

Control of parasitic gastroenteritis in cattle

This document is part of the COWS Technical Manual aiming to provide a sound basis for advice to industry. The manual also comprises chapters on controlling liver and rumen fluke, lungworm, ectoparasites and insect pests, and integrated control of parasites on cattle farms



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

Section 1: Top 10 tips for controlling parasitic gastroenteritis in cattle

Identify Risk	1. All grazing cattle are exposed to gut worms and consequently can suffer production losses; younger cattle are most at risk of disease until they acquire immunity.
	2. Permanent pastures grazed by young stock within the previous six months present a high risk. The safest pastures are newly planted leys following a cereal/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 the late winter.
Treat Appropriately	5. Anthelmintic treatments should be targeted at individuals or groups at appropriate times to ensure epidemiological and performance objectives are reached. Specific diagnostic tests and monitoring growth rates and overall performance can help achieve this. Ask your 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 young stock and anthelmintic cover should be provided over the first two months of the grazing season to minimise pasture contamination with worm eggs. Ask your vet or SQP to explain.
	7. Effective anthelmintic treatment at housing can minimise the risk of worm disease at the end of winter. Ask your 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. Regularly check dosing equipment to ensure the prescribed dose is delivered.
	9. Anthelmintic resistance in one genus of intestinal worm, <i>Cooperia</i>, has been detected in many countries around the world, including the UK. So far, this issue does not seem to be causing significant economic losses, but this is an early warning to manage and use anthelmintics responsibly.
	10. There are many risk factors for anthelmintic resistance, but the best way of limiting selection pressure on worm populations is to avoid treating too many cattle too often and ensure the correct dosage is used. Good disease control and production can be achieved by using anthelmintics in a responsible way which focuses on treating individuals or groups at appropriate times and recognising that many animals can thrive without frequent treatments.

Section 2: Nematodes or Roundworms, *Ostertagia ostertagi* and *Cooperia oncophora*

Introduction

Cattle can acquire infections with any of several species of roundworms when grazing pastures, but amongst these 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. Much more commonly, infected animals experience a loss of production, which may be economically important, but can be difficult to

Key concerns

- Roundworms found on all cattle pastures
- Disease mostly seen in calves and young cattle
- Can reduce growth rates by 30%

detect without accurate observation and recording.

In young stock, gastrointestinal parasitism can reduce growth rate by ~30%, even with a low level of worm challenge, and this makes it difficult to achieve growth targets for both beef animals and replacement heifers. Even in adult cows, which are likely to be more immune to worms than calves, infections can cause a ~1kg per day drop in daily milk yield.

Disease

Ostertagiosis (parasitic gastritis)

O. ostertagi develops in the gastric glands in the abomasum, where it damages both glandular tissue and surrounding cells. A diseased abomasum is thickened and the parasitized glands can be easily seen on the inside surface of the stomach. This pathology has a number of knock-on effects, including reduced acidity of the stomach contents, which leads 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.

This process explains the occurrence and manifestation of the complex called parasitic gastritis (PG) in (young) grazing cattle (also called Ostertagiosis Type I). Less commonly, disease can appear towards the end of the housing period and this is known as Ostertagiosis Type II.

Ostertagiosis Type II - This condition generally only affects a small proportion of animals in a group, but it can be serious and even 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 next three weeks following ingestion (as normally happens earlier in the season), these larvae undergo a period of arrested development and lie quiescent 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.

Cooperia and Ostertagia (parasitic gastroenteritis)

The pathology resulting from infections with *Cooperia* in the small intestine is much less obvious than that induced by *O. ostertagi* in the abomasum, but there is still microscopic

Ostertagia lesions on abomasal mucosa



Photo courtesy of Moredun Research Institute.

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 concurrently and there is some evidence for synergy between the species insofar as the damaged small intestine cannot compensate for the poor protein digestion in the parasitized abomasum, hence 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, diarrhoea. Loss of nutrients and fluids is partially responsible for the loss of weight and body condition in affected animals, but the greatest cause of ill-thrift is reduced feed intake, which can account for 60-70% of weight loss in young parasitized 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*.

Signs of *O. Ostertagi* infection

- Loss of appetite
- Loss of body weight and condition
- Disrupts digestion leading to diarrhoea

Section 3: Gut worms: the parasitic way of life

Biology

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

Parasitic nematodes are generally host-specific, so cattle nematodes, for example, rarely successfully infect sheep or other livestock, although 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. Thus, sheep can graze pastures infested with cattle nematode larvae (and *vice versa*), but these larvae will not develop within sheep; thereafter the pasture will pose a much smaller risk to grazing cattle. It should be

Adult female *Cooperia oncophora*



Photo courtesy of Moredun Research Institute.

noted that mixed grazing may be inappropriate as a control measure for parasites, such as liver fluke, *Fasciola hepatica*, which readily infects both sheep and cattle (see Section 6 of 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, but microscopy is needed to appreciate finer structures and to see immature larvae.

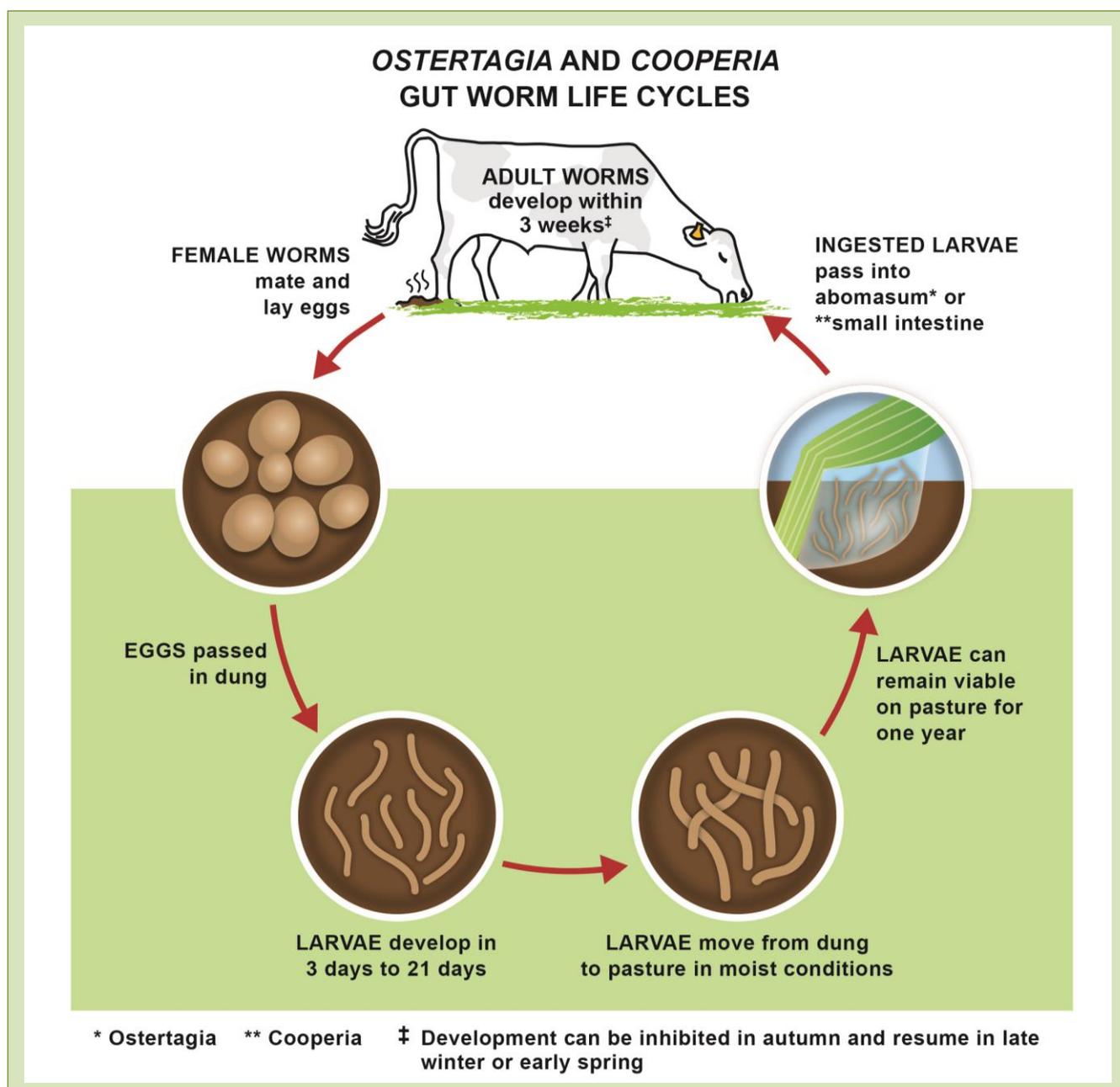
The life cycle of cattle parasitic nematodes, illustrated here, is 'simple'; that is to say that no intermediate hosts are involved during the free-living stages (in contrast to the ruminant liver fluke) and transmission among animals is via infective larvae from pasture. These worms do not have a migratory 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, and can be completed within a week or less when the environmental temperatures are 15 to 23°C and within three weeks at temperatures of around 10°C.

Moisture is also important, particularly to enable infective larvae to leave the pat and get on to 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, but probably more important is the effect of rainfall, which softens the pat and helps disperse larvae over greater distances.

Once on pasture, infective third stage larvae (L3), protected by an outer sheath (retained 'skin' of second stage larvae), are relatively resilient and can survive for many months and in small numbers 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. Grazing management for worm control is based on knowledge of when and how long it takes for infective larvae to appear on pasture and for 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 *O. ostertagi* and the proximal small intestine for *Cooperia* spp.



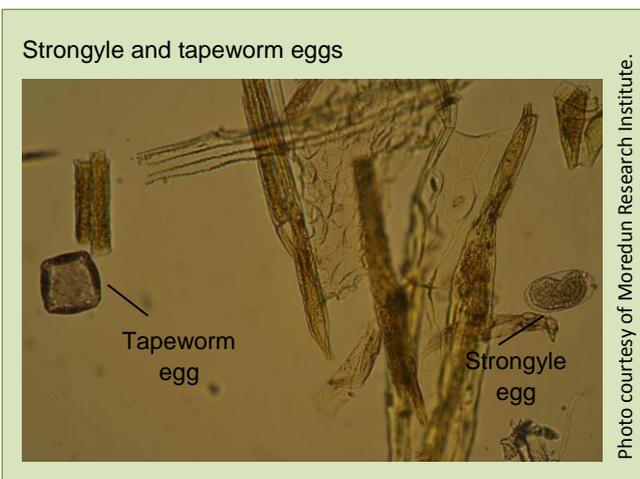
Unless worms undergo inhibited development, larvae establish and develop into adult worms within about three weeks, at which time females start to lay eggs. The interval between ingestion of larvae and appearance of worm eggs in dung is known as the pre-patent period. This is an important measurement,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

Inhibition of larval *O. ostertagi* 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, but in late winter or early spring, something triggers them to resume development. If the numbers of developing larvae are small, then there may be few consequences for the host, but if large numbers of larvae resume development and emerge as adults simultaneously there is considerable damage to the abomasum with serious consequences (see the Section 2 box on 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 *C. oncophora* and up to two grazing seasons for *O. ostertagi*. Even then, immunity is not complete: cattle of all ages still have worms, particularly *O. ostertagi*, which can result in abomasal pathology similar to that seen in young cattle. The main difference between immature and adult cattle is that older cattle rarely show obvious signs of clinical parasitism, with most harbouring small burdens of adult worms, which lay fewer eggs.



Acquiring immunity

- Can take up to two grazing seasons
- Immunity is never complete
- Only develops when cattle graze pasture

There is a common belief that highly effective parasite control with anthelmintics can interfere with the acquisition of immunity, but there is limited evidence to support this. Several studies have shown functional immunity is present in yearlings irrespective of the number and type of anthelmintic treatments 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-naïve when they go out to grass in the following year.

Section 5: Epidemiology

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

Over the winter period there is limited and slow development of eggs and larvae in dung pats deposited before housing, due to generally 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. It is for this reason that survival of larvae declines more rapidly during a mild winter because higher temperatures mean that metabolism is faster and energy reserves disappear at a faster rate. In contrast, survival of larvae is higher following a cold winter, particularly under snow, as larvae survive in a quiescent state at low temperatures and snow cover is thought to prevent dehydration.

Once cattle are turned out in spring, they encounter residual over-wintered larval populations on pasture and acquire infections. When weaned calves in their first grazing

Risk Factors

- Increase after a cold winter
- Cattle in first grazing season have no immunity
- Peak larvae numbers reached in July
- Can be reduced with grazing strategies

season with limited or no immunity ingest larvae, a high proportion of worms establish in the gut and develop to adults.

Adult female worms in naïve hosts are generally prolific egg layers and infected calves may pass high concentrations of eggs in dung following the pre-patent period. Thereafter, a new generation of eggs develop into larvae within dung, but as temperatures are typically low in spring, this takes several weeks – for development to be complete. As the season advances and temperatures increase, development time to infective larvae shortens and, if calves remain on the same pasture and are not treated appropriately with anthelmintics, larval numbers on pasture typically peak in early July and remain high until animals are housed. It is these high levels of pasture L3 that can lead to outbreaks of clinical disease.

Seasonal patterns of challenge are broadly similar with older cattle and in beef suckler herds, but 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; because 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.

Third stage larvae - *Cooperia oncophora*



Photo courtesy of Moredun Research Institute.

Section 6: Control

Before considering options for control it is important to establish what a farmer is trying to achieve with their cattle. PGE most commonly results in sub-optimal production and therefore 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 to be 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, so should not be ignored.

The commonest control tools are anthelmintics, but most livestock farms will have some hay/silage aftermaths available for the second half of the grazing season, which can be exploited. In addition, if sheep are on the farm, they can participate in mixed or

Control options

- Consider grazing strategies
- Strategic anthelmintic use to minimise pasture contamination
- Therapeutic anthelmintic use based on monitoring stock
- Housing treatments can be beneficial

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 is present on a farm because of the risk of cross infection between sheep and cattle.

The use of anthelmintics can be categorised in various ways, but fundamentally they are used:

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

Strategic approaches

To be effective, strategic treatments need to be initiated early in the grazing season – at or within three weeks (the pre-patent period) of turnout – to 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 administration of long acting injection or bolus formulations at turnout or administration of macrocyclic lactones (MLs) that have a shorter persistence (3-6 weeks), at turnout and then after an interval of six to eight weeks; occasionally a third treatment after a similar interval is required for a long grazing season.

These protocols are based on the assumption that cattle remain on the same pasture throughout the grazing season; if animals are

Aim to minimise pasture contamination



Photo courtesy of Moredun Research Institute.

moved, then 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 beef suckler calves in their second grazing season.

Therapeutic use of anthelmintics

If no measures have been taken to limit pasture contamination with parasites through pasture management or anthelmintics, grazing cattle will be exposed to ever increasing risks of disease over the season and will experience production losses and may suffer clinical PGE. To address this, cattle can either 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.

Both strategic and therapeutic approaches can provide satisfactory levels of PGE control, although animal performance is generally superior in young cattle which are not exposed to high levels of challenge (i.e. in strategic programmes), so veterinarians and advisors must decide which systems are best suited to meet individual farmer expectations and targets, within the constraints of farm structure and management.

Housing treatments

Treatment of cattle for parasites is a common practice at housing and using anthelmintics at this time can be considered as either strategic or therapeutic or both. Therapeutic because worm burdens are removed and cattle are free of their negative impact for the remainder of the housing period. Strategic because when cattle go out to pasture the following year, they will not immediately contaminate pasture with worm eggs. In addition, providing 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 common practice 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 (see the [COWS Integrated Parasite Control](#) chapter).

**Use the product most suitable for the time of year and management of the cattle involved.
See www.cattleparasites.org.uk for products available**

Section 7: Monitoring and Diagnostics

The pattern of appearance of infective L3 on permanent pasture is quite stereotypic, although the magnitude of risk can vary year-to-year, depending primarily on prevailing weather conditions. For example, a very dry summer can mean infective larvae remain in the pat until rain falls in autumn and this may be followed by a rapid increase in pasture infectivity late in the season.

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

Parasite monitoring

- Faecal egg counts of limited use
- Milk tests may be useful in dairy herds
- Regularly weighing young cattle is a good guide of the need for treatment

there is an increasingly important role for diagnostics to help with decision making or if things go wrong and, irrespective of what control approach is used, it is important to monitor all animals closely through the grazing season.

Worm egg counting

Taking dung samples and measuring the concentration of worm eggs therein is popular amongst researchers and clinicians, but while routine faecal egg counts (FEC) can provide useful information on infection dynamics over a grazing season and in the differential diagnosis of gastroenteritis and ill-thrift in young cattle at grass, there are also several limitations (see table).

A FEC can indicate an approximate number of eggs contaminating pasture at the time of sampling, which can in turn provide an estimate of the magnitude of parasite challenge and likely risk of disease over the next weeks and months.

The standard method for FEC is the McMaster and it measures number of eggs per gram (epg) to the nearest 50. Particularly when interpreting FEC from different ages of cattle, it is important to remember a FEC is a concentration of eggs in dung, not an absolute number. The FEC must be multiplied by the fresh weight of dung produced per day to gauge the level of potential pasture contamination.

A useful rule-of-thumb is that cattle deposit dung at a rate of ~5% of liveweight. Thus a 200kg calf produces $200 \times 0.05 = 10\text{kg}$ of faeces/day = 10,000g. If it has a FEC of 200

epg, then total egg output is $200 \times 10,000 = 2$ million worm eggs/day.

It is interesting to perform the same calculation for an adult cow weighing say 500kg; it produces $500 \times 0.05 = 25\text{kg}$ dung/day. Adult cattle commonly have FECs of less than 50 epg, so using a value of say 20 epg, the daily deposition of worm eggs by a cow is $20 \times 25,000 = 500,000$ worm eggs per day.

For investigations into older cattle, which typically have FECs less than 50 epg, and for conducting a FEC reduction (FECR) test (see below), it is preferable to use a method with a sensitivity of 1-10 epg.

Under UK conditions, FEC is not consistently or linearly related to:

- a) Current population of adult and/or immature worms in the gastrointestinal tract of individuals.
- b) Pathology caused by the worms and knock-on effects on host physiology.
- c) Growth rate (as affected by worms) in young cattle.

Thus FEC should be interpreted in the context of a risk assessment for the severity of parasitism alongside other parameters, such as pasture history, animal age, production level and time of year.

What Faecal Egg Counts can tell you	What Faecal Egg Counts cannot tell you
FEC provide an <i>indication</i> of levels of pasture contamination due to numbers of nematode eggs being passed out in faeces	FEC cannot reliably provide an accurate determination of total worm burden
FEC indicate presence of egg-producing adult female nematodes, but this is not necessarily quantitative	FEC is not linearly related to liveweight gain in young cattle
FEC indicate which animals are contributing most to pasture contamination, providing a selection criterion for targeting anthelmintics to limit faecal egg output	FEC cannot determine nematode species in a dung sample, unless eggs are morphologically distinct (such as with <i>Nematodirus</i> spp.)
FEC can indicate how effective an anthelmintic is at reducing trichostrongyle egg excretion	FEC cannot detect nematode stages which are not laying eggs (i.e. adult male worms, larvae or inhibited stages)
Recent research on a small number of farms has shown dairy cows with a positive FEC at calving may have a reduced milk yield in that lactation	FEC do not indicate eggs are viable and alone cannot differentiate between: <ul style="list-style-type: none"> • eggs surviving due to anthelmintic resistance • eggs surviving due to under-dosing or maladministration • reinfection or resumption of egg laying (due to inappropriate sampling periods after administration)
FEC can be included in the differential diagnosis of ill-thrift and scour in pastured cattle	Standard FEC methods using salt flotation are not recommended for detection of lungworm, liver fluke or tapeworm eggs

Plasma pepsinogen

The concentration of pepsinogen in blood plasma is related to the extent of abomasal damage caused by parasites, such as *O. ostertagi*. It is quite commonly used in research, but has some value in the field. Its

limitations include: it can only be used for abomasal worms, results are variable between laboratories and it requires invasive blood sampling from several animals.

Worm antibodies

Antibodies are a measure of the host immune response to parasites, but it is important to recognise they do not always correlate with the level of protective immunity, but may be associated quantitatively with exposure to worms. Whilst early research examined the dynamics of antibody responses in young

grazing cattle to the two common species *O. ostertagi* and *C. oncophora*, recent work has focussed on *O. ostertagi* in adult (dairy) cattle. This found the concentration of *O. ostertagi* antibodies in the bulk milk tank is related to exposure and can provide a good estimate of potential loss of milk caused by the parasite.

Performance monitoring

Perhaps one of the most interesting recent changes in diagnostics has arisen through research into Targeted Selective Treatment (TST). This approach centres on targeting anthelmintics at individual animals which will benefit most from treatment. In young animals, TST is based on the observation and assumption that, in the presence of adequate nutrition and absence of other endemic diseases, parasitic gastroenteritis is the main

factor determining growth rate at grass. Thus, regular weighing of young stock at pasture and treatment of those failing to reach growth targets can result in good worm control, while reducing the number of anthelmintic treatments. It was this research that confirmed the findings from earlier studies that FEC does not reliably identify cattle suffering poor weight gains as a result of parasitic gastroenteritis.

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. Select those animals with a FEC of ≥ 200 epg* and treat with anthelmintic at 100% recommended dose rate
2. Collect faecal samples immediately before 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 or calibrated tape). Ensure dose is swallowed, injected appropriately or guidelines for pour-on administration are followed.
4. Collect faecal samples in the same manner 14-17 days after anthelmintic administration; or 7-10 days after levamisole treatment.
5. In all cases, process faecal samples at the lab as soon as is feasible. Samples should be remixed 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 chance of misclassifying resistance.
6. Calculate percentage efficacy using the formula:

$$\% \text{ FECR} = ((\text{Mean Day 0 FEC} - \text{Mean Day 14 FEC}) / \text{Mean Day 0 FEC}) \times 100^*$$

Treat at a lower threshold, for example 50 epg, if a FEC method of higher sensitivity, for example 1-10 epg, is used.

Section 8: Anthelmintic resistance

Anthelmintic resistance (AR) is a global problem that is common and widespread in sheep and goat helminths and has also been observed in cattle parasites. In Europe, the parasite species currently implicated most in AR is *C. oncophora*, eggs of which are often found in faecal samples taken after treatment with a macrocyclic lactone product, especially topical formulations. The impact of this issue on parasite control appears to be quite limited so far, but clearly this situation could deteriorate if resistance becomes more common or develops in *O. ostertagi*, which is more pathogenic.

Many scientists think anthelmintic resistance is an inevitable consequence of natural selection in nematodes subject to treatment and thus the question is not 'if', but 'when' it will occur. 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, but extrapolation from countries such as New Zealand and from other species like sheep

provide some pointers. Of these, perhaps the most obvious risk is the use of high frequency, short interval treatments on intensively stocked young cattle, for example administering a macrocyclic lactone at four week intervals, 6-8 times over successive grazing seasons on the same fields. Underdosing may also allow (partially) resistant worms to survive treatment.

High frequency treatments are relatively rare in Europe and this may explain why it is only recently that resistance has been identified here. 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 Selective Treatment \(TST\) approach in Section 7](#), can help farmers continue with successful production into the future. Also see [Section 4: Parasiticide resistance in the COWS Integrated Parasite Control chapter](#).

Section 9: Planning control on farm

Many farmers and clinicians would prefer to have formulaic advice for wide application, but worm control requires local knowledge, so appropriate options can be assessed accurately. For example, there is no point in talking about mixed grazing, if the farm has no sheep. Nevertheless, general worm biology is the same, irrespective of the farm, and so consequently is the under-pinning epidemiology.

Primarily, for the individual farm it is important to consider:

- production objectives for the different classes of cattle
- farm infrastructure, particularly in relation to pastures, grazing management and handling facilities
- the presence of other helminth parasites, such as lungworm and liver fluke

- efficacy of available anthelmintics.

Equipped with this information, it should be 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 should be possible to decide on control options, using management and anthelmintics. The actual choice of anthelmintic is ideally determined through discussions with the farm veterinary practice and suitably qualified person (SQP) at retail outlets. For more information also see the [COWS Integrated Parasite Control chapter](#).