Control of lungworm in cattle

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

The manual also comprises chapters on controlling roundworms, liver and rumen fluke, ectoparasites and practical parasite control planning.

COWS
Control Of Worms Sustainably

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

With thanks to Professor Eric Morgan, Queen's University Belfast, Professor Diana Williams and Dr John Graham-Brown, University of Liverpool for help and guidance in updating this COWS chapter

Section 1: Top tips for controlling lungworm (parasitic bronchitis)

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

Identify Risk

1. Lungworm outbreaks are unpredictable, but are more prevalent in wetter, western areas of Britain. They usually occur in summer and early autumn. In endemic areas, younger cattle are at risk until they acquire immunity through exposure to lungworm larvae
2. Suspect lungworm infection if there is coughing or respiratory distress in grazing cattle, particularly in first season grazing calves
3. Animals exposed to lungworm usually develop resistance to re-infection. Lack of exposure may result in clinical signs occurring in older cattle, including milking cows. Previously immune animals may exhibit signs if immunity wanes, or pasture infectivity is high
4. Bought-in calves or adult cattle may introduce lungworm onto a farm, so quarantine and treat all incoming cattle. Most anthelmintics used for control of gut roundworms are effective against lungworms. Check product choice with the vet, suitably qualified person (SQP) or registered animal medicines adviser (RAMA)

Treat Appropriately

5. Routine vaccination should be considered for calves born into herds with an identified lungworm problem, or where there is a previous history of lungworm on the farm (available until the end of 2027)
6. If strategic anthelmintic programmes are used to control parasitic gastroenteritis (PGE) in first year grazing cattle, this will also prevent build-up of lungworm larvae on pasture over the grazing season. This will, however, prevent any development of protective immunity and will increase selection pressure for drug resistance
7. In lungworm outbreaks, prognosis will depend on the severity of the disease and timeliness of treatment. In more advanced cases, treatments may not resolve and could exacerbate illness. Deaths may occur in severely affected groups
8. Severely affected animals may require additional supportive treatment, which should be discussed with the vet

Plan ahead

9. Work out a control strategy. Disease can occur rapidly due to relatively fast development time on pasture and low numbers of parasites required to initiate disease. Farms with a previous history of lungworm should consider vaccination (available until the end of 2027)

Section 2: Introduction to lungworm (parasitic bronchitis, dictyocaulosis, husk, hoose)

Lungworm is an economically important parasitic infection of the bovine respiratory tract caused by the nematode *Dictyocaulus viviparus*.

This species mainly affects cattle but is occasionally reported in other ruminants such as deer.

The parasite is widespread, with patchy distribution across the UK.

Risk is generally higher in wetter areas. There is also a strong seasonal pattern of infection, with most cases occurring in late summer and autumn. However, due to complex epidemiology, infection and disease risk are difficult to predict accurately at farm-level.

Lungworm infection can cause substantial production losses. The impacts of severe infections and outbreaks are profound in terms of stock losses and costs of treatments, with longer-term production

effects resulting from recovered animals never achieving their productive potential.

Studies also show lungworm infection in youngstock is associated with reduced liveweight gain, while infection in adult dairy cows has been associated with milk yield losses of up to 1.74kg/cow/day.

As with many other parasitic roundworms, lungworm infection is acquired through consumption of grass on pastures contaminated with its infective larval stage.

Clinical signs of lungworm are most commonly seen in first-year grazing cattle in late summer and autumn, but can also occur earlier in the year and in older cattle. The most characteristic clinical sign of lungworm infection is widespread coughing within the group. Death can occur in heavy infections.

Cattle of any age can be exposed to and become infected with lungworm. However, dairy or dairy-cross calves, spring-born beef suckler calves in their first grazing season and autumn-born beef suckler calves in their second season, are typically considered at greatest risk due to a lack of previous exposure.

Once exposed to a lungworm infection, animals typically obtain a degree of immunity that prevents further disease risk. However, older animals can still harbour light infections and can act as an important source of pasture contamination. They are therefore a risk to younger animals.

Key concerns of a lungworm outbreak

- Sudden, severe outbreaks can occur unexpectedly
- Impacts on youngstock performance
- Reduced milk yield in adult cows
- Deaths can occur despite treatment with anthelmintics

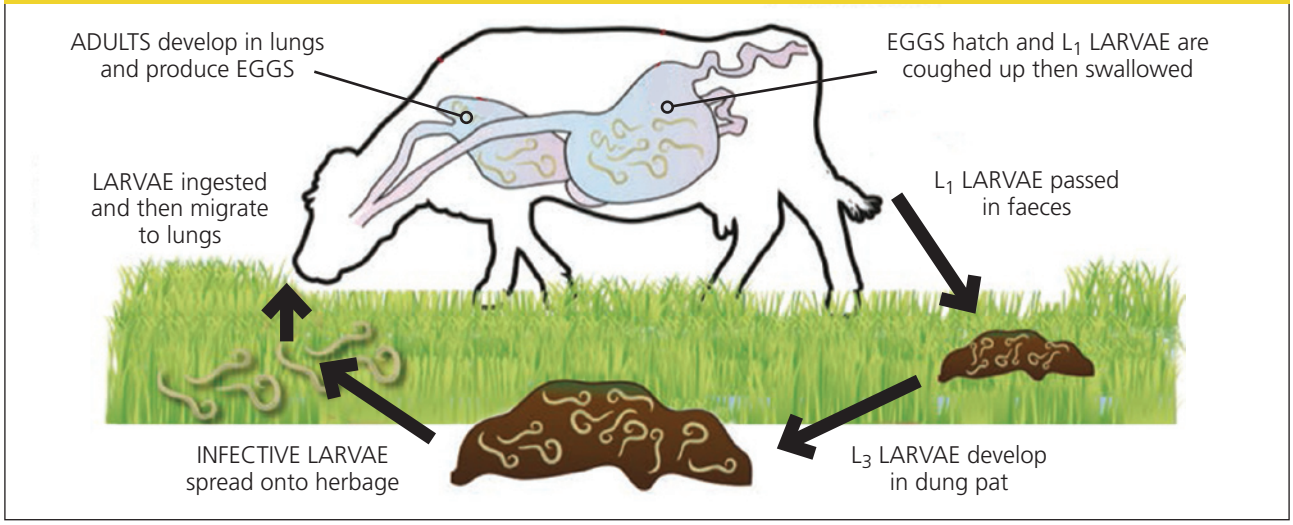
Figure 1: Younger animals are most susceptible to infection.



Animals not exposed to lungworm in early life remain at risk from disease, having never been able to develop immunity. Sudden, heavy pasture challenge can occasionally lead to clinical disease in cattle, even if they have been previously exposed.

Section 3: The parasite

Figure 1: Lungworm life cycle



Adult worms are slender, thread-like and measure 4–8cm in length. The sexes are separate.

Life cycle

The life cycle is similar to that of gastrointestinal roundworms, except that the females produce eggs containing the first stage larvae (L_1). These hatch soon after either in the lungs, or during the process of being coughed up, swallowed and then passed through the infected animal's digestive tract.

Ultimately, hatched lungworm L_1 are excreted onto pasture in freshly voided faeces. These larvae are characteristically sluggish in their motion and their intestinal cells are filled with dark brown food granules. These pre-parasitic larval stages do not feed. Under optimal conditions the infective L_3 stage is reached within five to seven days, although at lower temperatures this process may take several weeks.

The L_3 leave the faecal pat to reach the herbage through their own movements or by airborne spread utilising the fungus *Pilobolus*, commonly found growing on the surface of cow pats.

Larvated egg containing L_1.



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After ingestion, L_3 larvae first pass through the rumen, penetrate the intestinal wall and after about a week, travel to the lungs via the lymphatic system. Here, they first enter the alveoli and migrate upwards through the airways as they mature into adults. This typically occurs between three and four weeks post-infection. Fully mature, patent adults are normally found in the main stem bronchi and trachea.

Section 4: Development of the disease and clinical signs

Development of the disease

Lungworm infection is characterised by inflammation of the airways and pneumonia and is associated with two main diseases. Inflammation of the airways (parasitic bronchitis) is characterised by the presence of large numbers of adult worms in frothy white mucus in the bronchi. The presence of collapsed areas around infected bronchi (parasitic pneumonia) is caused by the aspiration of eggs and L_1 into the alveoli.

Most of the major clinical signs occur during the pre-patent and patent phases and are caused by primary parasitic pneumonia, as well as opportunistic secondary bacterial pneumonias.

The development of inflamed airways and pneumonia result in coughing and increased breathing rates (tachypnoea). Partial or even complete blockages of larger airways, caused by heavy burdens of adult parasites, lead to severe breathing difficulties (dyspnoea), with affected animals showing obvious signs of distress, such as open mouth breathing and

Over the course of a clinical infection, four main stages can occur:

1. The **Penetration Phase** (days 1–7 post infection) during which the infective L_3 stage penetrate the intestine of the host and migrate to the lungs
2. The **Pre-patent Phase** (days 8–25 post infection) during which larvae develop to adults in the lungs
3. The **Patent Phase** (days 26–60 post infection) when the worms are mature adults and producing eggs
4. The **Post-patent Phase** or **Recovery Phase** (days 61–90 post infection). As the animal acquires immunity (or following treatment with anthelmintics), adult worms are killed and expelled and damaged lung tissue begins to heal

adoption of an 'air hunger' stance.

This increased respiratory effort can also lead to further lung damage, as air is forcibly drawn into the lung tissue rather than remaining in the airways (respiratory emphysema). Fever may occur when there is secondary bacterial infection.

These signs are also commonly accompanied by varying degrees of anorexia, leading to loss of weight and condition. The severity and duration of signs are influenced by a range of factors including the number of L_3 ingested and the rate of their ingestion, along with the age and nutritional status of the infected animal.

In severe outbreaks, an occasional cough may be heard from a week onwards. By the second and third weeks, coughing becomes widespread. Affected animals can rapidly lose condition. Occasionally, massive infection can cause severe and sudden difficulty in breathing in some animals, often followed by death in 24 to 48 hours.

Within any affected group, differing degrees of clinical severity are apparent:

- Mildly affected animals cough intermittently, particularly when exercised
- Moderately affected animals have frequent bouts of coughing at rest, with increased and laboured breathing. Squeaks and crackles are frequently heard over the posterior lung lobes when using a stethoscope
- Severely affected animals show severe tachypnoea and dyspnoea. They frequently adopt the classic 'air-hunger' position of mouth breathing with the head and neck outstretched, and the tongue stuck out each time they try to cough. There is usually a deep harsh cough, salivation, anorexia and sometimes mild fever. Often the smallest calves are most severely affected

Clinical signs

- Widespread coughing in grazing cattle
- Loss of condition
- Increase in respiratory rate (tachypnoea)
- Difficulty breathing (dyspnoea)
- Secondary bacterial infections and pneumonia

Adult cattle can develop both clinical signs of parasitic bronchitis and sub-clinical production limiting infections. This is because they have failed to develop adequate immunity due to lack of exposure earlier in life, or immunity has waned due to lack of re-exposure over subsequent grazing seasons.

Most animals gradually recover during the post-patent phase. Clinical signs begin to ease but can persist for weeks to months, due to the ongoing dissolution and expulsion of dead and dying parasites and the slowing effect this has on healing.

Occasionally, in heavily infected animals there can be a severe (often fatal) flare up of clinical signs, resulting from aspiration of dead and dying worm material into the alveoli (post-patent parasitic bronchitis). Furthermore, convalescing animals remain at risk from secondary bacterial infection, particularly if housed in a poorly ventilated building.

Due to the nature of infection and disease, lung tissues often heal imperfectly with permanent scarring, resulting in life-long impacts on health, welfare and productivity.

Section 5: Epidemiology

Dictyocaulus viviparus is widespread and a large number of herds in the UK are affected, although many do not show signs of disease. It is only when a combination of circumstances favourable to the parasite arises that there may be an outbreak of disease.

In endemic areas, lungworm infection typically persists from year to year in two ways:

Overwintered L_3

Whilst lungworm L_3 will often die off rapidly on pasture over the summer months, cooler, wetter conditions in autumn and winter allow their survival on pastures through to the next season in sufficient numbers to initiate early season infections.

High risk conditions

- Warm wet summers
- High stocking rates
- Lack of prior immunity

Carrier animals

Similar to some gastro-intestinal roundworm species, when acquired late into the grazing season, some lungworms will inhibit their development in the lungs and survive within their infected host over winter. They resume their development and become adult parasites the following year. Such 'carrier' animals do not generally show any sign of infection or disease, but act as an important source of pasture contamination for the following season. Yearlings are the most important carriers, but adult cattle may also fill this role.

In some cases, clinical signs occur in at-risk groups of animals at the start of a season, particularly if turned out on pastures heavily contaminated by carrier animals. However, more typically, early exposure to either overwintered larvae or those shed by carrier animals result in light initial infections.

Disease outbreaks typically occur in the latter half of the grazing season – June/July onwards. This is usually where high risk animals either remain on or are put onto pastures that become highly contaminated with a second generation of infective L_3. This may be following prior grazing by lightly infected animals, including both the at-risk animals and carrier animals. Such circumstances are notoriously difficult to predict in terms of location and timing.

Lightly infected animals typically show no obvious signs of infection or disease, meaning it is virtually impossible to identify them, or which pastures pose the greatest ongoing risk.

Even where pastures have been contaminated, the subsequent development and emergence of L_3 is heavily influenced by environmental conditions. This means the magnitude of risk can vary significantly from pasture to pasture, from farm to farm and from year to year.

If re-infection rates from pasture remain low, immunity develops as animals are exposed over a long period to a low level of infection. Faecal larval counts in such animals will also decrease as immunity develops, reducing pasture contamination and ongoing risk further. Under such circumstances, disease will probably never occur, but it is very likely that the parasite will be carried over into the next season.

Where large numbers of L_3 have developed in a field, the timing of their emergence from cow pats can vary significantly. This is influenced to a significant degree by moisture levels, in particular rainfall and the conditions which encourage growth of *Pilobolus* fungi. Both of these help to mechanically disperse L_3 onto surrounding herbage. Infective larvae may also be dispersed on boots, animals' feet and by birds. Once on pasture, L_3 are highly susceptible to desiccation and will survive for only a limited period in warm weather.

While wet summers are more favourable for lungworm transmission, outbreaks can still occur in dry seasons after a brief period of rainfall, where large numbers of L_3 are dispersed onto pasture en masse.

Current infection and modelling data suggest the conditions favouring lungworm transmission in the UK are changing. Some regions, notably the south and east of England, are becoming less favourable, whilst others are increasing or remaining stable – ie in the north and west.

These changing patterns further complicate risk forecasting and prediction. In particular, whilst unfavourable conditions reduce likelihood of transmission and disease risk overall, the corresponding waning of immunity in these regions may leave herds vulnerable to sporadic and severe outbreaks, if favourable conditions return periodically.

Key differences between lungworms and gut worms

- Clinical disease occurs at much lower levels of infection with lungworms compared to gut worms
- Generally, lungworms have a shorter free-living development and survival period on pasture
- Carrier animals are an important source of lungworm infection for younger animals
- Lungworm risk is not present on every farm every year, making it difficult to predict
- Immunity to lungworm is not acquired universally by all grazing cattle

Immunity

Immunity to *D. viviparus* infection is usually acquired following natural exposure. Consequently, in most cattle patent adult infections do not last longer than two to three months.

Immunity to lungworm infection in cattle has two major impacts:

1. It reduces the number of infective larvae reaching the lungs. This aspect of immunity is relatively short-lived and wanes after around four months where cattle are removed from infected pastures. Farmers need to be aware of this when returning previously

exposed cattle to heavily infected grassland
2. Killing of adult worms in the lungs. Once acquired, this aspect of the immune response is retained for approximately two years

Even in immune cattle populations, a proportion will continue to harbour light burdens and shed low numbers of larvae onto pasture. Whilst this is a potential source of pasture contamination and risk for immunologically naïve cattle, these carrier animals can also be an important source of ongoing low-level exposure. This will help maintain the overall immune status of a herd with known history of infection and disease.

Re-infection syndrome

Since immunity to the infective larval stages wanes more quickly – potentially over the winter housing period, this will often need to be re-acquired in the subsequent grazing season.

This rarely causes a problem at turnout, as levels of L_3 in the fields will be typically low. However, if such cattle are returned to heavily contaminated pasture, large numbers of larvae can reach the lungs where the longer-lasting immune response to adult and pre-adult stages will kill them. This can result in a severe hypersensitivity reaction and acute illness known as 're-infection syndrome.' Cattle affected by this may die without obvious signs of respiratory disease.

Parasitic bronchitis in adult cattle

Parasitic bronchitis is seen in adult cattle in a number of circumstances:

1. As a herd-level phenomenon, or in a particular age group, if they have failed to acquire or maintain immunity due to a lack of natural challenge over previous years. Notably, the use of long-acting macrocyclic lactones (MLs) to control PGE in first season grazing animals, may also prevent them from acquiring meaningful immunity to lungworm infections
2. Where individual, or small groups of cattle are introduced to a farm with a history of lungworm from a herd where it is not present. Such animals will have no immunity and will be fully susceptible to the disease.
3. Where adults are penned in a heavily contaminated calf paddock and the level of exposure overwhelms their immunity.

In observed outbreaks in adult cattle, associated clinical signs varied between 3% and 100%, with only a small number of deaths. Half the number of affected animals had patent infections.

Section 6: Diagnosis

In the face of a potentially serious outbreak and the consequent need for timely treatment, diagnosis of lungworm infection is often based on clinical signs and grazing history alone.

Since disease typically occurs in young calves at grass for the first time, all members of a group may be affected to some degree, meaning assessment of multiple animals can be useful in determining the overall level of risk and need for action.

Since lungworm larvae hatch from their eggs before being shed, standard flotation-based faecal worm egg count (fWEC) methods will not identify patent infections. Instead, the main parasitological method for confirming lungworm disease is by detecting L_1 stage larvae in freshly collected faecal samples using the Baermann's sedimentation technique.

In patent lungworm infections, where clinical signs of disease are evident, there are usually many larvae present in the faeces. A low number of larvae or negative larval counts do not exclude lungworm disease, because cattle may be in the pre-patent or

First stage larva



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post-patent phase, or they may have partial immunity due to previous exposure ie re-infection syndrome.

Examination of sputum can also allow identification of both eggs and larvae and can confirm the presence of infections one or two days earlier than a faecal Baermann's sedimentation test.

When sampling from a group of animals in the face of a potential outbreak, it is generally advised to sample the animals first identified as showing clinical signs, as they are more likely to have progressed to patency by the time of investigation.

The sensitivity of the Baermann's method is profoundly affected by the sample used. Ideally, this should be collected directly from the animal (rectally) or from a freshly voided sample lying on the ground.

The test should be carried out on the same day as sample collection. In older, stored samples, lungworm larvae may die off and/or fail to migrate out of the faecal sample effectively. Conversely, such samples may also become contaminated with the larvae of gut roundworms, that have subsequently developed and hatched from eggs present in the sample.

A faecal polymerase chain reaction (PCR) test is available for the detection of *D. viviparus* DNA from L_1 larvae in a pooled sample of five animals. Whilst this method requires external submission and longer turnaround time, this test is preferred where diagnosis is not time critical, and it is not possible to set up a Baermann's sedimentation test. Unlike the Baermann's method, the faecal PCR is not dependent on the active migration of live larvae out of the faecal sample.

In addition to diagnostic methods to detect lungworm infection directly, a blood serum enzyme-linked immunosorbent assay (ELISA) is available for the detection of host antibodies to *D. viviparus*, to determine whether animals have been exposed to infection.

This test is more sensitive and can detect infection at an earlier stage, from around three weeks post infection. Conversely, once an animal has acquired antibodies to lungworm, these persist for weeks or months after the infection has been cleared by treatment or the animal's own immunity. It is therefore important to consider the circumstances under which this test is used.

Testing untreated and previously unexposed youngstock at housing, or as they progress through their first grazing can deliver useful information. Testing adult cattle that have been treated periodically with anthelmintics would not.

Variants of the antibody ELISA tests have been developed and tested for use with bulk tank and pooled milk samples, to facilitate herd-level diagnosis and monitoring. However, at present these methods are not available as diagnostic options.

Parasite detection

First stage larvae present in fresh faeces vary between about 300 to 450 µm in length and 25µm in width, with the intestinal cells containing numerous brownish chromatin granules. The head is rounded, with no protruding anterior knob (cf. *D. filaria* in sheep and goats) and the tail terminates in a blunt point.

On post-mortem examination, it is important to cut along the bronchial tree. The worms can be seen in the bronchi and bronchioles. Their location and size are diagnostic.

Section 7: Treatment and control

Treatment

Most anthelmintics available for use in the UK are licensed as effective against developing fourth-stage larvae and adult *D. viviparus*.

Cattle with clinical disease should be treated as early as possible, as delays may lead to severely affected animals for which treatments may either not improve, or may exacerbate disease. This is through the aspiration of dead or dying worms, which block the lower airways and alveoli, causing induced post-patent parasitic bronchitis.

Treated cattle should be removed from infected pasture and transferred to clean pasture if available or housed in well-ventilated facilities.

For youngstock, whatever treatment is selected, it is advisable to divide affected calves into one of the following two groups, as the prognosis will vary according to the severity of disease:

1. Calves which are only coughing and/or show laboured breathing, are usually in the pre-patent stage of the disease or only have a small adult worm burden. Treatment should result in rapid recovery. Such calves may not have developed a strong immunity and therefore should not be returned to their previous fields – the source of their infection, after treatment. Where it is not possible to move these animals to safe grazing, an ML product with residual activity may be used to prevent re-infection (see Table 1).
2. Calves that are feverish and showing severe signs of lungworm infection should be kept indoors for treatment and observation. Antibiotics and anti-inflammatories may also be indicated in severe cases, as well as encouragement to eat and drink. Even after these calves have been treated, a proportion may not recover and others may remain permanently stunted.

As lungworm infections are increasingly reported in older animals and adult cows, there may be occasions where milking cattle require treatment. It is important under these circumstances to observe milk residues and withhold periods, in line with the product datasheet.

Recent evidence of treatment failure with ivermectin and moxidectin – both ML products, against *D. viviparus* in UK cattle, is indicative of emerging drug resistance. Where treatments are indicated and administered, it is strongly advised that their efficacy is established through further clinical assessments and/or diagnostics, such as post-treatment Baermann's sedimentation.

Based on *D. viviparus*' pre-patent period of 21–28 days, L_1 should not be present for at least this time after treatment, plus the stated period of residual activity of the product used. In a study where ML treatment failure was identified, live L1 larvae were observed in the faeces of treated animals 15 days post treatment.

Control

The low infection threshold for bovine lungworm – only hundreds of adult worms compared to tens of thousands for PGE, and rapid waning of host immunity, mean disease outbreaks are unpredictable.

Use of clean grazing strategies are less reliable than for other roundworm infections. Similarly, existing diagnostics are limited, particularly in the face of an emerging outbreak, where timely treatment is required.

Table 1: Residual activities of macrocyclic lactones (ML) against lungworm

Active	Route	Protection (days)
Doramectin	Injection	35
	Pour-on	42
Eprinomectin	Pour-on	28
	Injection	14
Ivermectin	Injection	28
	Pour-on	28
Moxidectin	Injection 10%	120
	Pour-on	42

When selecting the most appropriate products to use against lungworm, it is important to consider how these might impact on, or be impacted by, other worming requirements, such as programmes for gastro-intestinal roundworm control. Farmers should seek the advice of the vet or SQP or RAMA.

Whilst these challenges exist, it is possible to apply a number of basic principles to identify risk and help control disease on farms where lungworm is present.

- The best method of preventing parasite bronchitis in first season grazing calves is immunisation with lungworm vaccine (available until the end of 2027). The live attenuated vaccine is given orally to calves aged eight weeks or more. Two doses are given, four weeks apart and cattle should not be exposed to challenge for a further two weeks to allow immunity to develop. Dairy calves can be vaccinated successfully at grass, provided this is given before significant larval challenge and the animals have not received an anthelmintic treatment.
- Youngstock, particularly those in their first grazing season and adult cattle where previous exposure is unlikely, ie they have been bought-in, are at highest risk from clinical disease and should be monitored and managed carefully
- Early treatment, in the face of clinical disease is recommended to prevent severe illness and losses
- Older animals are likely to be at lower risk of disease, but continue to act as a source of pasture contamination
- Pastures and grazing such as hay or silage aftermaths, can be considered effective clean

grazing, but may become contaminated more quickly than would be the case for PGE
- In hot, dry weather, previously contaminated pastures may become relatively safe if left ungrazed for one to two months, but not where conditions are wet and cooler
- Existing diagnostic tests are unlikely to be useful in the face of an outbreak, but may be of benefit in establishing infection status and to determine whether animals are more likely to develop clinical disease or protective immunity in future
- The use of either long-acting ML products or regular repeat dosing with short-acting products, as part of a strategic dosing strategy for PGE, will also control lungworm effectively. However this approach will also interfere with the development of immunity to lungworm through natural exposure and the development of full immunity following vaccination. This will also increase the pressure for anthelmintic resistance

On farms where there is no history of lungworm infection, vaccination is not necessary. Instead, a policy of preventing its introduction should be pursued through rigorous quarantine and biosecurity measures.

Information on individual products is available in the NOAH Compendium of Data Sheets for Animal Medicines at www.noahcompendium.co.uk or from the product manufacturer. Duration of activity of products can vary widely. Always check the latest product data sheet and/or product label before advising or administering products.