

Nematode parasites of sheep in the UK

There are about 20 different species of nematodes of sheep commonly found in Britain, the more important of which are shown in Table 19. Pathogenicity varies with genera and species, the numbers of nematodes present as well as host factors such as age (maturity), nutritional status and body condition. The tapeworm, *Moniezia expansa,* and other tapeworms for which the sheep acts as an intermediate host, are also covered at the end of this chapter. Liver fluke *Fasciola hepatica* (a trematode) is discussed in <u>Chapter 4. Anthelmintics, 4.3 Liver Fluke.</u>

Table 19. Nematode parasites of sheep

Site	Species	Features	Pathogenicity (High/Med/Low)
Abomasum	Teladorsagia (Ostertagia) circumcincta	'Small brown stomach worm' 0.8 – 1.5 cm	н
	Haemonchus contortus	'Barber's Pole worm' 1.5 – 3.0 cm long and stout. Very obvious to the naked eye.	Н
	Trichostrongylus axei	'Stomach hair worm' 0.3 – 0.6 cm	М
Small intestine	Trichostrongylus colubriformis Trichostrongylus vitrinus	'Black scour worm' 0.4 – 0.9 cm	M M
	Nematodirus battus Nematodirus filicollis Nematodirus spathiger	'Thin-necked intestinal worm'1.0 – 2.3 cm	H L M
	Cooperia curticei	'Small intestinal worm'0.5 – 0.8 cm	L
	Bunostomum trigononcephalum	'Hookworm'1.2 – 2.6 cm	Μ
	Strongyloides papillosus	'Threadworm' 0.4 – 0.6 cm	L
	Capillaria longipes	'Hairworm' 0.1-0.2 cm	L
Large intestine	Oesophagostomum venulosum	'Large bowel worm' 1.0 – 2.4 cm	L
	Trichuris ovis	'Whipworm' 4 – 8 cm	L
	Chabertia ovina	'Large-mouthed bowel worm' 1.4 – 2.0 cm	L
Lungs	Dictyocaulus filaria	'Large lungworm'. Live in bronchi, 3 – 10 cm	Μ
	Protostrongylus rufescens	Live in the small bronchioles. $1.6 - 4.0$ cm	L
	Cystocaulus ocreatus	Live in the small bronchioles. 4.0 – 9.0 cm	L
	Neostrongylus linearis	Live in the small bronchioles.	L
	Muellerius capillaris	0.5 – 1.5 cm 'Small lungworm'. Form nodules in lung parenchyma. 1.2 – 2.2 cm	L



The typical life cycle

The life cycles of the gastrointestinal nematodes are all very similar, with one or two minor exceptions, and the following description applies particularly to *Teladorsagia*, *Trichostrongylus* and *Haemonchus*.

Figure 13. Worm-life cycle.



There is no multiplication within the sheep and the life-cycle is direct i.e. no intermediate host. Adult female worms in the sheep lay eggs that pass out in the faeces and hatch; each egg releasing one first-stage larva (L1). The L1 develop and moult to second stage larvae (L2). The L1 and L2 are active and feed on bacteria in the faeces. At the second moult to the third stage larvae (L3), the cuticle of the L2 remains as a sheath, protecting the L3 but also preventing them from feeding. The L3 is the infective stage. L3 migrate on to the herbage where they are ingested by sheep. In the walls of the stomach or intestines they develop into fourth stage larvae (L4), before emerging as adult worms about 14 days later. The prepatent period (between ingestion of L3 and the appearance of eggs in the faeces) is generally between 16–21 days. Adult worms that are not expelled from the sheep by immune mechanisms or killed by anthelmintics survive for only a matter of weeks (typically less than 12) before dying naturally.

Strongyloides papillosus

The L3 has no protective sheath. L3 can infect the host by ingestion or by skin penetration. Transmission may also occur to lambs via the milk of the ewe. The prepatent period is about 9 days.

Bunostomum trigonocephalum

Infection of the host occurs by ingestion or through the skin. Following skin penetration the larvae pass to the lungs and then to the small intestine. The prepatent period is about 56 days.

Trichuris ovis

Infection of the host occurs through ingestion of the L1 in the egg. After ingestion the plugs at the ends of the egg are digested and the L1 released. All four moults occur within the sheep. The prepatent period is 1 to 3 months.



Epidemiology

Two terms are used to describe the conditions of pastures containing the free-living nematode stages. Pastures are 'contaminated' if there are eggs and larvae present, but pastures are only 'infective' if there are L3 present and climatic conditions are suitable for them to move up onto the herbage, where they can be ingested. Both rainfall and temperature influence the infectivity of pastures. The rate of development to the infective stage (L3) is dependent on temperature. Rain tends to increase the infectivity of pastures by assisting in the movement of L3 out of faecal pellets or pats and by providing the film of moisture necessary for L3 to migrate onto herbage. Rainfall records have been used to predict the peak of availability of nematode larvae on pasture, and temperature records are used to predict the risk of nematodirosis in lambs.

Development of L3 from eggs deposited in early spring may take 10–12 weeks but eggs deposited later in the season develop faster. Summer-deposited eggs can give rise to L3 in just 1–2 weeks. Consequently, eggs passed on to pasture in spring and early summer tend to reach the infective stage at about the same time, resulting in high levels of pasture infectivity from mid-summer onwards (see figure 14).



Figure 14. The epidemiology of nematode parasitism in sheep at pasture

L3 are most active during warm weather and, if they are not ingested, consume their energy stores and suffer high mortality rates. In autumn and winter, L3 can survive longer and some will over-winter on pasture (with our warmer, wetter winters this source of infection the following spring is becoming more significant). Some worm species are better at winter survival than others – *Haemonchus* larvae do not survive well in freezing temperatures but *Nematodirus* eggs can survive prolonged cold temperatures. Over-wintering L3 provide a source of infection to grazing sheep in late winter and early spring but do not survive long on pasture after ambient temperatures rise. Pasture infectivity tends to decline rapidly to low levels in late April or early May.

If spring-lambed ewes are placed on the pasture, contamination with worm eggs occurs first from the ewes themselves (early spring), and then later (late spring and summer) from the lambs as well. In the case of the ewes, the worms producing these eggs have survived over winter in the ewes or have developed from the ingestion of over-wintered L3 in early spring. In the case of the lambs, the worms have arisen from the ingestion of over-wintered L3 then, later in the season, from eggs deposited by the ewes. The source of the high infectivity of pastures in late summer and autumn is the deposition of eggs in spring and early summer.

This typical pattern is seen most clearly in the epidemiology of *Teladorsagia* and *Trichostrongylus*. The rise in pasture larval availability in early summer tends to be dominated by *Teladorsagia*, with *Trichostrongylus* spp contributing increasingly in late summer and autumn.



Haemonchus contortus has a very high biotic potential with each female worm capable of producing up to 10,000 eggs per day. Warm and wet conditions favour the rapid development of eggs to L3 and pastures can become highly infective <u>very quickly</u> almost any time between mid-spring and late autumn. This means that pastures can change from low *Haemonchus* infectivity in early spring to very high infectivity in summer and autumn, causing serious, unexpected outbreaks of Heamonchosis in lambs and ewes.

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Hypobiosis (arrested development)

The abomasal nematodes *Teladorsagia* spp, *H. contortus* and *T. axei* are capable of interrupting their development at the L4 stage and persisting for long periods in a state of dormancy or hypobiosis. They then resume their development and become normal, egg-laying adults. This interruption of development occurs principally to larvae ingested in the late autumn and winter. It can be considered as an evolutionary adaptation which delays egg production (and death) until the following spring when eggs deposited on pasture have a higher chance of continuing the worm's life-cycle. In the case of ewes, most of the *Teladorsagia* population in the host between November and February exists as hypobiotic larvae. Between April and September, there are very few, if any, hypobiotic larvae and most parasites exist as adult or actively developing forms.

Hypobiosis is important in sheep for three reasons:

- When hypobiotic larvae of *Teladorsagia* resume their development, they can be responsible for clinical disease in yearling sheep, similar to type II ostertagiosis or 'winter scours' seen in cattle. (See also Section 4.4)
- The worms developing from hypobiotic larvae in ewes are an important source of pasture contamination in the spring and early summer.
- Hypobiosis is the principle way *H. contortus* survives the winter in the UK. This also has implications for the selection for AR since it means that the proportion of the Haemonchus worm population 'in refugia' in spring is likely to be very low. Any anthelmintic treatment of ewes is therefore highly selective for AR.

The small intestinal parasites, including *Nematodirus* spp, *Trichostrongylus* spp and *Cooperia* spp, are also capable of hypobiosis but this does not appear to be an important feature of their epidemiology.

Nematodirus battus

For all *Nematodirus* spp, development to the L3 takes place within the egg. With *N* battus, hatching and release of the L3 occurs as a result of climatic stimulus, usually a period of chill followed by a mean day/night temperature of more than 10°C, but below 17°C. The prepatent period can be as short as 14 days.

Nematodirus battus has a much slower life-cycle (figure 15), with infection passed from a lamb crop in one year to the lambs born in the following year. The long survival of *Nematodirus* eggs permits this relatively long generation interval. As a result of the specific climatic requirements for egg hatching, large numbers of infective larvae can appear on pasture almost synchronously. This usually occurs between April and June each year but can occur at other times of the year. When mass hatching coincides with the presence of 6-12week old lambs, severe outbreaks of nematodirosis can occur (see also Section 3.5). A smaller peak of infections is also known to occur in the autumn time. It has been found experimentally that some populations of N. battus can hatch without prior chilling.



Figure 15. Nematodirus life-cycle



Reference

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