of the size of the infection, i.e. the intensity.

Liver function tests, particularly the GLDH (glutamate dehydrogenase) and GT (gamma glutamyl transpeptidase) can give an indication of liver damage, which may be due to fasciolosis. Antibodies of
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the flukes may be detected with the ELISA (enzyme-linked immunosorbent assay) performed on milk or serum, and the passive haemagglutination tests.

**Strobilate (adult) cestodes**

**Stilesia hepatica**

As the name implies, the worms occur in the bile ducts of a variety of ruminants. Two round paruterine organs form 2 distinct white lines that run parallel down the length of the gravid proglottides.

The life cycle is unknown. The intermediate hosts are reputedly soil mites of the family Oribatidae, which need rich, moist soil, and in which the infective stage, the cysticercoid, occurs. They cause neither clinical signs nor significant liver pathology, although massive infections may cause mild cholangitis. The worms are widespread throughout the subcontinent and are usually only encountered at autopsy or at the abattoir. Large numbers of livers are condemned because of the presence of this worm and represents considerable economic loss. Control by disturbing the habitat of the soil mites by ploughing, especially near kraals or places where dung accumulates.

*Stilesia hepatica* showing the paruterine organs on either side of the body (left) and the adult worm in the liver (right)
The larval cestodes or metacestodes

*Taenia hydatigena*

The final host is a canid, domestic dogs and jackal included. The immatures (metacestodes) are occasionally encountered in cattle. A large infection causes a condition known as *hepatitis cysticercosa*, which is characterized by migration tracts through the parenchyma of the liver. When fully grown, the metacestodes exit the liver in the region of the gall bladder and each forms the characteristic *Cysticercus tenuicollis* on the serosa. A differential diagnosis for *hepatitis cysticercosa* is fasciolosis and little can be done to treat the condition in the bovine. However, regularly deworming of all domestic dogs, properly cooking all meat (especially offal) before it is fed to dogs and preventing cattle to consume the tapeworm eggs in the faeces, do a lot to control the occurrence.

![A sheep’s liver with severe *hepatitis cysticercosa* (left) and fully developed metacestodes, on the outside of the liver (right)](image)

*Echinococcus granulosus*

Cystic echinococcosis or hydatidosis is fairly common in sheep and goats, and occasionally encountered in cattle. Once again, domestic dogs and jackal are the final hosts and ruminants are infected by eating or drinking food or water contaminated by faeces of these carnivores. The target organ for the metacestodes is the liver and the lungs, is commonly known as a hydatid cyst. Surgery is impractical, and as a rule animals are not treated. It is a zoonosis that receives world-wide attention.
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Sheep’s liver with numerous hydatid cysts (left) and a cyst in the heart of a bovine, opened to show the hydatid ‘sand’ or protoscoleces (right)

INFECTIONS OF THE CARDIOVASCULAR SYSTEM

The trematode *Schistosoma mattheei* (bilharzia worms) occurs in sheep and cattle but the prevalence in goats is not known. The sexes occur separately and the stout white male carries the slender black female in a ventrally situated gynaecophoric canal. Eggs hatch, miracidia enter the intermediate hosts, *Bulinus* (*Physopsis*) *globosus* and *B. (P.) africanus*, develop to sporoblasts and then to cercaria. The cercaria are the infective stage to the ruminants and enter percutaneously. The parasites are limited to those areas where the intermediate hosts occur.

The intermediate host of *Schistosoma mattheei* (left) and the cercaria with the typically forked tails

Adult worms occur in the mesenteric blood vessels, the portal system and less frequently the blood vessels of the lungs and urinary bladder. The entire adult life of the worm is spent inside blood vessels and eggs have to work their way through the walls into the gut or the bladder. They and must be freed from the faeces and diluted with a considerable amount of water before they will hatch.
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**Schistosoma mattheei** adult worms. The male is the larger thicker worm (left). The adult worms can clearly be seen in the mesenteric arteries (right)

The eggs, that contain a fully developed miracidium, work their way through the different layers of the blood vessel walls. In this they are assisted by proteolytic enzymes, secreted by the miracidium and the mechanical action of the spine during normal peristaltic movements of the bowel. The eggs cause the worst lesions and many are drained through the portal system to the liver and the lungs where they are trapped, causing a granulomatous hepatitis or pneumonia.

Adult worms are blood suckers and because the caecae end blind the worms periodically regurgitate the contents, that consists of digested and partly digested blood. The resulting haematin is phagocytosed by the macrophages and many of them end up in the lungs, imparting the characteristic grey colour the lungs and liver of sheep, but not of cattle.

The adult worms cause considerable damage to the blood vessel walls and this may interfere with the normal flow of blood. Dead and dying worms are removed from their sites by the mesenteric blood flow, which is connected to the portal system. The worms accumulate in the liver and this is either temporary or permanent, depending on the efficacy of the anthelmintic. This may lead to embolism with formation of infarcts and death.

Haematin accumulations in the lungs (left) and liver (right)
The cachectic carcass of a sheep showing the grey lung and liver (left) and severe thrombosis in the large intestine of a bovine (right)

Two syndromes, the intestinal and hepatic syndromes, are recognized in cattle. The intestinal syndrome is characterized by marked diarrhoea with continuous straining, sunken eyes, gnashing of teeth, bellowing and grunting during defaecation, marked mass loss, ataxia and anorexia. The hindquarters are soiled, melaena is often seen.

The hepatic syndrome is brought about when resistant animals are challenged with massive doses of cercariae. The resistance breaks down and animals are emaciated, have a wild expression in the eyes, knuckle over on the hind fetlocks, show ataxia, which is worse when turning on the hind limbs, hypermetria when running, disorientation, constipation and intermittent straining. The animal leans with its head against any convenient object and some eat their own faeces. The signs resemble *Matricaria nigaellaefolia* poisoning (Stootsiekte or pushing disease).

The presence of the characteristic eggs in the faeces or in squash preparations of blood and mucus from the rectum are a useful indicator in the early stages of the disease, but becomes less reliable as
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the egg production decreases with time. The autopsy with wide-spread granulomata and other lesions is pathognomonic.

Eggs in a squash smear of rectal blood (left) and granulomatous lesions in the bladder of a bovine

The nematode *Elaeophora sagitta* (=*Cordophilus sagittus*) occurs in aneurisms in the coronary vessels as well as in the small branches of the pulmonary artery especially in the diaphragmatic lobes. It is a rare infection of cattle and is usually only seen at autopsy or slaughter.

An opened aneurism in a coronary vessel (left) and the diaphragmatic lobe of the lung (right). The rather large *Elaeophora* is distinctly visible in both figures

Several kinds of microfilaria may also be present in a blood smear of especially cattle. These include *Setaria labiatopapillosa* and the *Onchocerca* species.
INFECTIONS OF THE SKIN

There are no helminth infections in the skin of sheep and goats. Although a number of nematodes and trematodes enter these hosts through the skin, the lesions are incidental and are limited to petechiae or allergic reactions, or both.

*Parafilaria bovicola* is an infection of major economic importance in cattle. The parasites occur in the subcutaneous tissues. The worms are 2-5 cm long, the males being the shorter ones.

When ready to lay eggs the female worms bore a small hole in the skin, most often on the withers and neck, through which the eggs that contain microfilariae are laid. The lesions bleed because of an anticoagulant secreted by the worm. The intermediate hosts are the face flies *Musca lusoria* and *Musca xanthomelas*. These flies feed on blood, ingesting the eggs at the same time and the infective L3 is transmitted to another animal when the flies feed again. Infection can also take place through the eye. The developmental period is anything from 195 to 245 days. The bleeding points, as they are known, are the only clinical signs and appear during warm hours of the day, their appearance being correlated with fly activity.

Seventy per cent of the subcutaneous lesions produced by the worms occur on the shoulder, withers and neck. These are usually superficial, slimy to the touch with a gelatinous consistency and are brownish to yellowish-green in colour. The brown colour is caused by haemorrhage and the yellowish-green by the accumulation of large numbers of eosinophils in the tissues. Early lesions resemble superficial bruising and as a result of trimming away the lesions, the carcass loses its value. Some cattle are so severely affected that the entire carcass is condemned. Lesions are most severe from August to January, and this coincides with the reproductive activity of the worms as well as the flies. Cattle do not appear to acquire any immunity and may be repeatedly re-infected.

The worms are common in the Bushveld of the Limpopo and Kwa Zulu Natal Provinces of South Africa, Zululand, Swaziland the northern and Eastern Cape, Botswana and Namibia. Optimum conditions for the occurrence of this parasite are areas with a mean annual rainfall of 400-700 mm, a frost period of less than 120 days and a mean annual temperature of 17-22 °C. These ecological requirements correspond to those of the fly intermediate hosts.
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The presence of the bleeding points on the specific areas is often sufficient for diagnostic purposes. Confirm by making smears of the blood from these areas, stain with Giemsa or DiffQuick and observe eggs and microfilaria.

Fly control is an important consideration, but should be done in conjunction with tick control. Use dipping compounds that are insecticidal in addition to acaricidal, in areas where Parafilaria occurs. Since this condition is more often seen in beef cattle, anthelmintic control will not be needed often, but when needed, use an anthelmintic that will eradicate Parafilaria as well, such as ivermectin at 0.2 mg/kg or doramectin and moxydectin. Note that the time needed for the complete resolution of the lesions is around 70 days; therefore animals should be dosed not later than 70 days before slaughter.
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Adult *Parafilaria bovicola* females in the subcutaneous tissues (top row); bleeding points on the withers and neck (second row, left), and flies congregating at a bleeding point (second row, right); carcass showing the *Parafilaria* lesions, also known as ‘false bruising’ (third row, left) and a close-up view of one of the lesions (third row, right); masses of eosinophils in the ‘false bruising’ lesion (bottom, left) and eggs with microfilaria in a blood smear made from a bleeding point (bottom right)

**INFECTIONS OF THE MUSCULATURE**

*Onchocerca* spp.

These parasites occur in the connective tissue of cattle. Their presence often results in the formation of hard nodules in which the nematodes are coiled up. The different species occur in different localities within the host, e.g. *Onchocerca guttferosa* is found in ligaments and *Onchocerca dukei* in the subcutaneous tissue. The parasites produce microfilariae which circulate in the blood. These are taken in by biting flies of the genera *Simulium* and *Culicoides* in which the infective stage develops and which transmit the worms to the next host.

An unidentified parasite, which could be a *Stephanofilaria* species has been found in cattle in the previous Venda, now in extreme north of the Limpopo province of South Africa. The lesions were associated with an intense *Haematobia meridialis* infestation and characterized by ulcerations behind the scapulae. The causative agent could, however, not be identified.
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Large *Onchocerca* sp. nodules clearly visible and indicated by arrows (left) and nodules in the muscle, also indicated by arrows (right)

**Taenia ovis**

The adult worms occur in dogs, jackal and hunting dogs and the larvae, *Cysticercus ovis*, in the muscles of sheep and goats. The larvae do not cause significant pathology but render the meat aesthetically objectionable and thus cause economical losses.

The metacestodes of *Taenia ovis* are indicated by the black arrows

**Taenia saginata**

The beef tapeworm uses cattle as intermediate host and humans as the final hosts. The metacestodes are known as cysticerci, and are not very numerous in cattle. One may find them in the active muscles, such as the diaphragm, and the iliopsoas and masseter muscles. They are particularly common in the cardiac muscles. The cysticerci are non-pathogenic and are incidental findings at necropsy or slaughter. It is, however, a condition of much economic importance, as many organs and carcasses are condemned because of the infection.
Numerous cysticerci in the heart (left) and a few in the muscles of a bovine (right)

INFECTIONS OF THE NERVOUS SYSTEM

The only parasite of the central nervous system is the metacestode of *Taenia multiceps* and the condition in the intermediate hosts, sheep, goats and rarely cattle, is known as coenurosis. The final hosts of these worms are dogs and wild carnivores and the intermediate hosts are sheep, goats and man. When the oncosphere hatches in the intermediate host, it bores through the wall of the intestines and is carried by the circulation to all parts of the body. Only those that settle in the spinal cord or brain will develop into the larval stage, *Coenurus cerebralis*. The coenuri grow slowly, taking months to mature. Their membranes are extremely thin and transparent so that the protoscoleces inside the coenurus can clearly be seen. They cause a syndrome known as gid or turning disease (draaisiekte, malkopsiekte) whose clinical manifestations depend on the location in the brain. Thus, circling, visual defects, peculiarities in the gait, hyperaesthesia or paraplegia may present. Coenuri may grow so large that they erode the surrounding bone of the cranium or the spinal column. The worms are rife in the Cape and south-western Orange Free State, but have not yet been recorded from KwaZulu-Natal Province and only at Potchefstroom and Delmas in the North-west Province of South Africa.
Coenurus cerebralis in situ in the brain of a sheep (top left); the coenuri removed from the brain (top right), coenurus with an extremely thin wall, showing the protoscoleces inside (middle left), a coenurus in the spinal cord (middle right), and the same removed from the vertebral canal (bottom left). The skull of a young goat that has been eroded by a coenurus (bottom right)

Larvae can be removed either surgically or by using praziquantel in the following manner:

Give praziquantel 100mg/kg once (or 50mg/kg twice). At the same time, give the normal dose of cortisone and aspirin 100 mg/kg twice a day. Continue the treatment with the anti-inflammatory drugs for 5 days. Thirty-six (36) hours after having given the praziquantel, start giving a diuretic. Depending on the reaction of the sheep, give at 12 or 24 hour intervals. Obviously, clean water should be available and the animal should not be stressed in any way.
REFERENCES