Control of roundworms in cattle

This document is part of the COWS Technical Manual which aims to provide a sound basis for advice to the industry.

The manual also comprises chapters on controlling lungworm, liver and rumen fluke, ectoparasites and integrated parasite control.

COWS is an industry initiative promoting sustainable control strategies for parasites in cattle

With thanks to Professor Eric Morgan, Queen's University Belfast for help and guidance in updating this COWS chapter
Section 1: Top tips for controlling roundworms (parasitic gastroenteritis or PGE) in cattle

Informed and sound preparation will minimise roundworm infection with positive effects on enterprise returns.

Identify Risk
1. All grazing cattle are exposed to round/gut worms and consequently can suffer production losses. Younger cattle are most at risk of disease until they acquire immunity. The potential production loss increases with increasing parasite challenge.
2. Permanent pastures grazed by youngstock within the previous six months present a high risk. The safest pastures are newly planted leys following a cereal or root crop.
3. Larval worms accumulate on pasture over the grazing season and infective stages typically peak from mid-summer (July) onwards when the risk of disease is highest.
4. If young cattle are not treated with an effective anthelmintic at housing they are at risk of disease in late winter.

Treat Appropriately
5. Anthelmintic treatments should be targeted at individuals or groups at appropriate times to ensure performance objectives are met. Specific diagnostic tests and monitoring of growth rates can help achieve this. Ask the vet or suitably qualified person (SQP) for details.
6. Anthelmintics can be used strategically to prevent worm populations increasing on pasture over the grazing season. Such treatments are generally focussed on youngstock. Wormer cover should be provided over the first two months of the grazing season to minimise pasture contamination with worm eggs. Ask the vet or SQP to explain.
7. Effective anthelmintic treatment at housing can minimise the risk of worm disease at the end of winter. Ask the vet or SQP about the correct products to use at housing.

Avoid resistance
8. Use anthelmintics correctly. Follow label instructions, weigh cattle if possible and avoid under-dosing. Check dosing equipment regularly to ensure the precise dose is delivered. Always store anthelmintics according to the manufacturers’ recommendations.
9. Anthelmintic resistance has been detected in Cooperia and Ostertagia intestinal worms in many countries including the UK. This must be treated as an early warning. It is essential that anthelmintics are used responsibly.
10. There are many risk factors for anthelmintic resistance. The best way of limiting selection pressure on worm populations is to avoid treating cattle unnecessarily and to ensure the correct drug and dose is used.

Section 2: Introduction to roundworms (nematodes), Ostertagia ostertagi and Cooperia oncophora

Introduction
Cattle can acquire infections with any of several species of roundworms when grazing pastures. The most common and important in northern Europe are Ostertagia ostertagi, which live in the abomasum, and Cooperia oncophora, which inhabit the small intestine. These worms are ubiquitous and can be found on all cattle farms where animals have access to grassland, even when only for short periods. Whilst these parasites are common, clinical disease...
(loss of appetite, scouring and poor condition), is generally only seen in young calves during their first grazing season, when control has been inadequate. Infected animals experience a loss of production, which may be economically important, but can be difficult to detect without accurate observation and recording.

In youngstock, gastrointestinal parasitism can reduce growth rate by up to 30%, even with a low level of worm challenge. This makes it difficult to achieve growth targets for beef animals or replacement heifers.

Even in adult cows, which are likely to be more immune to worms than calves, infections can cause up to 1kg per day drop in daily milk yield.

**Disease**

**Ostertagiosis (parasitic gastritis)**

*Ostertagia ostertagi* develop in the gastric glands in the abomasum, where they damage both glandular tissue and surrounding cells.

A diseased abomasum is thickened and the parasitised glands can easily be seen on the inside surface of the stomach. This pathology has a number of knock-on effects, including reduced acidity of the stomach contents leading to disruption of protein digestion, a release of appetite suppressing compounds and a proliferation of gut bacteria which are normally inactivated by low (acid) pH. However, the most significant effect is loss of protein to the gut.

Parasitic gastritis (PG) in young grazing cattle is also known as Ostertagiosis Type I. Less commonly, disease can appear towards the end of the housing period and this is known as Ostertagiosis Type II.

**Cooperia and Ostertagia (parasitic gastroenteritis)**

The pathology resulting from infections with *Cooperia oncophora* in the small intestine is much less obvious than that induced by *O. ostertagi* in the abomasum, but there is still microscopic damage to the intestinal lining that leads to poor digestion and absorption of nutrients.

Under normal field conditions, young cattle can be infected with both these species at the same time. There is some evidence for synergy between the species. The damaged small intestine cannot compensate for the poor protein digestion and protein loss in the parasitised abomasum. The impact of dual infections is higher than either of them individually. Disease resulting from mixed infections with stomach and intestinal worms is called parasitic gastroenteritis (PGE).

In severe infections, these changes in the gut can lead to the most obvious clinical sign, which is diarrhoea. Loss of nutrients and fluids is partially responsible for the loss of weight and body condition in affected cattle.

**Ostertagiosis Type II**

This condition generally only affects a small proportion of animals in a group, but it can be serious and lead to death.

The cause is the simultaneous development and emergence of inhibited (or arrested) larvae ingested at the end of the preceding grazing season. Instead of developing over the three weeks following ingestion (as normally happens earlier in the season), these larvae undergo a period of arrested development and lie dormant in the gastric glands as tiny, immature, fourth stage larvae (L4).

The L4 persist in the stomach for several months until a trigger (currently unknown) stimulates them to resume development, when they can cause extensive damage to the abomasum, leading to severe clinical signs in affected animals.

**Signs of O. ostertagi infection**

- Loss of appetite
- Loss of body weight and condition
- Diarrhoea
animals. However, the greatest cause of ill-thrift is reduced feed intake, which can account for 60–70% of weight loss observed in young parasitised cattle. Though not so marked, a loss of appetite also seems to explain much of the production loss observed in adult cattle infected with *O. ostertagi*.

###Section 3: The parasites

**Biology**

*Ostertagia ostertagi* and *C. oncophora* are members of a diverse class of worms known as the nematodes (commonly known as roundworms).

Parasitic nematodes are generally host-specific, so cattle nematodes, rarely successfully infect sheep or other livestock. However, there are a few exceptions, such as *Nematodirus battus*, a sheep nematode which occasionally causes disease in calves.

Host specificity in worm species underpins the use of alternative or mixed grazing strategies to reduce the risk of heavy parasite burdens in livestock. Sheep can graze pastures infested with cattle nematode larvae, but these larvae will not develop within sheep and thereafter the pasture will pose a much smaller risk to grazing cattle.

It should be noted that mixed grazing may be inappropriate as a control measure for parasites, such as *Fasciola hepatica* (liver fluke), which readily infects both sheep and cattle (see the COWS liver fluke chapter).

**Life cycle**

Adult parasitic roundworms of cattle are a few millimetres long and just visible to the naked eye if the abomasum or intestines of infected animals are examined post-mortem. Microscopy is needed to appreciate finer structures and to see immature larvae.

The life cycle of these cattle parasitic nematodes is direct. No intermediate hosts are involved during the free-living stages, in contrast to the ruminant liver fluke, and transmission among animals is via infective larvae from pasture.

These worms do not have a migratory phase within the host. There are two sexes and mating is required between males and females in order for the latter to produce fertile eggs. Larvae hatch from worm eggs passed out in dung.

The rate of development is largely temperature-dependent, providing adequate moisture is available in the pat. It can be completed within a week or less when the environmental temperatures are 15 to 23°C, and within three to six weeks at temperatures of around 10°C.

Moisture is also important to enable infective larvae to leave the pat and make their way onto herbage, where they are more likely to be ingested by cattle.
Larvae have limited mobility but can migrate a few centimetres from faeces if conditions are moist. More important is the effect of rainfall, which softens the pat and helps disperse larvae over greater distances.

Infected third-stage larvae (L3) are protected by an outer sheath, which is the retained ‘skin’ of second stage larvae. This makes them relatively resilient and they can survive on pasture for many months, some for over a year, under normal environmental conditions in the UK.

The L3 are sensitive to desiccation, so larval survival is reduced during hot, dry spells of weather. However, larvae can shelter in soil and emerge again after rain.

Grazing management for worm control is based on knowledge of when and how long it takes for infective larvae to appear on pasture and how long they survive. This information underpins strategies intended to limit exposure of grazing cattle to challenge.

When cattle ingest infective larvae, they pass to their preferential sites for development, the abomasum for Ostertagia and the proximal small intestine for Cooperia spp.

Unless worms undergo inhibited development, larvae establish and develop into adult worms within about three weeks, at which time females start to lay eggs. The interval between ingestion of larvae and appearance of worm eggs in dung is known as the pre-patent period. This is an important measurement, as it helps plan worm control programmes, particularly where the objective is to limit contamination of pasture with worm eggs.

Inhibition of larval Ostertagia is an adaptation for survival over winter, but it can also result in disease.

The typical sequence of events is that when cattle are grazing in autumn, a high proportion of larvae ingested stop developing a few days after entering the abomasum. They remain dormant within the gastric glands causing very little damage. However, in late winter or early spring, something triggers them to resume development.

If the numbers of developing larvae are small, there may be few consequences for the host. However, if large numbers of larvae resume development and emerge as adults simultaneously, there is considerable damage to the abomasum with serious consequences known as Ostertagiosis Type II.

## Section 4: Immunity

Cattle do acquire immunity when exposed to roundworms. However, it takes a relatively long time, roughly one full grazing season for Cooperia and up to two grazing seasons for Ostertagia. Even then, immunity is not complete. Cattle of all ages still have worms, particularly Ostertagia, which can result in abomasal pathology.

Older cattle tend to have smaller burdens of adult worms than young cattle, lower faecal egg counts and rarely show signs of clinical disease but can incur production losses.

The rate immunity develops is a function of exposure to infective larvae. Functional immunity can develop even when anthelmintic treatments are given during their first grazing season. However, if young cattle have no exposure at all to pasture as calves, for example calves born in summer in year-round-calving dairy herds, then they should be considered parasite-naive when they go out to grass in the following year.

Calves that are over-protected in their first season at grass by excessive anthelmintic treatment are also at risk of slow development of immunity.

There is a balance between achieving enough exposure to stimulate immunity, but not so much as to cause abomasal damage.

For Ostertagia, plasma pepsinogen testing at housing indicates level of exposure during the first grazing season and can help guide the following year’s strategy.

As a rule of thumb, cattle must be at grass for at least eight months on pasture that has been used for cattle in the previous year, to get enough exposure for immunity to develop.

This total time of effective contact (TEC), is likely to be split between two grazing seasons, and indicates
the time when on the second grazing farmers can expect reasonable levels of immunity to be in place.

In reality, levels of exposure will be affected by other factors, including the effect of climate on development and survival of infective larvae.

Immune development is also influenced by host factors including age, sex, nutrition and potentially, genetics.

Vaccines can accelerate immunity. Promising vaccines are in development for Cooperia but are not yet available. Results are less encouraging for vaccines against Ostertagia.

When they do become available, vaccines will be best used alongside, and in support of, other measures including grazing management and anthelmintics, and not as a like-for-like replacement.

Poor development of immunity in replacement heifers, can lead to high susceptibility in milking cows and the risk of production loss if they become exposed.

Paradoxically, herds in which this occurs tend to have high levels of detectable anti-nematode antibodies in the bulk milk. These herds are most likely to benefit from treatment of milking cows.

At the same time, grazing and worm control procedures in replacement heifers should be reviewed, to improve the development of immunity and reduce the need to treat milking cows in future.

Section 5: Epidemiology

Most cattle in northern Europe are housed over winter and there is virtually no acquisition of new worm infections, as animals are not grazing and survival of larvae in hay and silage is generally poor.

Over winter there is limited and slow development of eggs and larvae in dung pats deposited before housing, due to low temperatures. In this period there is also a gradual decline in the number of infective larvae found on pasture, as these stages do not feed and slowly use up their energy reserves. Survival of larvae declines more rapidly in spring because higher temperatures mean metabolism is faster. Consequently, larval energy reserves deplete more quickly.

Once cattle are turned out in spring, they can acquire infections by encountering residual over-wintered larval populations on pasture. Eggs are also shed from worms overwintering in the intestines of older cattle.

When weaned calves in their first grazing season with limited or no immunity ingest larvae, a high proportion of worms establish in the gut and develop to adults.

Seasonal patterns of challenge are broadly similar with older cattle and in beef suckler herds. However, levels of infection are generally lower, as immune cattle have smaller worm populations that produce fewer eggs.

Fields not grazed early in the season and used for hay or silage production can be available for grazing from July/August onwards. As there has been no cycling of infection and over-wintered larval populations decline exponentially, the risk of cattle acquiring heavy worm burdens while grazing such paddocks is lessened. When cattle are zero-grazed, farmers must be aware of the grazing history of the grass being harvested.

Changes in weather, such as dry summers or mild
autumns, can affect the level of larval challenge and consequently the risk of disease in cattle, as well as the development of immunity. Dry summers are likely to slow down the accumulation of larvae on pastures, but this could rise rapidly following rainfall.

Section 6: Control

Before considering options for control, it is important to establish what a farmer is trying to achieve. Parasitic gastroenteritis most commonly results in sub-optimal production, so control is aimed at preventing or restoring these losses.

In young cattle, probably the most important measure of performance is growth rate, whether animals are destined to be fattened for beef or replacement heifers. Average growth rates of 0.7kg per day or more, on pasture are required if beef cattle are to be finished before they are two years old and replacement heifers are to calve at 24 months. Although adult cattle are rarely the focus of routine worm control, they can be important epidemiologically and can experience production losses. They should not be ignored.

The most common control tools are anthelmintics. However, most livestock farms will have some hay/silage aftermaths available for the second half of the grazing season, and these can be exploited.

If there are sheep on the farm, they can participate in mixed or sequential grazing, which can potentially benefit both types of stock through both worm control and improved pasture utilisation. However, care must be taken when liver fluke are present on a farm, due to the risk of cross-infection between sheep and cattle.

Anthelmintics can be used:

a) Strategically – where target groups of animals are treated, with the primary objective being to limit the contamination of pastures with worm eggs
b) Therapeutically – to treat individuals or groups which are already suffering losses.

Strategic treatments

Strategic treatments are most effective when initiated early in the grazing season – at or within three weeks (the pre-patent period) of turnout. This will ensure worm eggs do not add to pasture contamination.

Thereafter, the aim is to minimise egg contamination of pasture up to mid-July, by which time the over-wintered larval population should have declined to insignificant levels.

Examples of this approach are to administer a long-acting injection or bolus formulation at turnout, or a macrocyclic lactone (ML) that has a shorter persistence of three to six weeks, at turnout, and then again after an interval of six to eight weeks. Occasionally a third treatment after a similar interval is required for a long grazing season.

The most effective strategic approaches will impose a high selection pressure for resistance development.

Computer models have been developed to predict the effect of weather, grazing patterns and treatment on pasture larval levels and these can be used to support sound decisions under climate change. Meaningful risk forecasts cannot be made without information on grazing history.

Control options

- Grazing strategies
- Strategic anthelmintic to minimise pasture contamination
- Therapeutic anthelmintic based on monitoring stock
- Housing treatments

Figure 5: Aim to minimise pasture contamination.
These can be modified with the help of diagnostic and performance indicators to lower the pressure.

These protocols are based on the assumption that cattle remain on the same pasture throughout the grazing season. If animals are moved, the risk posed by the next pasture needs to be assessed and appropriate steps taken to maintain control.

Strategic control with anthelmintics is classically applied to autumn/winter-born weaned calves in their first grazing season, and sometimes to spring-born calves in their second grazing season.

**Therapeutic use of anthelmintics**

If no measures are taken to limit pasture contamination through management or anthelmintics, grazing cattle will be exposed to ever-increasing risks of disease and experience production losses. They may suffer clinical parasitic gastroenteritis.

To address this cattle can be closely monitored and treated when they start to show signs of ill-thrift and diarrhoea. Or tactical treatments can be given to at-risk groups in anticipation of losses. However, this can lead to production losses and greater pasture contamination.

It is extremely important to take into account the risk of lungworm infection when making any changes to control measures aimed at gastro-intestinal worms.

**Housing treatments**

Treatment of cattle for parasites at housing using anthelmintics can be considered as either strategic or therapeutic or both.

This treatment is strategic because when cattle go out to pasture the following year, they will not immediately contaminate pasture with worm eggs.

This treatment is therapeutic because worm burdens are removed, and cattle are free of their negative impact for the remainder of the housing period.

In addition, as long as products containing macrocyclic lactones (MLs) are used, inhibited *O. ostertagi* larvae will be removed and the risk of ostertagiosis type II is minimised. Benzimidazoles may also be used, but their efficacy against inhibited larvae can be unpredictable.

It is efficient to use more than one parasiticide and/or combination products at housing, as it is also an opportune time to treat for lungworm and liver fluke and to prevent lice and mange infestations later in winter. Choice of products, should take into account the need for fluke control – and especially the choice of fluke active ingredient.

**Quarantine**

Incoming or returning stock should be treated to minimise the risk of introducing resistant roundworms to the farm.

Cattle can be treated with an anthelmintic, the treatment then efficacy tested and retreated with a different class of anthelmintic of required. If possible, cattle should then be moved to pasture with a low/moderate parasite burden.

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**Section 7: Monitoring and Diagnostics**

Normal seasonal patterns mean calendar-based control programmes can work and cattle do not necessarily require any additional parasite diagnostics.

However, diagnostics and performance monitoring can help with decision-making under variable weather conditions.

**Worm egg counting**

Taking dung samples and measuring the...
concentration of worm eggs can provide useful information about infection dynamics over a grazing season. Faecal egg counts (FEC) can be helpful in diagnosis of parasitic gastroenteritis but there are limitations. Note that low FEC in cattle can still be significant due to the large volume of dung produced.

**Plasma pepsinogen**

The concentration of pepsinogen in blood plasma is related to the extent of abomasal damage caused by parasites, such as *O. ostertagi*.

Pepsinogen levels at the end of the first grazing season can indicate if exposure has been too high, causing production losses, or too low with consequent poor development of immunity.

**Worm antibodies**

Antibodies are a measure of the host immune response to parasites. However, it is important to recognise they do not always correlate with the level of protective immunity.

The concentration of *O. ostertagi* antibodies in milk is related to exposure and can provide a good estimate of potential loss of milk caused by the parasite at herd and individual levels. High antibody levels can prompt treatment in dairy cows but should also trigger review of herd control strategies.

### Performance monitoring and targeted treatment

In the presence of adequate nutrition and absence of other endemic diseases, parasitic gastroenteritis is the main factor determining growth rate at grass.

Regular weighing of youngstock at pasture and treatment of those failing to reach growth targets can result in good worm control, while reducing the number of anthelmintic treatments.

This approach limits pasture contamination, whilst maintaining some supply of anthelmintic susceptible larvae to maintain an appropriate challenge. This method can also help to slow resistance development.

Parasites are not evenly distributed among animals within a herd. Treatment of individuals that are performing poorly can lead to a disproportionate reduction in total worm infections.

There is currently no practical method of measuring pasture contamination. Knowledge of grazing history with different classes of stock, can help identify fields with likely high, moderate or low larval levels at a given time.
Anthelmintic resistance (AR) is a global problem that is common and widespread in sheep and goat roundworms. It is increasingly being observed in cattle parasites.

In Europe, the parasite species currently implicated most in AR is *C. oncophora*, but it is increasingly reported in *O. ostertagia*. The impact of this issue is currently unknown but early action is necessary to maintain efficacy of current anthelmintics.

The challenge is how to limit selection for, and hence delay emergence of AR and how to manage it when it does appear. Objective identification of risk factors for AR on cattle farms is lacking in Europe.

Basic guidelines for performing an anthelmintic efficacy check:

1. Before doing a check, perform a FEC on a sample of 10–15 animals from the group to be tested, which should ideally be calves in their first grazing season.
   A pooled FEC is adequate and will save money. If egg counts are reasonably high (>200 epg) proceed with treatment. Treat at a lower threshold, eg. 50 epg, if an FEC method of higher sensitivity, eg. 1–10 epg is used.

2. Collect faecal samples on the day of treatment (Day 0). Place at least 10g in total sampled from three different areas of each pat into an individually identified plastic bag for each animal. Exclude air and keep cool.

3. Administer anthelmintic. Calculate dose from an accurate body weight (scales of weigh band). Ensure all the dose is swallowed or injected or poured-on appropriately.

4. Collect faecal samples in the same manner 14–17 days after anthelmintic administration, or 7–10 days after treatment with levamisole.

5. In all case, process faecal samples at the lab as soon as is feasible. Samples should be re-mixed before any sub-sampling to help account for variation within a single pat. A detection limit of 5 epg or less is considered best practice to reduce the chance of mis-classifying resistance.

6. Calculate percentage efficacy using the formula:
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   \%\text{FECR} = \left( \frac{\text{Mean Day 0 FEC} - \text{Mean Day 14 FEC}}{\text{Mean Day 0 FEC}} \right) \times 100
   \]
   The percentage efficacy should be at least 95%.

7. Pooled FEC can be used before and after routine treatment, even from fresh dung pats sampled at pasture to confirm good anthelmintic efficacy. This method is much cheaper and easier but not as accurate. Suspect efficacy from pooled counts should be followed up with a full check using individual samples whenever feasible. Individual tests allow a level of confidence in the calculated result.

Section 8: Anthelmintic resistance

The most obvious risk is the use of whole herd frequent or continuous dosing regimens. Under-dosing may also allow (partially) resistant worms to survive treatment.

There are a number of common-sense practices that should help to limit selection pressure on cattle worm populations (see Section 1: Top tips for controlling parasitic gastroenteritis in cattle) without compromising worm control.

In addition, some of the newer approaches to parasite control, such as the Targeted Treatment approach in Section 7, can help farmers continue with successful production into the future.
Section 9: Planning control on farm

Practical control advice should be tailored to the individual farm and consider:

- Production objectives for the different classes of cattle
- Farm infrastructure, particularly in relation to pastures, grazing management and handling facilities
- Presence of other helminth parasites, such as lungworm and liver fluke
- Efficacy of available anthelmintics.

Equipped with this information, it is possible to compile a risk assessment for parasitic gastroenteritis in all categories of stock.

This assessment is dynamic and needs to be reviewed several times during the grazing season. In addition, housing and turnout are good times to reflect on the previous months and plan for the future.

Once risks have been quantified, it is possible to decide on control options, using management and anthelmintics.

The actual choice of anthelmintic is ideally determined through discussions with the vet and SQP at retail outlets. For more information also see the COWS Integrated Parasite Control chapter.