

Control of lungworm 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, parasitic gastroenteritis, 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 lungworm (parasitic bronchitis)

Identify Risk	1. Lungworm outbreaks are unpredictable , but are more prevalent in wetter, western areas of Britain. 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, at grass.
	3. Animals exposed to lungworms 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. Quarantine and treat all incoming cattle for roundworms and fluke (see COWS Liver Fluke section tip 9 and the COWS Integrated Parasite Control chapter). Bought in calves or adult cattle may introduce lungworm onto a farm. Most anthelmintics used for control of gut roundworms are effective against lungworms. Check with your vet or Suitably Qualified Person (SQP).
Treat Appropriately	5. Routine vaccination should be considered for calves born into herds with an identified lungworm problem or when there is a previous history of lungworm on the farm. Once a vaccination program is started, all first year calves should be vaccinated and this continued annually for each calf crop.
	6. Vaccination is not justified in low-prevalence regions , or on farms with no previous history of lungworm.
	7. Anthelmintics can be used strategically in first-year grazing cattle to prevent build-up of lungworm larvae on pasture over the grazing season. As overuse of anthelmintics may potentially limit exposure to lungworm larvae to such an extent that cattle remain susceptible to infection, 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 your 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: Lungworm (Parasitic Bronchitis, Dictyocaulosis, “Husk”, “Hoose”)

Parasitic bronchitis (husk) 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 more prevalent in wetter areas, particularly those in the west of the British Isles.

Losses in severe lungworm outbreaks in growing cattle can average £50-£100 per head and lost milk production in adults 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.

Symptoms are most commonly seen in first-year grazing cattle in late summer and autumn, but can occur earlier in the year and in older animals. The most characteristic clinical sign of lungworm infection is widespread coughing within a herd. Mortality occurs in heavy infections.

Key Concerns

- Severe losses in disease outbreaks
- Reduced milk yield in adult cows
- Complex, unpredictable epidemiology
- Deaths can occur despite anthelmintic treatment

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

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

Animals exposed to lungworms usually develop resistance to re-infection. Lack of exposure in young cattle may result in clinical disease occurring in older cattle.

Sudden heavy pasture larval challenge, due to prevailing weather conditions, may lead to clinical signs of re-infection husk (see section 5).

Dairy calves are most commonly affected



Section 3: The parasite

Adult worms are slender and 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 passage through the digestive tract.

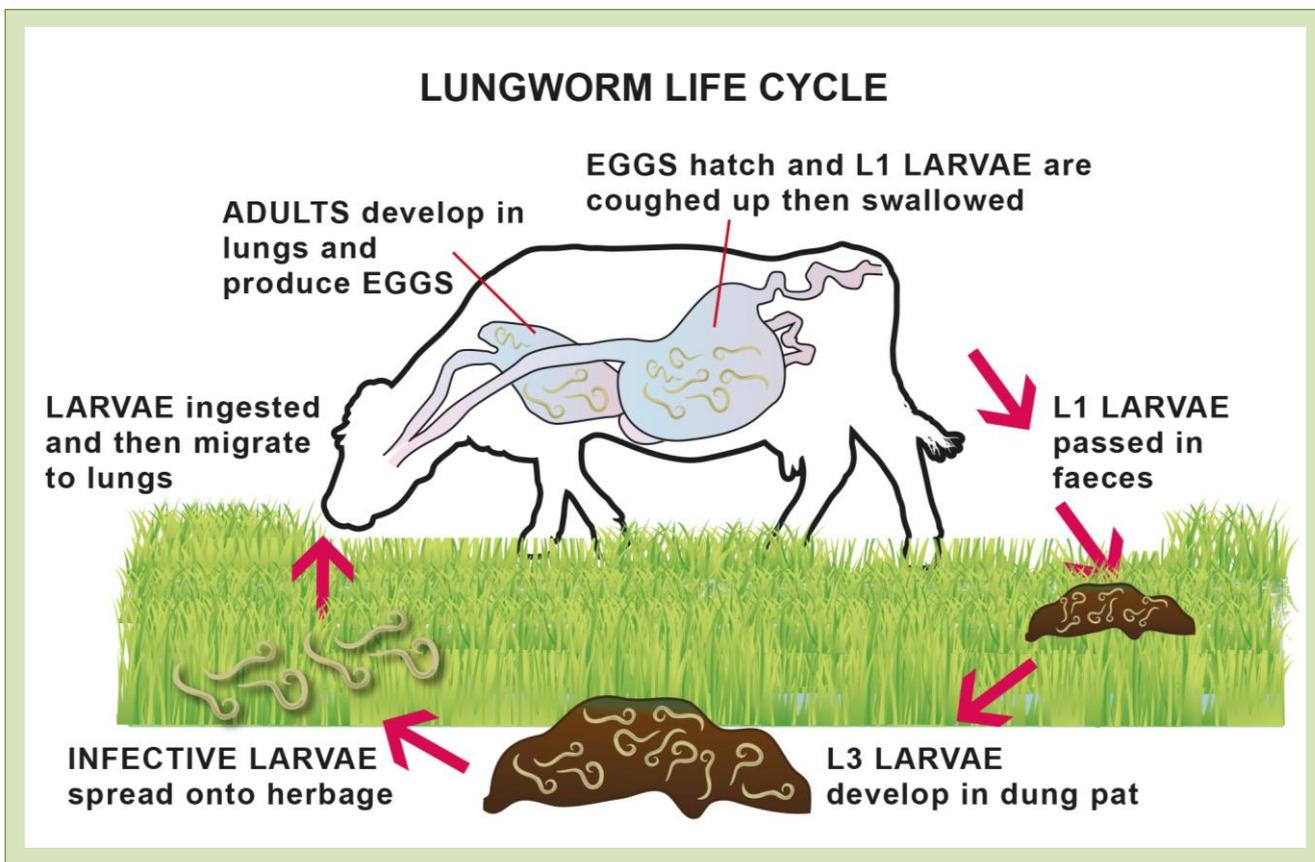
The L₁ present in freshly-voided faeces, are characteristically sluggish and their intestinal cells are filled with dark brown food granules. The pre-parasitic stages do not need to feed. Under optimal conditions the L₃ stage is reached within 5-7 days, but this may take longer depending on the environmental conditions. The L₃ leave the faecal pat to reach the herbage through their own movements or by airborne spread utilizing the fungus, *Pilobolus*.

Larvated egg containing L₁



Photo courtesy of Prof Mike Taylor

After ingestion, L₃ penetrate the intestinal mucosa and pass to the mesenteric lymph nodes where they moult. The L₄ then travel via the lymph and blood to the lungs, and break out of the capillaries into the alveoli about one week after infection. These migrate up the lungs and moult to L₅, becoming mature adults between three and four weeks after infection, when L₁ can be detected in dung. The adults are found in the main stem bronchi and trachea.



Section 4: Pathogenesis and Clinical Signs

Pathogenesis

Lungworm infection is characterised by bronchitis and pneumonia and typically affects young cattle during their first grazing season on permanent or semi-permanent pastures.

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

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

(p.i. - post infection)

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 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 disease.

Infection is associated with two main lesions. A parasitic bronchitis characterised by the presence of large numbers of adult worms in frothy white mucus in the bronchi; and 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.

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

In about a quarter of severely affected animals, there can be a flare-up of clinical signs, which is frequently fatal.

The aetiology is unknown, but 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 and 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 over the posterior lung lobes and are heard on auscultation.
- Severely affected animals show severe tachypnea (an increase in breathing rate) and dyspnea. They frequently adopt the classic 'air-hunger' position of mouth breathing with the head and neck outstretched, and the tongue is 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)
- Reduced milk yield in adult cows

On auscultation of the lungs, the inspiratory and expiratory sounds are harsh with varying degrees of emphysematous crackling also audible.

Most animals gradually recover, although a complete return to normality may take weeks or months. However, a proportion of convalescing calves suddenly develop severe respiratory signs, unassociated with fever, which usually terminates fatally 1-4 days later (post-patent parasitic bronchitis; see [Pathogenesis on page 5](#)).

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

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

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 occur from June until November; most commonly from July until September.

Larvae are susceptible to desiccation and survive on pasture for only a limited period. High summer temperatures limit larvae development and survival on pasture, whereas 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.

In some cases, clinical signs in first year calves may occur at the start of the grazing season when they are grazed on a

High Risk Conditions

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

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 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:

- (a) Overwintered L₃ may survive on pasture from autumn until late spring in sufficient numbers to initiate infection or, occasionally, to cause disease.
- (b) Carrier animals: small numbers of inhibited worms, which subsequently recommence development, can persist in the bronchi, particularly in yearlings, until the next grazing season.

Key differences between lungworms and gutworms:

- Generally lungworm have a shorter free-living development period and larval survivability
- Carrier animals are the most 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 10 days after a heavy infection.

If re-infection by exposure to infective pasture larvae does not occur, immunity declines from about three months post infection. Re-

infection is thus necessary to maintain immunity.

Generally only calves in their first grazing season are clinically affected because older animals have acquired immunity on farms where the disease is endemic.

Re-infection Syndrome

Lungworm challenge in older cattle, which have acquired immunity to *D. viviparus*, is not usually associated with clinical signs.

Occasionally, however, coughing may occur in previously immune animals which are exposed to massive larval challenge, which

might occur on pasture recently vacated by calves suffering from clinical husk.

It can be difficult to differentiate this syndrome from the early stages of a severe primary infection in non-immune cattle.

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 symptoms varied between 3% and 100% with only a small number of deaths. Half the number of affected animals had patent infections.

There appears to be no relationship between previous anthelmintic treatment or vaccination history and the appearance of clinical signs.

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).

Because they are far less sensitive than the Baermann technique, flotation techniques have limited use in detecting lungworm larvae in faeces.

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 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. Serological cross-reactivity with some gastrointestinal nematode species, and

First stage larvae



Photo courtesy of Prof Mike Taylor

the fact that antibody titres persist for 4–7 months, may complicate interpretation by producing false positive results. An increased number of eosinophils may be detected in an EDTA blood sample and is supporting evidence for *D. viviparus* infection, but not specific.

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 reported resistance issues.

Affected cattle should be treated as early as possible because severely affected animals may either not respond, or symptoms 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.

Those 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 of these animals should result in rapid recovery. Calves in this category may not have developed a strong immunity and should not be returned to grazing which was the source of infection after treatment.

If this is not possible, a parenteral macrocyclic lactone may be used as the residual effect prevents re-infection for an extended period (see table).

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

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 1%	42
	Injection 10%	120
	Pour-on	42

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.

Selecting the most appropriate products may need to consider other worming requirements, so farmers should seek advice from their vet or SQP.

Control

The unpredictability of disease due to lungworm infections precludes the use of clean grazing strategies for control.

The best method of preventing parasitic bronchitis in first-year calves is immunization with lungworm vaccine. The live attenuated vaccine is given orally to calves aged eight weeks or more. Two doses are given at an interval of four weeks and, to allow a high level of immunity to develop, vaccinated calves should be protected from challenge until two weeks after their second dose.

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 is not justified 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 (see Section 5 on Quarantine in the COWS Integrated Parasite Control chapter).

Control

- Vaccinate calves in herds with a history of lungworm
- Vaccination is not justified in low-prevalence regions, or on farms with no history of lungworm
- Treat all purchased cattle to prevent introducing lungworm to husk-free herds

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 and 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.