Lungworm – the problems

In this article, Ruth Vernon BVSc CertCHP MRCVS will look at immunity, diagnosis and possible risk factors that have changed the clinical picture of lungworm seen today

Parasitic bronchitis, also known as lungworm or husk, is an economically important disease of cattle caused by *Dictyocaulus viviparus*. This parasite is widespread but tends to be more prevalent in areas with higher rainfall. The common clinical sign is coughing in grazing cattle, but there can also be significant impact on milk yield, loss in body condition and, in the worst cases, mortality, if there is a heavy infection.

Years ago, lungworm was usually seen by veterinary surgeons in cattle during their first grazing season and commonly seen in wetter areas. However, more commonly these days, lungworm outbreaks are often seen in adult cattle, where the effects of clinical lungworm can have a catastrophic impact on herd milk yields in affected cattle. A lungworm outbreak can be very costly and has been estimated at approximately €155 per adult cow in the herd, with reduced milk yield losses averaging 4kg/cow/day making 50% of the total cost of disease. Subclinical disease has also been shown to cause loss in milk production of around 0.5kg/cow/day.

EPIDEMIOLOGY

Lungworm outbreaks are difficult to predict, as the early infections are difficult to detect and are usually a result of the second generation of worms being ingested. Lungworm larvae are susceptible to desiccation and, therefore, do not survive well on pasture, except at times of higher rainfall when the larvae can live for a longer period of time.

HIGH-RISK CONDITIONS FOR LUNGWORM

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

LIFECYCLE

The lifecycle of lungworm is similar to gutworm, except that the first-stage larvae (L1) are coughed up, swallowed, and then voided in faeces. In optimal weather conditions, the infective L3 stage is reached on the pasture within five to seven days. The L3 leave the faecal pat to reach the herbage either by their own movement or with the help of the fungus, Pilobolus. When L3 are ingested by an animal, they penetrate the intestinal mucosa and travel via the lymphatic system to the lung. In the lungs, they establish and mature to L5, which takes about three to four weeks after ingestion of L3. The adults are found in the bronchi and trachea, where they are very prolific at laying eggs.

DISEASE COURSE IN A HEAVY LUNGWORM INFECTION

1. Penetration phase: one to seven days post infection when larvae enter the body and penetrate the lungs.
2. Pre-patent phase: eight to 25 days post infection during which the larvae develop into adults in the lungs.
3. Patent phase: 26-60 days post infection when the worms are mature and producing eggs.
4. Post-patent phase: 61-90 days post infection, which is normally the recovery phase after the adult worms are expelled.

Most of the clinical signs are seen during the pre-patent and patent phases, and the pneumonia results in coughing. The severity of the symptoms relates to the number of larvae ingested, but the age of the host, weather conditions and nutrition can all affect the severity of the clinical signs. In some of the severely affected cattle, there can be a flare up of clinical signs, potentially resulting in fatality. The aetiology of this is unknown, but it is thought to relate to aspiration of dead larvae and aspiration into the alveoli and a possible allergic reaction to the dying material. This is termed post-patent parasitic bronchitis.

DIAGNOSTIC CHALLENGES

Clinically, lungworm should not really be a diagnostic

challenge for veterinary surgeons. A persistent dry cough in an animal with access to pasture is a common presenting sign. It is often more noticeable as the cattle are moved, eg. when they are brought in to be milked. However, other pathogens such as infectious bovine rhinotracheitis could be included in the differential diagnosis.

Faecal samples can be taken for analysis of L1 levels, with a minimum of 15g faeces being needed for this. It should be noted that false negative results will be seen in the prepatent period, as L1 are not yet being passed. To lower the chance of false negative samples, the faeces samples should be kept in the fridge and processed rapidly. The L1s die off rapidly, and even more so if they are kept in the vet's car. False positive results can occur if the samples are left for long enough for gastrointestinal eggs to hatch into L1.

A bulk milk test is available for use, but at the current cut off used, will only give a positive result when 30% or more of the herd is infected. A positive ELISA titre indicates a recent herd exposure, but it is not an accurate indicator of determining the immune status of an individual animal. The test used only detects an antigen that is extracted from L5 and so will not detect the prepatent period and, in fact, antibody levels do not increase significantly increase until four to five weeks after infection. This can present a significant diagnostic gap in the face of a clinical outbreak. An eosinophilia will raise the suspicion of a parasitic infection, but it is a non-specific test. Bronchiolar lavage can produce useful information in the pre-patent period as well.

IMMUNOLOGY

The immune response of cattle to lungworm infection also presents a challenge. The immune response has two parts:

1) Lowering the number of worms establishing in the lungs; and
2) Dealing with the adult worms that have managed to establish themselves, despite this immune response.

The immune response for incoming larvae is only about four months, whereas the immune response to the adults is remembered for at least two years. This means that when cattle stop ingesting larvae eg. at housing, they will be immune to larvae for months, and to adult worms for two years. Carrier cattle will shed larvae and are responsible for maintaining the immune memory in some herds. In cattle getting infected for the first time, the worms will usually develop to adults and the infection becomes patent causing what we call classical lungworm infection. This is usually seen in the first grazing season but can also be seen in adults by introducing carrier animals or spreading of contaminated slurry.

When immune animals have not been exposed to L3 for a few months, their immunity to incoming larvae may wane. If these cattle are then turned out onto contaminated pasture, they can suffer from reinfection syndrome. This results in a rapid killing of immature larvae and triggers a hypersensitivity reaction characterised by eosinophilia. These animals can present as very ill or sudden deaths and frequently respond very poorly to treatment. This form of lungworm is assumed to be caused by a hypersensitivity reaction due to the infective larvae.

VACCINATION

Vaccination should be considered on farms with evidence of lungworm, but it can be tricky to manage on some farms. The calves must be at least eight weeks old before they can receive their first dose of the vaccine. Then, a second dose is administered four weeks later, and the animals can be turned out two weeks after the second dose. Therefore, the vaccinated calves will need to be at least 14 weeks old at turnout, causing problems in spring-calved herds and in all-year-round calving herds.

CONCLUSION

While lungworm provides many challenges, provided the on-farm risk factors are managed, lungworm control programmes can be developed specifically for each farm.

References available on request.