

Control of lungworm 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 roundworms, liver and rumen fluke, ectoparasites and integrated parasite control.



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

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Section 1: Top 10 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 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, registered animal medicines adviser (RAMA) or suitably qualified person (SQP)

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. Once a vaccination programme is started, all first-year calves should be vaccinated, and this continued annually for each calf crop

6. Vaccination is not necessary in low prevalence regions, or on farms with no previous history of lungworm. This situation should be monitored as changes could lead to explosive disease outbreaks
7. If strategic anthelmintic programmes are used early in the season in first year grazing cattle, they will also prevent build-up of lungworm larvae on pasture over the grazing season. This may potentially limit exposure to lungworm larvae to such an extent that cattle remain susceptible to infection. So it may be advisable to consider vaccination prior to their second year at grass
8. Prognosis will vary according to the severity of the disease and deaths may occur despite anthelmintic treatment. In animals with respiratory distress, avoid orally administered anthelmintics
9. Severely affected animals may require additional supportive treatment, which should be discussed with the vet

Plan ahead

10. Work out a control strategy. Lungworm outbreaks can cause severe losses and even death in cattle. This can happen at relatively low pasture infection levels. Farms with a previous history of lungworm should consider vaccination as an integral part of their overall worm control strategy as part of herd health planning

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

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

This species mainly affects cattle but has also been reported in other ruminants such as deer.

The parasite is widespread, but infections are prevalent in wetter areas, particularly those in the west of Britain.

Losses in severe lungworm outbreaks in growing cattle can average £50–£100 per head and lost milk production in adult cattle may reach £3 per cow per day.

As with many other parasitic nematodes, infection is acquired by the ingestion of infective larvae from pasture. The epidemiology is complex, and outbreaks are often unpredictable.

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 herd. Death can occur in heavy infections.

Although dairy or dairy-cross calves are commonly affected by lungworm, autumn-born single-suckled beef calves are also susceptible when turned out to grass in early summer.

Spring-born suckled beef calves grazed with their dams until they are housed or sold, do not usually develop clinical signs, although coughing due to mild infection is common. However, typical disease may occur in weaned beef calves grazed until late autumn.

Animals exposed to lungworms usually develop immunity to re-infection. Lack of exposure in young cattle may result in clinical disease occurring when they are older due to lack of immunity.

Sudden heavy pasture larval challenge, due to prevailing weather conditions may lead to signs even if cattle have previously been exposed.

Key concerns of a lungworm outbreak

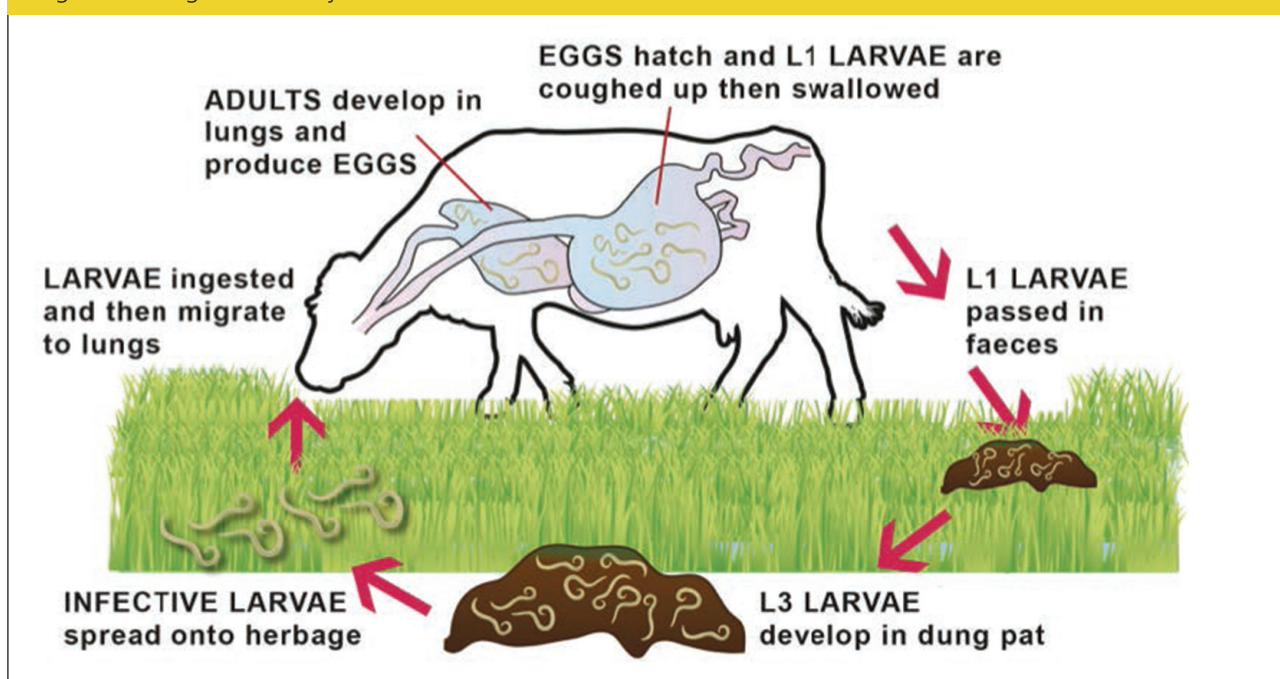
- Severe losses of performance
- Reduced milk yield in adult cows
- Unpredictable epidemiology
- Deaths can occur despite anthelmintic treatment

Younger animals are most susceptible to infection.



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 the gastrointestinal nematodes, except that eggs containing first stage larvae (L₁) are coughed up and swallowed and then hatch during the passage through the digestive tract.

The L₁ present in freshly-voided faeces are characteristically sluggish and their intestinal cells are filled with dark brown food granules. The pre-parasitic stages do not need to feed. Under optimal conditions the L₃ stage is reached within five to seven days but may take several weeks at lower temperatures.

The L₃ leave the faecal pat to reach the herbage through their own movements or by airborne spread utilising the fungus *Pilobolus*.

After ingestion, larvae penetrate the intestinal mucosa, then travel to the lungs and break into the

Larvated egg containing L₁.



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alveoli about one week after infection. These migrate to the airways, becoming mature adults between three and four weeks after infection, when L₁ can be detected in the dung. The adults are 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 bronchitis and pneumonia and typically affects young cattle during their first grazing season on permanent or semi-permanent pastures.

Most of the major clinical signs occur during the prepatent and patent phases and are caused by primary parasitic pneumonia.

The gradual development of bronchitis and pneumonia results in coughing and increased breathing rate, accompanied by varying degrees of anorexia, weight loss and laboured breathing. Fever may occur when there is a secondary bacterial infection. The severity and duration of signs relate to the number of larvae ingested and the rate of ingestion. The age of the host, climatic conditions and nutrition may also influence the course of the disease.

Infection is associated with two main diseases. A parasitic bronchitis characterised by the presence of large numbers of adult worms in frothy white mucus in the bronchi. Secondly, the presence of collapsed areas around infected bronchi. This is a parasitic pneumonia caused by the aspiration of eggs and L₁ into the alveoli.

In the course of a heavy primary infection, four stages can be distinguished:

1. The **Penetration Phase** (days 1–7 post infection) during which larvae penetrate the body of the host and migrate to the lungs
2. The **Prepatent Phase** (days 8–25 post infection) during which larvae develop in the lungs
3. The **Patent Phase** (days 26–60 post infection) when the worms are mature and producing eggs
4. The **Post-patent Phase** (days 61–90 post infection) which is normally the recovery phase after the adult worms have been expelled

During the post-patent phase, although the clinical signs are abating, the tissues are still inflamed, and the residual diseases may persist for weeks to months.

In some severely affected animals, there can be a flare-up of clinical signs, which is often fatal. This is thought to be due to the dissolution and aspiration of dead or dying worm material into the alveoli.

This clinical syndrome is often termed post-patent parasitic bronchitis. The other cause, usually in animals convalescing indoors, is a superimposed bacterial infection of the imperfectly healed lungs leading to acute interstitial pneumonia.

Clinical signs

In severe infections, an occasional cough may be heard at the end of the first week after infection. By the second and third weeks, coughing becomes widespread. Occasionally a massive infection can cause severe and sudden difficulty in breathing (dyspnoea) in some animals, often followed by death in 24–48 hours.

During the patent phase, the respiratory rate increases; affected animals cough frequently, and dyspnoea may occur. Affected animals can rapidly lose condition.

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

Clinical signs

- Widespread coughing in grazing cattle
- Loss of condition
- Increase in respiratory rate (tachypnoea)
- Difficulty breathing (dyspnoea)
- Reduced milk yield in adult cows

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

Most animals gradually recover, although a complete return to normality may take weeks or months.

However, a proportion of convalescing calves can suddenly develop severe respiratory signs, unassociated with fever, which usually terminates fatally one to four days later (post-patent parasitic bronchitis).

Adult cattle can be affected with parasitic bronchitis if they fail to develop adequate immunity, through lack of exposure to infective larvae as calves, or lack of subsequent re-exposure and are then grazed on heavily contaminated pastures in adulthood. This is sometimes first seen as a reduction in milk yield with subsequent coughing.

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.

Outbreaks are difficult to predict because primary infections are not often detectable. When there is an outbreak, it is usually due to the second generation of worms ingested from pasture, generally from June until November, but most commonly from July until September.

High risk conditions

- Wet summers
- Heavy stocking densities
- Lack of immunity due to low exposure to infective larvae

Larvae are susceptible to desiccation and survive on pasture for only a limited period. High summer temperatures limit larvae development and survival on pasture. In areas with high average summer rainfall, larvae may survive longer. Wet weather also promotes

the release of larvae from the faecal pat. As well as airborne spread on the spores of the *Pilobolus* fungus, L₃ may also be dispersed by mechanical means such as boots, animals' feet and birds.

As with parasitic gastroenteritis, there is a seasonal pattern of inhibited development in the lungs, with carrier animals acting as an important source of contamination of pasture in the following spring. In previously exposed herds, asymptomatic carrier animals may excrete a low number of larvae in the faeces. Yearlings are the most important carriers, but adults may also act as carriers and excrete small numbers of larvae without showing clinical signs.

In some cases, clinical signs in first year calves may occur at the start of the season when they are grazed on a pasture heavily contaminated by carrier animals. More typically, where early season infections result from overwintered larvae, there is an increase in faecal larval counts, which contaminate the pasture.

Faecal larval counts then usually decrease again in four to eight weeks as a result of animals developing immunity.

If re-infection rates from pasture remain low, disease will probably never occur, as immunity develops during the long period of low trickle infections. When disease does occur, it is often several months after primary exposure to infection.

In endemic areas, lungworm infection may persist from year to year in two ways:

1. Overwintered L₃ may survive on pasture from autumn until late spring in sufficient numbers to initiate infection or occasionally to cause disease.
2. Carrier animals: small numbers of inhibited worms, which subsequently recommence development, can persist in the bronchi particularly in yearlings, until the next grazing season. They are more important than other roundworm species carriers, since they will contribute to pasture contamination and the L₃ dose required to cause disease in other animals is low.

Current modelling studies predict that conditions for lungworm transmission are likely to be less suitable in the south and east of the UK but remain similar in the north and west. This could manifest as lower levels of disease in the south and east, but the subsequent falling levels of immunity may increase vulnerability in some herds.

Key differences between lungworms and gut worms

- Generally, lungworms have a shorter free-living development period and larval survival
- Carrier animals are an important source of lungworm infection
- Appearance of lungworm disease is unpredictable and low-risk grazing is not possible

Immunity

A strong immunity to *D. viviparus* infections is usually rapidly acquired under field conditions. In most cattle, patent infections do not usually last longer than two to three months because of immunity, which starts to develop from ten days after a heavy infection.

The immune response to lungworm has at least two major components:

1. Reduction in numbers of larvae reaching the lungs. The memory of this response is short at around four months and can wane quickly in cattle removed from infected pastures. Farmers should be aware of this when returning previously exposed cattle to heavily infected grassland
2. Destruction with adult worms that have reached the lungs. Once acquired this type of response can be remembered for about two years.

Even in exposed cattle, 'sterile immunity' is unlikely and a proportion of immune cattle will continue to shed low numbers of larvae to pasture. This can be beneficial in maintaining the immune status of the herd by providing a sustained mild challenge.

Re-infection syndrome

Immunity to larval entry can wane within months, eg during housing. This rarely causes a problem at turnout as levels of larvae on pasture are low. However, if cattle are returned to heavily contaminated pasture, then large numbers of larvae can reach the lungs.

The longer-lasting lung immune response will kill large numbers of young mature worms. This is called 're-infection syndrome'. The rapid die-off of young worms causes a hypersensitivity reaction and acute illness. Cattle may die without obvious signs of respiratory disease.

Parasitic bronchitis in adult cattle

Parasitic bronchitis is seen in adult cattle under two circumstances:

1. As a herd phenomenon, or in a particular age group within a herd, if they have failed to acquire immunity through natural challenge in earlier years

2. Disease is occasionally seen where an individual adult is penned in a heavily contaminated calf paddock.

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

Diagnosis is based on the clinical signs and grazing history. The disease occurs typically in young calves at grass for the first time when all members of a group may be affected to some degree. Usually, the clinical signs, the time of the year and a history of grazing on permanent or semi-permanent pastures, are sufficient to enable a diagnosis to be made.

The main parasitological method of confirming lungworm disease is by detecting the L₁ stage in faecal samples using the Baermann technique. Examination of sputum for eggs and larvae is rapid and sensitive, and the presence of patent infections can often be detected one or two days earlier than with faecal examination.

In patent lungworm infections where clinical signs of disease are evident, many larvae are usually present. A low number of larvae or negative larval counts do not exclude lungworm disease because cattle may be in the prepatent or post-patent phase, or may have been previously exposed (re-infection syndrome).

Consider sampling the first animals showing clinical signs, as they are more likely to have progressed to patency by the time of the investigation.

Flotation techniques have limited use in detecting lungworm larvae in faeces because they are far less sensitive than the Baermann test.

Bulk milk antibody ELISA is of use to assess exposure at a herd level. The sensitivity of the ELISA test at detecting patent infections is low, but can be used to detect sub-clinical infections and therefore potential production losses, or to predict if a clinical outbreak of dictyocauliosis might occur. This is particularly relevant if routine monitoring of bulk milk samples changes from negative to positive.

Some recent results suggest taking a pool of milk

First stage larva



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from first lactation heifers can be a more sensitive way of detecting exposure to lungworm.

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.

An ELISA test can be used to detect serum antibodies to *D. viviparus*. As sero-conversion does not usually occur until four to six weeks post-infection, animals with prepatent infections (or re-infected animals), may be sero-negative.

This might be an option in beef herds, but the number of cattle that need to be sampled to pick up an infected animal within the herd if prevalence is low, is usually cost prohibitive.

Section 7: Treatment and control

Treatment

All available anthelmintics are highly effective against developing fourth-stage larvae and adult *D. viviparus*. To date, there are no confirmed resistance issues.

Affected cattle should be treated as early as possible because severely affected animals may either not respond, or disease may be exacerbated, as dead or dying larvae block the lower airways and alveoli.

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

Whatever treatment is selected, it is advisable to divide affected calves into two groups, as the prognosis will vary according to the severity of disease.

Calves which are only coughing and/or with laboured breathing, are usually in the prepatent stage of the disease or have a small adult worm burden, so treatment should result in rapid recovery. Such calves may not have developed a strong immunity and should not be returned to the grazing, which was the source of infection, after treatment.

If this is not possible, a macrocyclic lactone (ML) may be used as the residual effect prevents re-infection for an extended period (See Table 1).

Calves that are feverish and showing severe signs of lungworm infection should be kept indoors for treatment and observation. Even when affected calves are treated with an anthelmintic, a proportion may not recover, and others may remain permanently stunted. In addition to anthelmintics, severely affected animals may require antibiotics if they have a fever, anti-inflammatories and hydration if they are not drinking.

As lungworm infections are increasingly reported in older animals and adult cows, there may be occasions where milking cattle require treatment. However, many of the available anthelmintic products are not licensed for use in lactating dairy cattle. To prevent anthelmintic residues in milk and meat, label recommendations for withdrawal times must be observed.

Control

The low infection threshold for disease and rapidly waning immunity means lungworm outbreaks are

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

Active	Route	Protection (days)
Doramectin	Injection	35
	Pour-on	42
Eprinomectin	Pour-on	28
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 any other worming requirements. Changes to strategic anthelmintic programmes for gastrointestinal nematode control may have an impact on lungworm control. Farmers should seek advice from their vet, RAMA or SQP.

unpredictable. Consequently, the use of clean grazing strategies is less reliable than for other roundworm conditions.

The best method of preventing parasite bronchitis in first season grazing calves is immunisation with lungworm vaccine. 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.

The residual effects of long-acting endectocides or sustained release bolus preparations will interfere with the development of immunity following lungworm vaccination. Therefore, avoid vaccination during the period of their activity, and do not use until 14 days after the second vaccine dose.

Dairy calves can be vaccinated successfully at grass provided the vaccine is given prior to encountering a significant larval challenge and the animals have not recently received a long-acting anthelmintic or bolus device.

Re-infection by pasture larval challenge is necessary for boosting immunity. Vaccination will prime the first stage of the immune response (to incoming larvae) and the exposure at grass allows development of immunity to adult worms in a controlled fashion.

If cattle are vaccinated but not exposed to field challenge after some months, re-vaccination should be considered.

Vaccination is not necessary in low-prevalence regions of lungworm disease or on farms with no previous history of lungworm. In such herds, a policy of preventing the introduction of infection should be rigorously enforced by good quarantine measures.

Infection status, particularly in dairy cows can be monitored using ELISA antibody testing.

Although vaccination is effective in preventing clinical disease, it does not completely prevent establishment of small numbers of lungworms. Consequently, pastures may remain contaminated, albeit at a low level. For this reason, it is important that all calves on farms where lungworm is endemic, should be vaccinated whether they go to pasture

in the spring or later in the year. A calf vaccination programme must be continued annually.

Control of parasitic bronchitis in calves can be achieved by use of prophylactic anthelmintic regimens either by strategic early season treatments or by the administration of rumen boluses, in much the same way as for the control of parasitic gastroenteritis.

A potential downside of these measures is that, through rigorous control in the first grazing season, exposure to lungworm larvae may be so limited that cattle remain susceptible to infection. In such situations, it is advisable to consider vaccination prior to the second year at grass.

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.