Lecture 17: Sheep health: Internal parasites

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Learning Objectives

On completion of this topic you should be able to:

- Describe the prevalence and importance of gastro-intestinal parasitic disease of sheep;
- Understand the effects of gastrointestinal nematode and liver fluke infection on sheep and wool production;
- Understand the principles of control of gastro-intestinal nematode and liver fluke infection.

Key terms and concepts

Epidemiology, pathogenesis, resistance, resilience, life cycles, risk factors, preventive, strategic, tactical and curative disease control, integrated parasite management

Introduction to the topic

The major disease problems of the Australian sheep industry are parasitic in nature (See Lecture 17, Table 17.1). This is typical of grazing industries where parasites that spend part of their lifecycle in the environment prove difficult to control, particularly during the environmental phases of the lifecycle.

17.1 Classification of the major gastro-intestinal parasites of sheep

Of the internal parasites of sheep the vast majority inhabit the gastro-intestinal tract. **Lungworm** infection can be important locally but is not a major disease under Australian conditions and will not be considered further. **Nasal bot** infection with the larvae of the fly *Oestrus ovis* is widespread and discomfiting but appears not to have major effects on productivity. Apart from **coccidiosis**, a disease caused by a protozoan parasite, all of the major gastro-intestinal parasites of sheep are **helminths** ("worms") and the rest of this topic will focus on these.

Gastro-intestinal **helminthiasis** (also called helminthosis) is the single biggest disease problem of sheep in Australia. Of the helminths the **nematodes** (roundworms) are most widely distributed and by far the most important of the helminth parasites (Figure 17.1). **Liver fluke** is also an important parasite, but has a much more restricted distribution. Tapeworms are common and spectacular for their size, but appear to have few adverse effects on productivity.

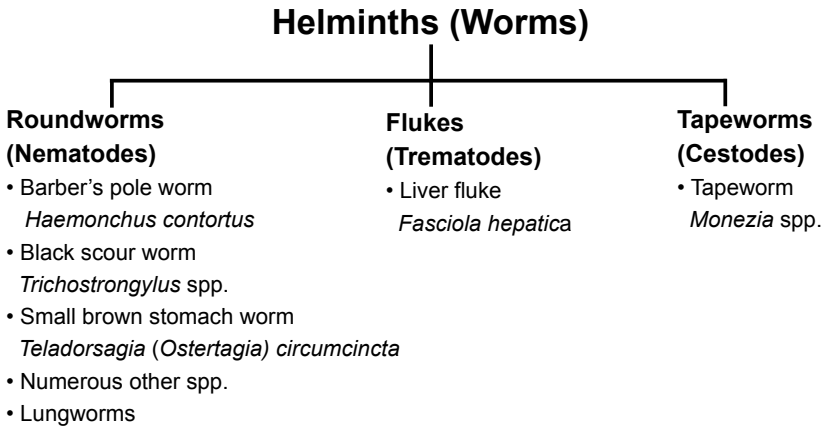

Figure 17.1: Broad classification of the major helminth parasites of sheep (Walkden-Brown and Besier 2006).

17.2 Gastro-intestinal nematode infection

This is the major disease problem of the sheep industry in all but the low rainfall pastoral areas of Australia. It is a chronic disease, with loss of animal production being a major feature. However, infection can be acute and also fatal, particularly in the case of Barber's pole worm.

Species and life cycle

Table 17.1 lists the common species in Australian sheep. The three most important species in Australia are:

Haemonchus contortus (Barber's Pole worm). The mature worm lives in the abomasum, attaching to the stomach lining and feeding by sucking blood. It thrives in warmer, wetter regions and seasons. This is the major nematode in summer rainfall areas of Australia.

Trichostrongylus *spp*. (Black Scour worm). The mature worm lives in association with the epithelium of the first 3-4 metres of small intestine, probably living on gut cells and fluids. Several species occur depending on climate, so it is the most widespread of the worms. These worms can handle cooler, dryer conditions better than *Haemonchus* and are the most universal of the common nematodes. Different species affect different climatic regions. *T. colubriformis* is the main species in summer rainfall regions while *T. virtinus* is the main species in winter rainfall areas.

Teladorsagia (or Ostertagia) circumcincta. (Small Brown Stomach worm). The mature worm lives deep in the crypts of the abomasum. This is a cold tolerant parasite of cool/cold wet regions. It is a major sheep nematode in winter rainfall areas.

The **life cycle** is very similar for these species and involves phases in the host animal (parasitic) and in the environment (free living) (Figure 17.2). This poses a major challenge in control, because while it is relatively easy to target the parasite while it is in the host, the environmental stages of the life cycle are much more difficult to attack. The life cycle is direct, with no intermediate hosts. Sheep become infected with nematodes when feeding on contaminated pastures, consuming infective larvae while grazing. The larvae pass into the gastrointestinal tract where they develop into mature adults. These adults mate and the females lay eggs which pass out in the faeces of the host. Once the eggs hatch, the larvae undergo three stages of development within the faecal pellet (without multiplication), with the L3 stage being the infective stage. The L1 and L2 larvae live on bacteria and fluids in the faeces but the infective L3 larvae are encased in a protective sheath and cannot feed until ingested by a host. L3 migrate from the faeces onto the pasture and into the soil if there is sufficient moisture and are ingested by sheep from the pasture. Large numbers of eggs, L1 and L2 larvae perish from desiccation and low oxygen tension and typically only 0-5% of eggs develop into infective L3 larvae. Once ingested, L3's undergo exsheathment and develop into adults over about a 16 day period.

Table 17.1: Common gastrointestinal nematodes of sheep in Australia and their prevalence in the different rainfall zones (Walkden-Brown and Besier 2006).

Specific name	Common name	Location	Egg production*	Rainfall zone	
				Summer	Winter
Haemonchus contortus	Barber's Pole worm	Abomasum	5,000-10,000	+++	+
Trichostrongylus spp.	Black Scour worm	Small intestine	100-200	+++	+++
Ostertagia circumcincta	Small Brown Stomach worm	Abomasum	100-200	++	+++
Nematodirus spp.	Thin Necked Intestinal worm	Small intestine	50	++	++
Oesophagostomum spp.	Large Bowel worm/Nodule worm	Large intestine	5,000-12,000	++	++
Chabertia spp.	Large Mouthed Bowel worm	Large intestine	?	+	+++
Trichuris spp.	Whip worm	Large intestine	?	+	+

*Daily egg production by a single adult female worm

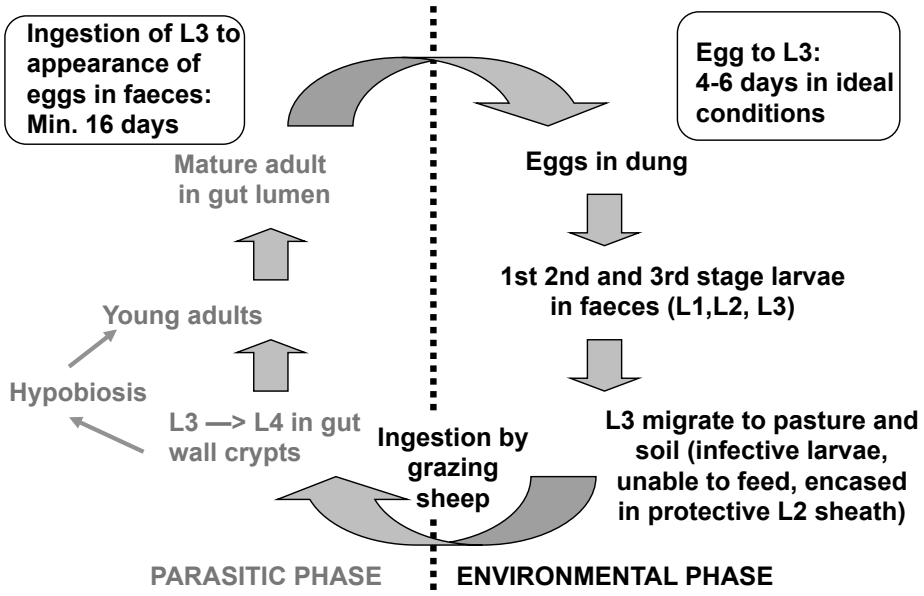

Figure 17.2: Lifecycle of typical gastro-intestinal nematode parasite of sheep. This lifecycle is common to all of the major genera such as Haemonchus, Trichostrongylus and Ostertagia (Walkden-Brown and Besier 2006).

In *Nematodirus*, the L1 to L3 development occurs within the egg making it particularly resistant to adverse environmental conditions. In *Trichuris*, the eggs hatch once they are eaten by the host. The duration of the lifecycle of sheep nematodes varies widely, depending on environmental conditions and worm species. But under optimum conditions it can be as short as 3 weeks from egg to egg (16 days in the host and 5 days in the environment). When environmental conditions are unfavourable some L4 larvae can enter into **hypobiosis** (arrested development) in the wall of the gut and remain in this state, only resuming development when conditions improve. The occurrence of hypobiosis appears to vary considerably between species, environments and seasons, and the practical importance to worm life cycles in Australia is poorly understood

17.3 Factors affecting distribution and prevalence (Epidemiology)

Environmental factors

These largely determine the distribution of parasites as shown in Table 17.1 and their prevalence on pasture during the year as shown in Figure 17.3.

- **Temperature.** An air temperature of >10°C is required for larval development with some development at lower temperatures for cold tolerant species and more heat required by *Haemonchus* (O'Connor *et al.*, 2006). In Armidale, there is virtually no development of *H. contortus* or *T. colubriformis* over the winter months due to cold inhibition (Bailey *et al.*, 2008a). Rate of development and the speed of the lifecycle increases with increasing temperature. Very high temperatures are lethal, but the effects of high temperatures are mainly mediated by increased desiccation. L3 are the most resistant stage of the lifecyle and they can desiccate and stand extremes of heat and cold. Survival of L3 is reduced with increasing temperatures and moisture, as these facilitate using up the metabolic reserves of the L3. In contrast to the lack of
- **Moisture.** Desiccation is the major cause of losses in the environment. Eggs, L1 and L2 are most affected. Approximately 50mm soaking rain/month with favourable temperatures is required to maintain development. Under hot summer conditions up to 125mm/month may be required. In dry weather few if any L3 escape faecal pellets. *Haemonchus* generally requires significant rain within 4 days of faecal deposition for development and release of L3 to occur.

The most favourable combinations of temperature and moisture are often found in during spring and autumn although summer rainfall areas have a major summer problem with *Haemonchus* (Figure 17.3).

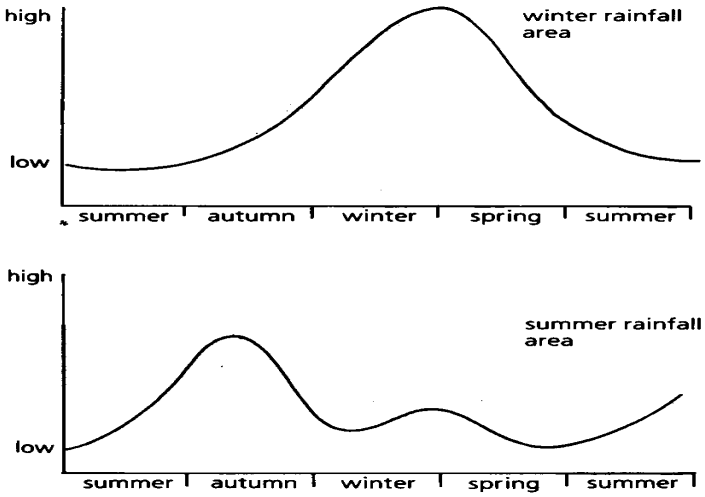

Figure 17.3: Typical larval distribution patterns on Australian sheep pastures in the winter and summer rainfall zones (Brightling 1994).

Parasite factors

- **Egg production.** This varies greatly between species (Table 17.1) and to some extent is largely a function of adult parasite size. High levels of egg production allow rapid build up in parasite numbers and explosive outbreaks of disease to occur (eg. *Haemonchus*)
- **Pathogenicity** of adult worms. *Haemonchus* adults are approximately 6-8 times more pathogenic than *Ostertagia*, *Nematodirus* and *Trichostrongylus* adults and L4 larvae
- **Resistance to cold and desiccation**. *Haemonchus* eggs and larvae are most susceptible to desiccation, *Trichostrongylus* and *Nematodirus* most resistant. Similarly, *Haemonchus* and *Oesophagostomum columbianum* eggs and larvae are most susceptible to cold, with *Ostertagia* and some *Trichostrongylus* species most resistant
- **Anthelmintic resistance**. This is a very serious and growing problem! It widespread for the levamisole ("clear drench") and benzimidazole ("white drench") groups and closantel, and is increasing rapidly with the macrocyclic lactones ("oily drench" or "mectins").

Host factors

- **Immunity**. Sheep develop acquired or "age immunity" as they grow. Young sheep in the first 18 months of life are the most susceptible to infection. Immunity is influenced by:
    - **Worm species**. *Haemonchus* induces less immunity than the other species and remains a threat to adult sheep
    - **Degree of challenge**. Immunity requires exposure to infection
    - **Host genotype.** There is within and between breed variation in resistance to helminth infection. The heritability of faecal worm egg count is approximately 0.25
    - **Physiological state**. Immunity is almost totally lost from around the time of lambing to about 2 months into lactation. This leads to a "peri parturient rise" in faecal egg counts.
- **Host nutrition.** This affects both resistance to infection (numbers of worms in the host) and resilience (ability to continue to produce for a given number of worms)
- **Stocking rate/grazing management**. This has profound effects on the level of pasture contamination with eggs and infective L3 larvae (see section on control).

17.4 Clinical signs

The major clinical signs of nematode infection in sheep are: **ill-thrift** in young stock, **anaemia** and **scouring (diarrhoea).** Table 17.2 shows the signs associated with the major worms in Australia.

- *Haemonchus contortus*: the main sign is anaemia (paleness), weakness (collapsing when driven) and often, deaths. Ill-thrift occurs in chronic infections, though burdens frequently increase rapidly from an insignificant level to a point causing deaths with little warning, before ill-thrift is seen.

Severe infection with *Haemonchus* can result in the accumulation of fluid under the lower jaw, known as "bottle jaw", due to the loss of protein from plasma. Scouring is not a feature of the disease **haemonchosis**.

- Scour worms: this includes *Trichostrongylus*, *Ostertagia*, *Nematodirus*, *Chabertia* and *Oesophagostomum* spp, plus some minor species. The major sign of large burdens is scouring and dags, but the more common sign is ill-thrift and weight loss due to smaller burdens. However, both scouring and ill-thrift can also be caused by other factors other than worm burdens and for this reason, are poor indicators of infection. Also, by the time these clinical symptoms are evident, substantial sub-clinical losses will already have occurred. Infection with *Trichostrongylus spp*. is often (but not always) associated with dark green to black scours. This provides a suitable environment for the development of larvae of the sheep blowfly (i.e. breech strike).

In most cases, sheep are infected with a number of worm species at the same time, and signs such as scouring are usually due to a mixed burden of *Trichostrongylus*, *Ostertagia* and others.

Table 17.2: Main clinical signs and severity associated with different worm species (Walkden-Brown and Besier 2006).

Genus	Anaemia	Scouring	Ill thrift
Haemonchus	+++	-	+++ [b]
Trichostrongylus	-	+++	+++
Ostertagia	-	+++	+++
Nematodirus	-	++	++
Oesphogostomum	-	+ [a]	+++
Chabertia	-	++ [a]	++
Trichuris	-	++ [a]	+++

[a] Faeces may contain mucus or blood
[b] Can kill adult sheep

17.5 Pathogenesis (pathophysiology) of infection

The mechanisms by which nematode infection brings about the observed loss of production and clinical signs are as follows:

Reduced feed intake

This is one of the most important pathophysiological consequences of worm infection. In the case of *Ostertagia*, reduced feed intake accounts for 60-70% of the effect of the parasite on the host although the causal mechanism has not been identified. Increasing the level of infection with *Trichostrongylus colubriformis* induces reductions in feed intake, especially in young sheep (Figure 17.4). However, note that as the infection progresses, the animals develop immunity and feed intake can return to normal values. Pair feeding studies indicate other factors apart from reduced feed intake are involved, including altered protein metabolism.

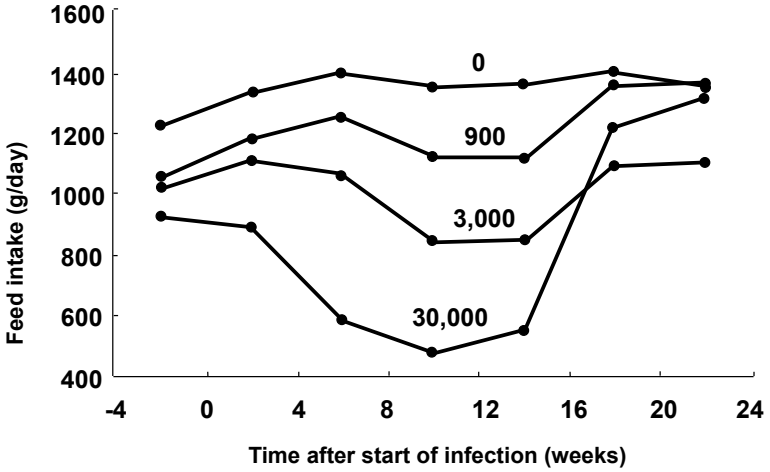

Figure 17.4: Mean daily feed intake for groups of 4 lambs continuously infected with 0, 900, 3,000 or 30,000 infective larvae of Trichostrongylus colubriformis per week (Steel and Symons 1979).

Altered protein metabolism

This is also very important and results in reduced nitrogen retention as a consequence of:

- Loss of plasma and erythrocytes leading to hypoproteinaemia and anaemia. Losses may be due to direct blood sucking (eg. *Haemonchus*, or as a result of inflammation of the gut (other species). Much of these losses is re-digested and absorbed but this is an energy inefficient process
- Increased production and loss of mucus and epithelial cells. These are high in cysteine and methionine (important for wool production) and are poorly re-absorbed.

Maldigestion and malabsorption

These do not appear to be very important due to the capacity of the gut to compensate for damaged sites by using uninfected regions.

Hypersensitivity to worm larvae

Although scouring in sheep with a poor immunity to worms (eg, lambs, ewes during the lambing period, sheep under nutritional stress) is associated with heavy burdens of adult worms, a different situation applies in sheep which have developed a good immunity but which have not been exposed to worm larvae for a long period. Adult sheep in winter rainfall regions often scour profusely when they first meet large numbers of worm larvae (usually in winter), as a result of an excessive immune reaction aimed at expelling the larvae. Although these larvae do not establish as adult worms (and hence worm egg counts are zero or very low), the inflammation to the gut causes fluid loss, seen as scouring. This situation has been recognised relatively recently (Larsen *et al.* 1994) and is not widely appreciated by sheep producers, who often attribute scouring to particular pasture plants. However, it is now believed to be responsible for most of the scouring where a large proportion (can be over 50%) of otherwise healthy adult sheep are affected. It typically lasts for a couple of weeks in individuals but it may be 6 weeks before all sheep have ceased scouring, and they then maintain a solid immunity while larval infection continues. Often, only some of several adult sheep flocks on a farm are affected in any one year. No treatment is yet known, as drenches have minimal effect (worm egg counts are very low), although slow release capsules protect against it. Further research is needed to clarify the major causal factors and possible control measures.

17.6 Effects on wool production

Research over the years has shown that, depending on severity, worm infection has the following overall effects:

- **Wool production** - 10-30% reduction in annual wool production in young stock depending on level of infection. Reduction of 5-20% in mature sheep. Short term reductions can be up to 70%
- **Yield** - Unaffected
- **Mean fibre diameter** - Reduced by 0.5-2µm
- **Staple length** - Reduced
- **Staple strength** - Unchanged or reduced
- **Processing losses -** Increased losses during carding and combing.

Figures 17.5 to 17.7 below show the effects on productivity of four parasite control treatments at two locations, Armidale (summer rainfall) and Hamilton, Victoria (Winter rainfall). The experiment was conducted with 100 weaner wethers per treatment set-stocked at 12.4 per hectare at Armidale and 16 per hectare at Hamilton. The treatments were:

- **Salvage -** (Most wormy). Treat individual animals only to prevent death (58+10 = 68 individual animal treatments)
- **Curative -** Treat animals when clinical signs present (4 thiabendazole + 1 rafoxanide = 5 drenches)
- **Preventive** - Traditional practice, include rafoxanide (5+5 = 10 drenches)
- **Suppressive -** (Least wormy). Monthly thiabendazole plus rafoxanide in Armidale November-April (11 + 5=16 drenches).

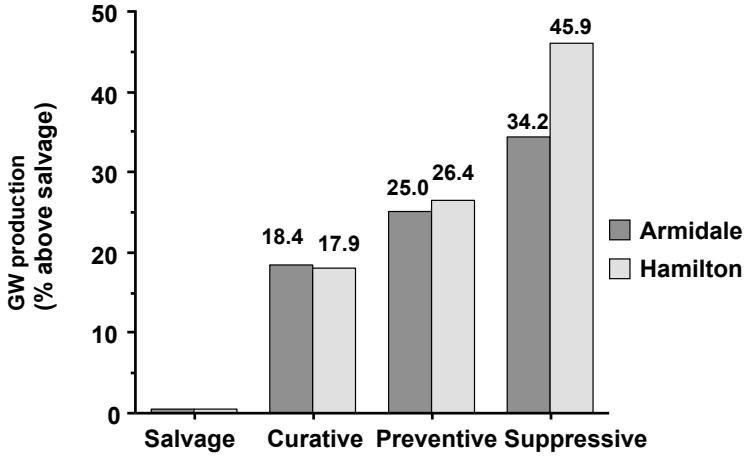

Figure 17.5: Effect of different worm control programs from 5-7 months of age on fleece weight at first shearing in Merino wethers (Johnstone *et al.* 1979).

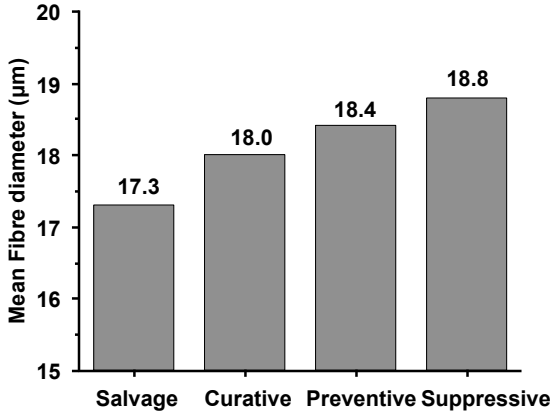

Figure 17.6: Effect of different worm control programs from 5-7 months of age on mean fibre diameter at first shearing in Merino wethers (Johnstone *et al.* 1979).

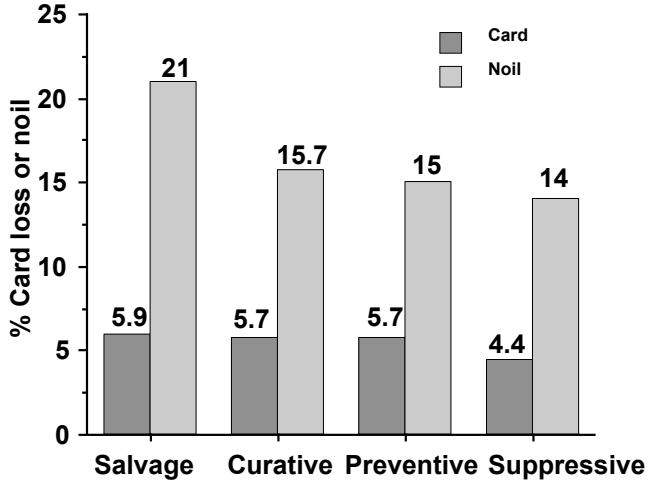

Figure 17.7: Effect of different worm control programs from 5-7 months of age in Merino wethers on some processing characteristics of the first fleece (Lipson and Bacon-Hall 1979).

17.7 Diagnosis

A provisional diagnosis may be made on the basis of clinical signs (ill thrift, scours, anaemia) and history of treatment and climatic conditions. However the diagnosis is confirmed with 2 following steps.

- **Worm egg count** (WEC). This is the number of nematode eggs per gram of faeces. However, the three major species (*Haemonchus*, *Trichostrongylus* and *Teladorsagia*) and others all produce eggs of similar size and shape, making it difficult to identify the prevalent species
- A **larval culture** (hatching the eggs) **and differentiation** (identification of species on the basis of the L3 morphology) is required to correctly identify the species present and enable sound interpretation of WEC as the species differ in egg production. *Trichostrongylus* and *Ostertagia* produce similar numbers of eggs, such that where they are the major species present, WEC provides a useful guide to total number of worms present. But when *Haemonchus* is present, WEC can be extremely high even though only a small number of adult worms are present. This species produces up to 50 times more eggs per adult female than the other two. WEC in excess of 3000 eggs per gram is indicative of infection with *Haemonchus*.

Post-mortem examination of the gastrointestinal tract enables species identification and **total worm counts**. This allows both immature and mature worms to be counted and provides an accurate measure of worm burden. However, this method is laborious and not suitable for routine farm monitoring for worm control.

- *Haemonchus* (Barber's Pole Worm) is up to 40 mm long and 4 mm wide. They are visible in the abomasum during post-mortem inspection. The worm is characterised by the red (blood-filled) digestive tract spiralling along white egg-filled oviducts (in the case of females) giving rise to the parasite's common name
- *Trichostrongylus* (Black Scour Worm) is light brown in colour, up to 8 mm long and 1 mm wide. It is a so called "hair worm" and is almost impossible to see while in the intestine. It must washed from gut contents and stained appropriately to be identified
- *Ostertagia* (Small Brown Stomach Worm) is light brown in colour, up to 10 mm long and 1 mm wide. As it is another hair worm it is hard to see without proper processing of gut contents.

17.8 Control

Control has relied traditionally on **chemical (anthelmintic) treatment** supplemented with basic **grazing management,** but has now expanded to include a range of strategies such as **breeding for worm resistance** and more advanced grazing strategies. These methods can be combined into **integrated parasite management (IPM)** packages that maintain good worm control but limit the development of anthelmintic resistance (Eg. Kelly et al, 2010).

Anthelmintics represent the major strategy employed in controlling nematode infection. A range of anthelmintics exist on the market, including both narrow- (species specific) and broad- (control a number of species) spectrum drenches. The broad-spectrum drenches are normally effective against *Haemonchus, Trichostrongylus, Ostertagia* and *Nematodirus*, and include white drenches (benzimidazole group), clear drenches (levamisole group), oily drenches or "mectins" (macrocyclic lactones) and the newer Amino-acetonitrile derivative (AAD) group (monepantel) and Derquantal (in combination with abamectin). Anthelmintics may be administered orally via drenching, by injection, or intra-ruminally via sustained release devices.

There are four broad approaches to the use of anthelmintics.

- The **curative** approach involves treatment only when clinical signs are apparent. It is not economical to use this approach due to the large losses in production that can be incurred
- The **tactical** approach bases the decision to treat on weather conditions favourable for the parasite or if monitoring of WEC indicates that a drench should be given
- The **strategic** approach entails giving a fixed number of treatments at specified times to delay the seasonal build up in numbers. It is based on detailed epidemiological understanding of the disease in a given area. Examples include the WormKill, DrenchPlan, WormBuster programs in different regions. These have fallen out of favour as they appear to select quite strongly for anthelmintic resistance.
- The **suppressive** approach uses anthelmintics in a blanket fashion in an attempt to suppress the disease. This was used widely in the 1960s and 1970s following the introduction of highly effective broad-spectrum anthelmintics but it is expensive and results in rapid selection for anthelmintic resistance and so has not been recommended for many years.

Anthelmintic resistance is a major problem for the industry with resistance by worms to drenches being very widespread in Australia, affecting all major worm types (Besier and Love 2003). Failure to kill sufficient worms is a significant sub-clinical problem as worms continue to reduce growth and

production. In severe cases, sheep may die from haemonchosis because ineffective products were used. With only a small number of anthelmintic groups available, it is essential that sustainable approaches are used to manage resistance.

The main strategies recommended for resistance management are:

- minimise drenching by using strategic programs, and monitor worm egg counts so unnecessary drenches are not given
- never under dose – drench to the weight of the heaviest animal in the flock
- test drenches for effectiveness by a faecal egg count reduction test so appropriate products are used
- ensure that drenching program do not cause increases in resistance because they constrict the worm population so future generations are derived chiefly from resistant worms.

The latter recommendation is relatively recent, as it is recognised that resistance will increase if sheep do not pick up enough worms after drenching to dilute any resistant worms which survive the drench. Where worm larvae are present in large numbers on pasture, which is typical on green pastures, there may be no need for specific measures to ensure this. Where there is little survival of worm larvae on pasture, so sheep will not pick up worm larvae after a drench (for example, when sheep are on dry pastures), it may be necessary to deliberately avoid drenching some sheep or flocks so some worms survive and eventually lead to enough pasture contamination with larvae that resistant worms will be diluted. All refugia-based strategies carry the risk that too many worms may be left in the system - in sheep or on pastures, so it is essential to monitor at intervals with worm egg counts."

Grazing management is largely based upon placing the most susceptible stock on the pastures least contaminated with worm larvae. Typically this involves putting lambing ewes and freshly weaned lambs onto “clean” pastures. Producing relatively clean pastures is easier said than done however. Methods include:

- Use of sown pastures or crop residues
- Spelling of pastures to allow L3 to die. The spelling of pastures to reduce the population of infective larvae has been demonstrated to be highly effective in tropical regions as larvae deplete their energy reserves much more quickly (5-9 weeks) in these conditions. Remember that L3 cannot feed due to their protective sheath so will always eventually die if not ingested. In cooler temperate regions, rotational grazing has been historically been less successful as spelling times to reduce L3 have had to be longer than is optimal for pasture management. However it has recently been demonstrated that short duration long interval rotational grazing (“**cell grazing**”) is highly effective at reducing infections, particularly with *H. contortus* (Colvin *et al.*, 2008; 2012*)*. These systems involve grazing paddocks heavily for short periods (1-4 days) with long rest periods (60-120 days) between grazings.
- Prior grazing by resistant animals. This acts by prolonging the period between grazings by susceptible animals (ie. provides an effective “spell” of the paddock), opens up the pasture so that sunlight and desiccation can increase the kill rate of existing L3, and probably least importantly, to “mop up” infective larvae by ingesting them, but preventing them from developing into adults. This may involve:
    - Prior grazing with cattle. This is the most effective as there is little cross infectivity between sheep and cattle parasites. The exception is that *H. contortus* can cycle in young calves.
    - Prior grazing with older wethers.
    - Prior grazing with animals carrying an effective sustained release capsule. However the use of sustained release capsules favours the development of anthelmintic resistance.
- “Smart grazing” strategies. Where resistant animals are not available it is possible to clean up paddocks by stocking them with freshly drenched animals, then removing them again before egg production starts (around 19 days). High stocking densities can be used, and this provides an additional use of the pasture without additional contamination, enabling a long effective spelling period. These methods (even with longer graze periods than 19 days) have been shown to work very well in both winter rainfall (Niven *et al.*, 2002) and summer rainfall (Bailey *et al.*, 2009) environments (Figure 17.8).

The growing problem of anthelmintic resistance is driving research into a range of newer control measures that can be integrated with anthelmintic usage and grazing management into more effective integrated control programs that are effective in controlling worms while also slowing the rate of development of anthelmintic resistance. Some of these newer approaches are below.

Breeding for Increased Genetic Resistance by placing greater emphasis on resistance to gastrointestinal nematodes in the breeding objectives. The heritability of WEC is about 0.25. Australian Sheep Breeding Values for WEC are readily available for a range of sires through Sheep Genetics® (Lambplan® and MerinoSelect).

Strategic use of Nutrition. The provision of protein supplements to young stock or periparturient ewes can be used to increase the resistance and resilience of stock to infection, giving lower worm egg counts and sub-clinical losses. However the economics of this need to be carefully evaluated as feeding for worm control alone is rarely economic. However, when coupled with increased productivity economic responses can be obtained

Biological Control of the free-living larval stage has some potential. The most promising candidate is *Duddingtonia flagrans*, a net-trapping, nematode-destroying fungus. These nematophagous fungi normally feed on a range of free-living soil nematodes. It has been shown that the fungal spores can survive passage through the gastrointestinal tract, significantly reducing the number of infective larvae that develop in the faeces and the level of contamination of pastures. The aim is to achieve reductions in clinical and sub-clinical diseases while also stimulating development of acquired immunity in young stock

Vaccination. Despite the ability of sheep to develop strong natural immunity to worms, effective vaccination has still not been commercialised despite much research work. However a new vaccine against Haemonchus contortus is in late stage development currently (2012) and is expected to be commercially available by 2014.

Wormboss. A key industry decision support tool and worm control information site developed by the Sheep CRC is available on the web (see: http://www.wool.com/Grow_WormBoss.htm).

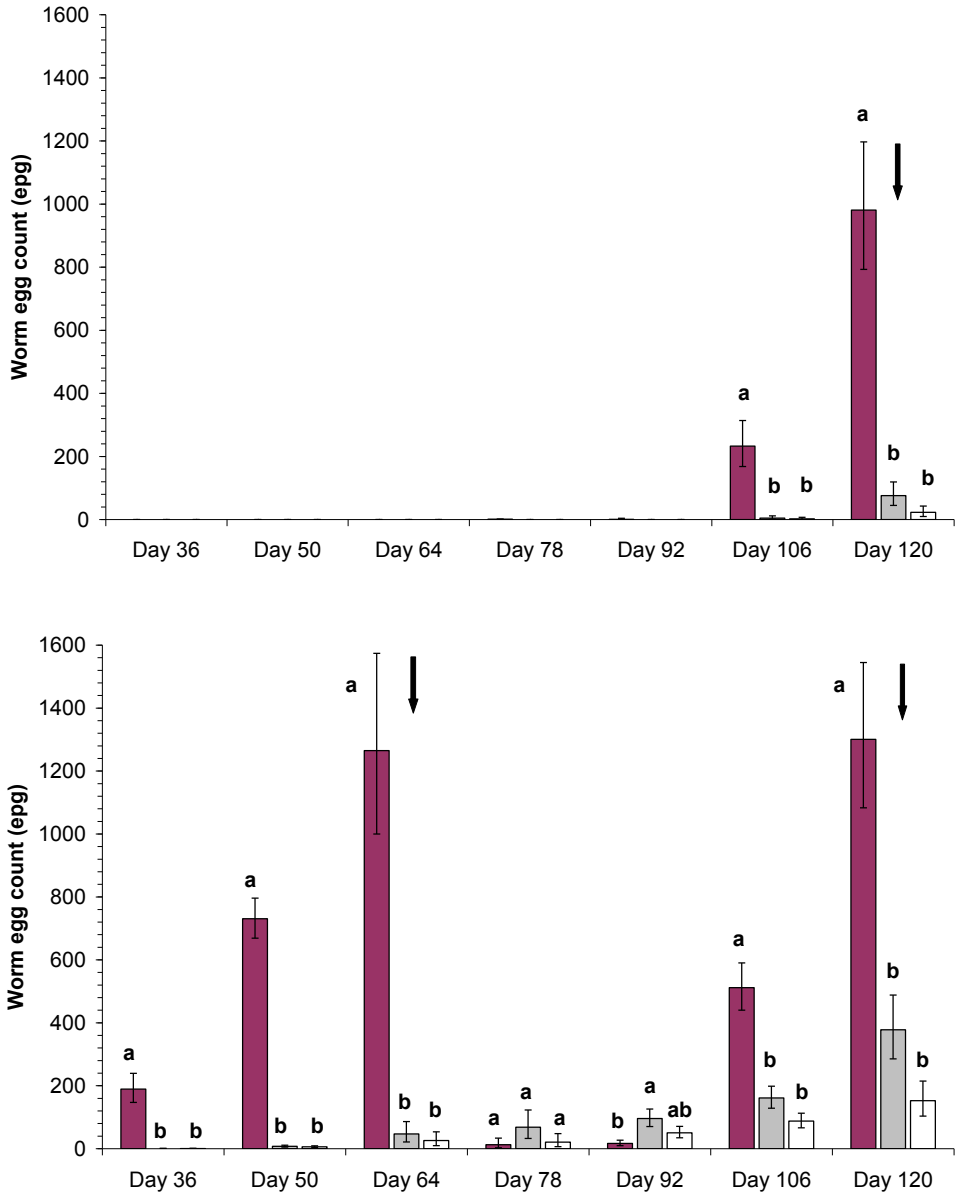

Figure 17.8: Worm egg counts of lamb (top) and ewe (bottom) for lambing ewes grazing paddocks that had been previously prepared for lambing by set stocking with sheep (■), grazing with cattle (▒), or "smart grazed" with sheep (□). Anthelmintic treatment is indicated by arrows beside the group treated. Means not sharing a common letter within each sampling point differ significantly ($p<0.05$) (Bailey *et al.* 2009).

17.9 Liver fluke (*Fasciola hepatica*) infection

Fasciola hepatica and its life cycle

Liver fluke occurs mainly in the higher rainfall areas of SE Australia, encompassing a wide range of climatic types from the Mediterranean-type environment of western Victoria and southern South Australia to the sub-tropical south-east coast of Queensland (Figure 17.9). It is absent from Western Australia. The distribution of liver fluke coincides with the distribution of *Lymnaea tomentosa*, the main intermediate snail host s. The main habitats of the snails include temporary springs, billabongs and swamps. It is also found in irrigation areas, but large lakes and rivers are unsuitable habitats. It is highly prolific and can spread rapidly when climatic and physical conditions are suitable.

The life cycle of the liver fluke is complex, requiring a freshwater snail for part of the cycle. Mature fluke are found in the bile ducts of the liver of the host animal. The eggs produced are passed down the duct, into the small intestine and passed out with faeces. The eggs hatch in up to 3 weeks under warm moist conditions (spring and summer), but may take up to 3 months in winter. The larvae, known as miracidia, then infect the host snail (which is usually *Lymnea tomentosa*, the mud snail). Without this host, the larvae cannot develop. The larvae remain in the snail for up to 12 weeks, multiplying into several hundred second stage larvae known as cercariae, which are passed into the water. A single miracidium may yield over 1000 cercariae. These larvae form cysts (metacercariae) and adhere to pasture plants. Infection occurs when the sheep ingest the cysts while grazing. The cysts dissolve in the gastrointestinal tract of the host, releasing immature fluke. These burrow into the intestinal wall and migrate to the liver. Burrowing through the liver to the bile ducts may take up to 6 weeks. They develop to maturity in 6-7 weeks. The minimum period for whole lifecycle is 4-5 months. Liver fluke are flat and leaf-like in shape, and grey to brown in colour. They measure 1-2 cm in length when mature.

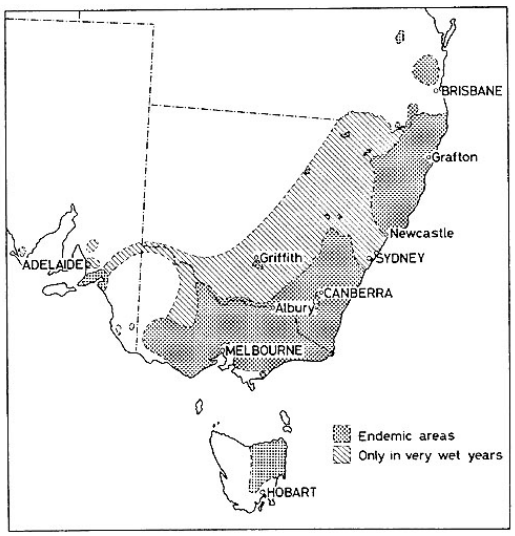

Figure 17.9: The distribution of *Fasciola hepatica* in Australia (Barger *et al.* 1978).

Disease syndromes, clinical signs and effects

Liver fluke is a very serious pathogen of sheep and cattle. It is a long-lived blood-sucking parasite found in the bile ducts of the liver. No immunity to this parasite develops in sheep so it is found in all age classes, but adult cattle do develop effective immunity. There are four different diseases associated with liver fluke.

- **Acute liver fluke** is caused by severe damage to liver 5-6 weeks after ingestion of metacercariae. It is due to immature fluke burrowing into liver tissues. Affected sheep become dull, weak, lose appetite and may die within 24-48 hours of clinical signs appearing
- **Chronic liver fluke** is caused by liver damage arising from mature flukes in the bile ducts and a lesser number burrowing into liver tissue over a period of weeks or months. Affected sheep lose weight slowly and weaken, become anaemic (characterised by pale membranes in mouth and eyes) and may develop “bottle jaw”. Affected sheep look severely “wormy” but do not respond to normal worm drenches that are ineffective against fluke
- **Sub-clinical liver fluke** occurs when the numbers of fluke are low. Affected sheep show depressed productivity in terms of growth, wool production and reproduction

- **Black disease** occurs when the larvae migrate through the liver, stimulating *Clostridium novyi* spores to germinate and multiply. It is characterised by sudden death.

Liver fluke infection results in loss of protein from the host due to the loss of blood and plasma in the bile ducts. Wool growth can be significantly reduced with as few as 25 adult flukes present. Fleece weights are typically reduced by 10-40% (Figure 17.9) indicating that sub-clinical losses can be appreciable.

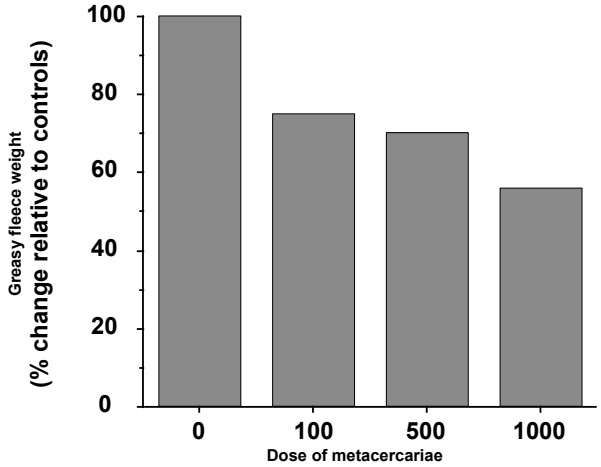

Figure 17.9: Reduction in greasy fleece weights following infection with 100, 500 and 1000 metacercariae (Edwards *et al.* 1976).

Diagnosis and treatment

Immature fluke cause damage to the liver but do not produce eggs. Thus acute liver fluke disease can occur before eggs appear in the faeces. A range of blood tests are available to assess liver damage but they do not indicate the cause of the damage. Diagnosis of acute liver fluke is based on post-mortem examination. Chronic liver fluke can be diagnosed by worm egg count, as the eggs have a characteristic shape. Post-mortem examination is also used in diagnosis of this disease. *Lymnea tomentosa* can also be identified by its characteristic cone-shaped shell and clockwise spiral when viewed from the apex.

There are a number of anthelmintics available which have action against liver fluke, but they vary in the level of control provided over each stage of the parasite. For example, **triclabendazole** (Fasinex®) gives the most effective control over all stages of liver fluke, whereas **closantel** (Seponver®) or **nitroxynil** (Trodax®) are effective against mature fluke and immature fluke 6 weeks after entering the liver. Other treatments such as **albendazole** (Valbazen®) or **oxyclosanide** (Nilzan®) are only effective for controlling adult fluke and have no action against immatures.

Control

A fluke control program has three components:

- **Strategic drenching** to reduce pasture contamination and to reduce liver damage. Drenching in autumn through to early winter removes fluke before they mature and produce eggs. Drenching with Fasinex® at the end of February, mid-May and end of July effectively eliminates pasture contamination
- **Grazing management**. As cattle build up stronger immunity to liver fluke and are less affected by acute liver fluke than sheep, grazing affected pastures with adult cattle in summer is one strategy for reducing contamination. Fencing off highly contaminated areas is another common strategy
- **Snail control**. Chemical control of the snail is not economically feasible as the snail breeds prolifically. Eradication involves destruction of their habitat, requiring drainage of the area. This may not be feasible for water conservation reasons.

Readings

There are no prescribed readings for this lecture. Students are advised to access and read papers that interest them in the references section.

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