WORMS

By Jeff Fyffe BVSc, MRCVS, MACVSc

OSTERTAGIA

Know as

- Tissue worm
- Muscle worm

The most important parasite of the abomasum is a group of worms belonging to the Ostertagia genus.

Like most gastrointestinal worms, they require warm moist conditions for their development on the pasture. Heavy stocking rates or stock stressed by poor nutrition or overcrowding will be more susceptible to parasitism of all types.

Life Cycle

The Ostertagia have a direct life cycle, laying eggs in the gut that develop into infective larvae on the pasture.

After ingestion, the larvae may continue to develop into egg laying adults during spring or have arrested development in the wall of the abomasum during winter. These arrested larvae are referred to as Fourth Stage Ostertagia.

This explains why the faecal egg counts in winter and early spring are low.

Clinical Signs

The Ostertagia are not as severely pathogenic in deer as in sheep and cattle. However, heavy burdens will cause weight loss and generalized ill-thrift.

A particular syndrome is seen in Elk that is termed Fading Elk Syndrome.

Fading Elk Syndrome

Elk and their hybrids appear to be more susceptible to Ostertagia. In these animal the Ph of the abomasum is altered which affects the absorption of Copper. The combined effects of damage to the wall of the abomasum and the poor Copper absorption causes ill-thrift in Elk over winter.

Treatment

In all deer except Elk, routinely treat with Ivomec or Cydectin pre- and post-rut.
In Elk, in addition treat EVERY two weeks during winter, with a double dose of Cydectin plus a four times dose of Copper Bullets pre-rut.

LIVER FLUKE

In Australia liver fluke disease is caused by the liver parasite known as Fasciola hepatica. The disease may by acute or chronic and is best recognized in sheep and cattle. Various animals have different resistance levels to liver fluke which explains the pattern of the disease in different species (*Table 1 below*).

Distribution of Liver Fluke in Victoria

Liver fluke are prevalent in the North-East, Goulburn Valley and some areas in the North-West. They are also found in some regions of Gippsland and the Western District.

Life Cycle of Fasciola hepatica"

The parasite matures in the bile ducts of the host and their eggs pass down the bile ducts and are excreted in the faeces. The eggs hatch into miracidia which actively invade a particular type of snail known as Lymnaea tomentosa. Once in the snail they develop into sporocysts. The sporocysts then multiply within the snail to produce large numbers of cercariae.

Temperatures above 5^{o}C are necessary if the miracidia are to reach the snail. The best temperature range is 15 to 24^{o}C. Miracidia must enter a snail within 24 to 30 hours or they die.

Temperatures above 10^{o}C are necessary before the snails will breed or before the miracidia can develop in the snail.

Thus no development takes place during the winter and in spring the eggs that have accumulated over winter hatch.

Development ceases in the snail in winter but resumes as the spring and summer arrive in the snails that survive over winter.

The snails that act as the intermediate host for the liver fluke prefer low lying swampy areas with slowly moving water. Land with small streams or springs must also be considered dangerous. Irrigation land is the most dangerous.

The cercaria emerge from the snail 5 to 8 weeks later depending on the temperature. This pattern of emergence is fairly predictable for each area.

A separate cycle may occur in the autumn in some areas.

After emerging from the snail as cercaria, they encyst on herbage and wait to be eaten by the final host. The encysted cercaria are known as metacercaria and will be in largest numbers in late summer and autumn. Under severe winter conditions the cercaria do not survive.

Hay harvested in moist conditions and not properly dried may be infected for up to 8 months. Silage formation kills the metacercaria within 3 months.

Once eaten the cyst is destroyed and the metacercaria pass through the intestinal wall and migrate through the peritoneal cavity to the liver.

Initially the young fluke migrate randomly through the liver but eventually enter the bile ducts after about 4 to 5 weeks. They mature and begin to lay eggs about 10 to 12 weeks after infestation. Adult fluke can live for many years.

Pathogenesis

Acute fluke disease occurs 5 to 6 weeks after the ingestion of large numbers of metacercaria and is due to the sudden invasion of the liver by masses of young flukes. Sufficient liver tissue may be destroyed to cause acute liver failure together with haemorrhage into the abdominal cavity. Acute fluke disease is mostly seen in species that have low resistance to fluke.

Chronic fluke disease develops slowly and is due to the activity of the adult flukes in the bile ducts. The principal effects are blockage of the bile ducts, destruction of liver tissue, fibrosis (scarring) of the liver and anaemia. There is a loss of protein which is more pronounced when feed conditions are restricted. Chronic fluke will limit growth rate and feed conversion. Chronic fluke disease is seen in those species that have delayed resistance to fluke.

Clinical Findings

In acute fluke is a disease of animals with a low resistance to fluke. It is most frequently a disease of sheep and sudden death is the most common syndrome occurring in summer and autumn corresponding to the release of large numbers of cercaria in a particular area and for the opportunity for sheep to access these cercaria. Subacute fluke disease is also seen in sheep and results from the eating of large numbers of metacercaria over a longer period of time. The major symptoms are weight loss and anaemia.

With chronic fluke disease, small numbers of metacercaria are ingested over a long period of time by animals with a delayed resistance to the fluke. There will be weight loss, anaemia and chronic diarrhoea. This is the typical situation with cattle and deer. In addition chronic fluke disease allows the right conditions for the bacterium Clostridium novyi to cause black disease. This is one of the diseases that stock are vaccinated against when 5-in-1 vaccine is used.

Snail Control

Eradication of the host snails is the most effective method of full fluke control and eradication. This however is often very difficult in low lying, wet areas with a mild climate. Snails multiply extremely rapidly hence partial snail eradication will have limited benefit.

Snail eradication is almost impossible in irrigation areas such as the Goulburn Valley. There are available several different types of snail poison that are safe for stock. These need care and precision in their application and are a useful additional method of fluke control.

Treatment

The drugs to be used against fluke should ideally destroy the migrating immature fluke as well as the adults in the bile ducts. The most effective drug against Fasciola hepatica is Triclabendazole. This is known by the trade name “Fascinex”. It is effective against fluke as young as 1 day of age.

Drenching Program

In general treatment should be given towards the end of winter to remove egg laying adults from the liver and in those areas where metacercaria are likely to survive over winter, another treatment in the spring. Further treatments should be given in the autumn to prevent pasture contamination.

Drenching with “Fascinex” at 8 week intervals will reduce dramatically the liver damage and over a period of a few years, combined with snail control, will reduce the environmental levels of fluke. After one to three years, the frequency of drenching should be able to be reduced to three times a year at winter, autumn and spring.

Cost/Benefit of Drenching

Chronic liver fluke infection has been shown to reduce the growth rate of beef cattle. It is reasonable to assume that the same situation will occur in deer as well as damage to the liver. In Victoria it is not uncommon for up to 50% of deer livers from the Goulburn Valley to be condemned due to liver fluke damage. These same animals would most probably have grown out to better weights if the liver fluke had been controlled.

The dose rate of “Fascinex 120” is 10ml per 100Kg live weight. This is a cost of around 30 cents per 100Kg. On a yearly basis the cost is about $2.00 per 100Kg. A modest improvement of 10% in body weight as a result of drenching will easily cover costs even at the eight weekly drenching cycle. This is in addition to the salvaging of the liver as a valuable co-product.

Withholding Period

“Fascinex” has a 28 day withholding period for meat.

Some Hosts of the Liver Fluke and Their Resistance Status		
Early Resistance	Delayed Resistance	Low Resistance
Horse	Cattle	Sheep
Donkey	Buffalo	Goat
Pig	Man	Hamster
Wombat	Red Deer	Mouse
	Fallow Deer	Rat
	Rabbit	Possum
		Kangaroo

Table 1

ELAPHOSTRONGYLOSIS

(Exotic to Australia)

Causative agent

The parasite is a worm known as Elaphostrongylus cervi., however there may be at least two members of this family of parasite that affect different deer.

Known as

- Tissue worm
- Muscle worm

Distribution

North America (specifically in Caribou in Newfoundland), Scotland, Holland, Czechoslovakia, Norway, Sweden, western and central regions of the old Soviet Union and New Zealand.

The parasite was almost introduced into Australia. However, good quarantine regulations and protocol averted this.

Life Cycle

- Adult females in the host tissue lay their eggs directly into capillaries
- Eggs pass to the lungs where they hatch
- First-stage larvae pass to the respiratory tree
- Coughed up and swallowed
- Passed in faeces
- Enter the foot of snail or slug and develop to infective third stage larvae
- Snail must be eaten by the host
- Develop in the host to adults from fourth and fifth stage larvae
- Migration of the parasite occurs over the surface of the CNS to the final destination in the muscles of the front and hind limbs

Species Affected

Principally reindeer and caribou. A wide range of cervid hosts can be affected:

- Red Deer
- Wapiti
- Fallow
- Maral
- Sika
- Roe

Clinical Signs

- First-stage larvae will cause pneumonia (retardation of growth and exercise intolerance)
- Blindness
- Nervous symptoms
- Hind limb inco-ordination
- Cervical rigidity
- Ataxia
- Posterior paresis
- Heavy losses in Reindeer from ataxia and paresis
- Carcass condemnation in NZ from unsightly muscle lesions

Pathology

- Interstitial pneumonia without large numbers of adult worms
- Greenish discolouration of muscle tissue
- Parasites seen in or on CNS tissue

Diagnosis and Differential Diagnosis

- First stage larvae in faeces – often unreliable and repeated testing required
- Larvae MUST be distinguished from Dictyocaulus viviparus
- Copper deficiency
- Rabies
- Brain abscesses
- Tumours
- Chronic capture myopathy

Treatment

- All “white” anthelmintics and Ivermectin cause temporary suppression of larval output
- No effective treatment at this time

Significance

- In introduced into Australia it would have an adverse effect on venison production due to downgrading or condemnation of carcases
- Differential diagnosis is critical to protect Australia’s E cervi free status
- Differential diagnosis important in all disease states that cause neurological disorders in deer

PARELAPHOSTRONGYLOSIS

(exotic to Australia)

Causative Agent

The parasite is a worm known as Parelaphostrongylus tenuis. It is a NONPATHOGENIC parasite of White-Tailed Deer and found in the sub-dural spaces of this species. In other ungulates – severe clinical signs of CNS disease occur.

Known as:

- Moose disease
- Brain worm
- Meningeal worm

Species Affected

White Tailed Deer are resistant to clinical disease and act as reservoirs of infection to other deer:

- Moose
- Caribou
- Red Deer
- Wapiti
- Fallow Deer
- Mule Deer
- Goats – very sensitive
- Sheep
- Llama
- Antelopes
- Black-Tailed Deer
- Guinea Pigs

Cattle appear to be very resistant.

Clinical Signs

- Depends on species and number of larvae
- Goats and Fallow Deer die of acute peritonitis
- Red Deer and Wapiti show neurological signs including:
    - Listlessness
    - Tolerance to humans
    - Inco-ordination
    - Lameness
    - Circling
    - Blindness
    - Paralysis

Diagnosis and Differential Diagnosis

- First stage larvae in faeces – often difficult to find, unreliable test and repeated testing required
- Larvae MUST be distinguished from Dictyocaulus viviparus and E cervi
- Copper deficiency
- Rabies
- Brain abscesses
- Tumours
- Chronic capture myopathy

Life Cycle

Basically very similar to E cervi.

Treatment

- No anthelmintic effective against adult worms
- Ivermectin effective against very early stages of infection by third stage larvae
- Larvae appear in faeces 28 days later

Significance

- Devastating potential where White-Tailed Deer are in association with Red Deer and Wapiti farms
- Control measures costly – mollusc and White-Tailed Deer exclusion
- Another parasite Australia does not want
- A major control problem in North America
