# HUSK, LUNGWORMS AND CATTLE

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**Alastair Hayton** discusses how best to manage the lungworm issue, and details the causes behind a disease rapidly increasing in prevalence

**AS with many parasitic infections, there has been a rise in lungworm prevalence in the UK and, likewise, an increase in the percentage of adult lungworm cases as a proportion of overall cases.**

It is a highly pathogenic disease, causing severe losses in affected stock. Disease control planning is an important aspect of a herd's health management. This planning is made all the more difficult in that its presentation on a farm is unpredictable, with potential outbreaks throughout the grazing season. This unpredictability is due to the relatively small numbers of larvae required to cause disease, the difficulty in knowing when initial infections will be acquired and how high this challenge will be. This article seeks to address the underlying rationale for control programmes and outbreaks.

## Epidemiology

The *Dictyocaulus viviparus* worm causes the disease, which has a similar life cycle to gutworms. Adult worms in the lungs produce eggs (each worm can produce several thousand eggs) that hatch almost immediately.

The larvae from these eggs (L1) migrate up the trachea, are swallowed and consequently passed out in the faeces, thereby contaminating pasture. L1 can develop to the infective L3 stage in faeces within one week, so infection levels can develop very quickly.

The L3 larvae are spread out from the dung via water (rain) and by the sporangia of the *Pilobolus* fungus, which allows them to spread further. Both fungal and larval development are very dependent on having warm, wet conditions and, when these conditions transpire, there can be a very rapid build up of infective larval levels on pasture. Conversely, periods of drought will severely reduce pasture contamination levels.

Cattle ingest the larvae, which then penetrate the gut wall, before migrating to and travelling through the lungs until they reach the airways, where they mature into adults. The period from infective larvae ingestion to egg production is three to four weeks. The passage of larvae through the lung, the presence of adults in the airway and the host's subsequent inflammatory response cause the clinical signs typical of this disease.

The cycle of pasture contamination is similar to gutworm. The initial contamination presents in the spring, arising from the survival of overwintered L3 or as a result of larvae being passed by carrier animals at turnout. Non-immune animals grazing this pasture are then responsible for cycling the parasite (autoinfection), with a subsequent rapid build up of pasture contamination as long as there are favourable external conditions.

The disease can occur where the primary infections are high or, in the second generation, where environmental conditions are favourable to larval survival and development at pasture.

Lungworm can cause severe respiratory disease, with associated production losses such as weight loss, decreased milk yield and reduced fertility. In addition, in a very small percentage of affected animals, a hypersensitivity reaction to the parasite can be seen – leading to acute respiratory disease and/or death.

## Immunity and disease outbreaks

All cattle are at risk until they develop immunity, and infection with relatively few lungworms can cause clinical disease. Immunity is acquired following sufficient exposure to the infective L3 challenge, but the rate of development varies between animals. This can be slower due to continuous exposure to low-to-moderate numbers of larvae over several lungworm generations, or relatively fast when exposed to a high larval challenge in a single lungworm generation. However, this immunity is also relatively rapidly lost.

In the absence of a lungworm challenge, protective immunity can be lost as early as six months. "Re-infection syndrome" occurs in animals that have acquired immunity and then suddenly face a massive larval challenge. This challenge reaches the lungs, where larvae are killed by the immune reaction. It can be difficult to differentiate this from a severe primary infection, where adult lungworms are present and faecal larvae can be detected.

This dynamic is important, as it implies the importance of ensuring a continuous "controlled

exposure” to the parasite so that immunity can be developed and maintained while avoiding the potential for subclinical and/or clinical disease to occur. This controlled exposure is all the more difficult given the previously discussed rapid build up of infection that can be seen at pasture when given the right conditions.

## Stock scenarios where outbreaks are likely

Being able to recognise the scenarios can help identify when stock may be at risk, why disease outbreaks may occur and help plan control strategies. As with most diseases, these risks can be divided between those affecting the development of immunity and those likely to increase the levels of exposure.

## Causes of failure to acquire immunity

• **Failure to vaccinate stock.** Vaccination will add predictability to an unpredictable disease, and will guarantee that animals will have some exposure to the disease. Vaccination, however, does rely on subsequent pasture exposure for full immunity to develop and persist.

• **Adverse environmental conditions resulting in poor larvae survival and dispersal.** Prolonged periods of dry weather will reduce pasture numbers.

• **Anthelmintic use.** There is a reasonable concern that where pasture contamination is already low, such as when environmental conditions are unfavourable to larval development and survival, an anthelmintic control programme may lead to very low levels of pasture L3, thus reducing the stimulation of immunity to lungworm. Conversely, inadequate anthelmintic usage at high exposure levels could lead to disease. As it is impossible to predict usage levels prior to deciding what anthelmintic regime is to be used and the likely level of challenge (weather dependent), this makes a choice of strategy very difficult.

Trials using ivermectin injections and sustained releases of ivermectin boluses at three, eight and 13 weeks post-turnout have shown that satisfactory immunity was gained to lungworm on these systems, so it is impossible to be dogmatic on the system choice. The salient point should be that, whatever system is used, performing serological tests on stock prior to subsequent turnout (particularly prior to the second grazing system) is a sensible precaution for ensuring the previous grazing season has allowed adequate immunity to develop.

• **Prolonged housing.** Immunity wanes from as early as six months without exposure to lungworm.

• **Low stocking rates.**

## Causes of increased lungworm exposure

• **Introduction of infection in a previously naïve herd.** For obvious reasons, and where farms are thought to be free of lungworm, biosecurity measures should be discussed.

• **Introduction of naïve animals into an infected herd.** It is important to be aware that bought-in cows with no previous exposure will be susceptible to disease on either a farm that has a history of disease or on one where youngstock are routinely vaccinated. This is because some irradiated larvae in the vaccine are able to develop to adult stages, leading to pasture contamination. Furthermore, while older cattle in the established herd may have developed immunity, disease can still occur in these animals when new animals are added, as the naïve animals cause a significant build up of larvae on the pasture – thereby overwhelming their immunity.

• **Delayed pasture challenge.** Outbreaks in the latter part of the grazing season are common occurrences, when a combination of environmental conditions and the cessation of anthelmintic control (for example, when bolus delivery systems have finished) allow an open window for infection to develop. This is particularly the case where previous pasture contamination has kept levels low.

• **Inadequate anthelmintic control at pasture.** Failure to worm stock adequately can obviously increase the risk of larval build up on pasture.

• **High stocking rates.**

• **Warm, wet weather.**

## Control

Control methods differ from those for gutworms, in that there is an effective vaccination against lungworm to help prevent disease.

This is given to susceptible stock prior to turnout, and relies on the animals subsequently being exposed to lungworm larvae during grazing to allow them to develop full immunity. As such, anthelmintic control needs to be planned so that exposure windows are able to develop, such as with pulse-release devices or the three, eight and 13-week ivermectin regimes. Failure to expose animals in this way may result in a lungworm risk in subsequent years.

If vaccination is not practised and reliance is placed on strategic worming, then – as with gutworm – it should be borne in mind that the potential for disease can occur right through the grazing season. Consequently, control will need to be applied throughout the grazing period.

When there is a concern that lungworm immunity may have waned in any age of stock (for instance, when stock show very low or absent levels of antibody to lungworm prior to turnout), it may well be worth considering vaccinating these animals, regardless as to previous vaccinations.

## Treatment

Lungworms are susceptible to the three major classes of benzimidazoles, levamisole and macrocyclic lactones (MLs).

MLs are more effective against a wider range of developmental stages than levamisole. This is important, because a failure to remove all lungworms can result in the potential for treated animals to become carriers, and a recurrence of disease may be seen following the continued development of larvae in the host. Levamisole is rapidly absorbed and eliminated from the body (within 24 hours). The persistent activity of MLs means that, following treatment, subsequent rapid re-infection is avoided when cattle have to remain on infected pastures.

For a long time, levamisole has been recommended as the treatment of choice for lungworm. This follows observations of a relatively small cattle population that showed there were better post-treatment survival rates in severely affected animals in the levamisole-treated group when compared to benzimidazoles. This was attributed to the nature of the action of levamisole, which results in paralysis of the worms. This allows the worms to be removed by local physiological mechanisms, rather than the more severe immunological reactions generated when the worms were killed by benzimidazole.

Whether using MLs (which also result in the death of the parasite) would also result in similar observations has not been elucidated. MLs are slower acting in their actions and, therefore, may not generate such levels of adverse reaction as opposed to benzimidazoles and levamisole, which act much faster.

Beyond anthelmintic treatment, in moderately to severely affected animals adjunctive treatment with NSAIDs and antibiosis is required to reduce inflammation and secondary infections.

In the face of an outbreak, all stock should be treated with anthelmintics, as it is impossible to differentiate between prepatent and patently affected animals, and it will also reduce the potential for further pasture contamination. Regardless of treatment choice, death can occur in severely affected animals if sufficient damage has occurred. In outbreaks affecting lactating cows, the other important issue – when selecting an appropriate anthelmintic – is the requirement to avoid a milk withdrawal, which would currently be only applicable to one commercial product.

## Diagnostics

Faecal larval counts are still the standard diagnostic tool for lungworm and provide for a high specificity and moderate sensitivity, with reasonable indications of the infection levels, although it will not detect infections in the prepatent phase.

Individual animal serology can also be performed. This is useful to identify whether exposure to

lungworms has occurred, with high levels suggesting recent and/or high levels of exposure. However, it cannot differentiate between active and historical infection, as antibody levels can remain elevated for several months following exposure.

A bulk milk ELISA test for lungworm is in development. Perhaps the most useful information arising from negative bulk milk ELISA results would suggest either a prepatent or postpatent infection status, or that no exposure to *D viviparus* infection had occurred during the past six months. Therefore, there is probably no protective immunity present, which – given the rise in adult infections and the massive pathogenicity of lungworm – could provide farmers and vets with very useful information as to appropriate control measures.

## Further reading


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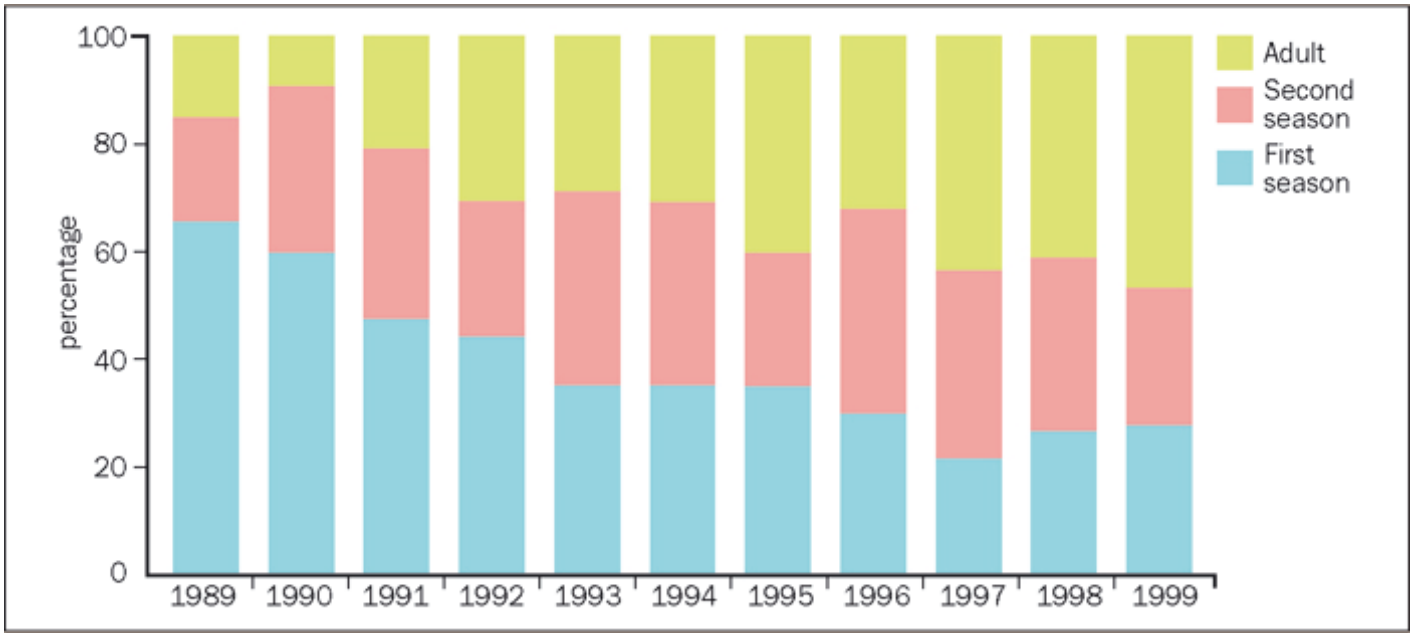
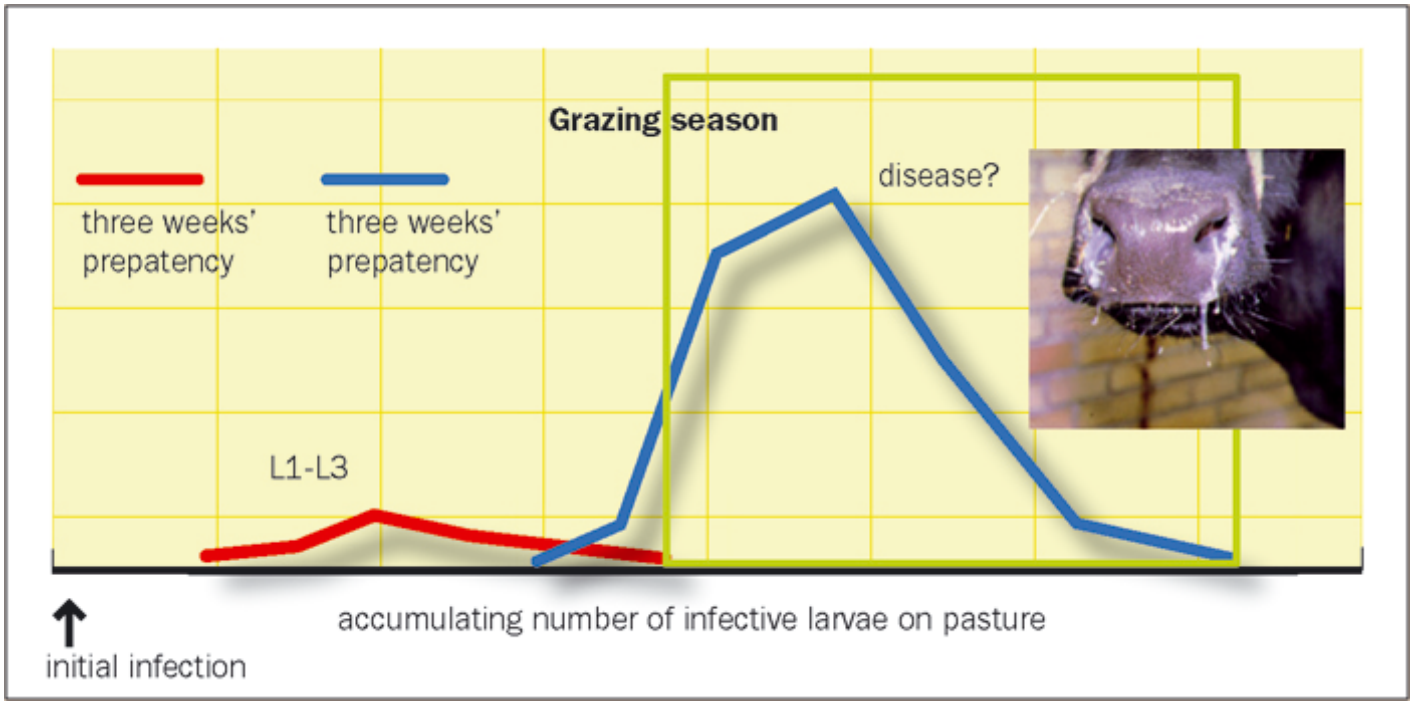
***In the absence of a lungworm challenge, protective immunity can be lost as early as six months.***

Photo: SXC/ WATJE.




***The rise in importance of adult infections of lungworm in the UK.***

Image source: VIDA.




***Schematic of lungworm epidemiology.***

Image source: SCHENIDER T.