Lungworm – time to vaccinate

Sarah Campbell MVB MRCVS outlines three key aspects of lungworm control that veterinary practitioners should employ when advising on individual farm systems: immunisation; pasture management; and strategic dosing

Control of parasitic bronchopneumonia caused by *Dictyocaulus viviparus* (bovine lungworm) poses an increasing challenge to beef producers and dairy farmers despite the huge amount known about the disease and the vast array of anthelmintic products commonly used to treat infection.

The prevalence of lungworm varies each year depending on weather conditions. From 2010-2017, there was an increase in the number of cases of parasitic bronchopneumonia diagnosed, peaking at 21% of diagnosed respiratory disease submissions in 2017 (Figure 1).[1] During 2018, Dictyocaulus species were detected in 9.4% of bovine respiratory disease (BRD) submissions (Figure 2).[1] Last year, the warm and wet weather observed from late summer and autumn aided in the spread of infective larvae throughout pasture and promoted infection.

D. viviparus is one of the most commonly diagnosed causes of respiratory disease leading to morbidity and even death in cattle.[1] Often considered to be a condition of younger animals during their first grazing season, it is becoming more frequently diagnosed as a cause of severe respiratory distress in adult stock. Outbreaks mostly occur between July and September. However, infection can arise during an extended period if optimal weather conditions allow (Figure 3).[1]

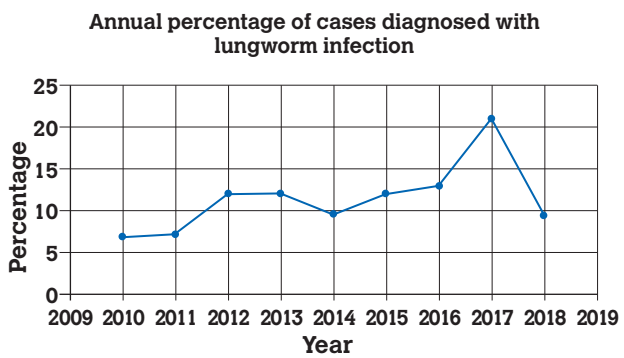

Figure 1: Trends in the incidence of parasitic pneumonia in carcases (all ages) submitted to regional veterinary laboratories (RVLs) from 2010-2018.[1]

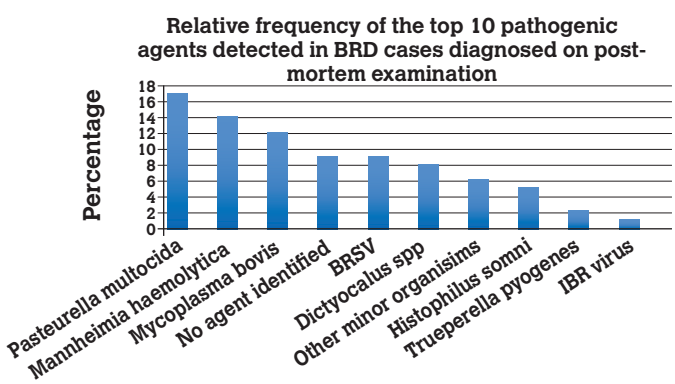

Figure 2: Relative frequency of pathogenic agents in bovine respiratory disease cases diagnosed on post-mortem examination of all carcases in RVLs in 2018 (n=542).[1]

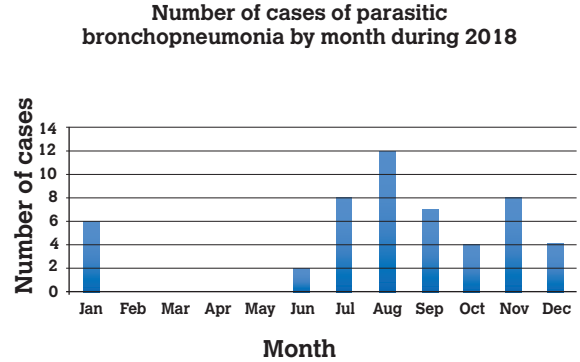

Figure 3: Number of cases of parasitic bronchopneumonia diagnosed by month during 2018 (n=51).[1]

Alone, lungworm can cause mild to severe respiratory disease in cattle during the grazing season. In addition, the adult stages can create the ideal conditions for secondary viral and bacterial agents to do further lung damage. Therefore, mixed infections are not uncommon. The condition is frequently referred to as parasitic bronchitis, parasitic bronchopneumonia, husk, hoose or dictyocaulosis and is endemic in temperate areas with high rainfall such as the weather experienced in the UK and Ireland. The cost of an outbreak of lungworm infection has been estimated conservatively at €167 per adult cow. The main costs associated with an outbreak are attributed to reduced milk yield, infertility and deaths.[2]

The effect of lungworm depends on their location within the respiratory tract, the number of infective larvae ingested and the immune status of the animal. Constant exposure to a low burden of infective larvae allows the animal to develop and maintain reliable immunity.

A lungworm outbreak is characterised by coughing and respiratory distress, and typically affects young cattle during their first grazing season. Persistent coughing generally reflects the irritation caused by the presence of adult parasites in the main airways (Figure 4). Cattle can have difficulty breathing, occasionally exhibiting open-mouth breathing, and may or may not have a high temperature. Deaths can occur if untreated and those that do survive can become poorly thriven. Surviving animals usually develop strong immunity. Occasionally, if an older animal with acquired immunity is suddenly exposed to a massive larval challenge from a heavily contaminated field, severe clinical signs may result. In recent years lungworm infection has been observed in older cattle due to the repeated use of long-acting anthelmintic products during the first grazing season. Some of these products have a residual activity and prevent calves from being sufficiently exposed to lungworm infection to develop immunity.[3]

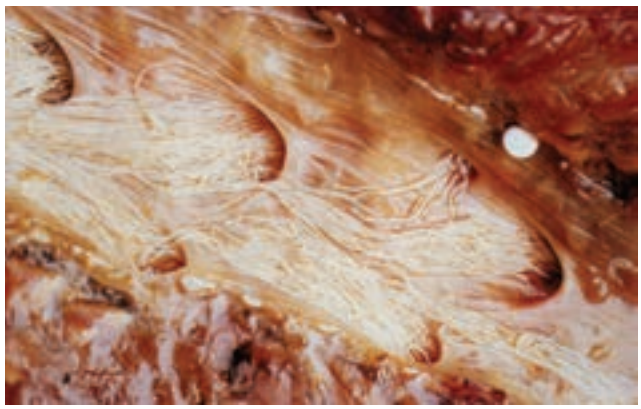

Figure 4: Adult lungworm present in the main airways of a bovine detected during post mortem examination

LIFE CYCLE

Lungworm infection is contracted through ingestion of L3 infective stage larvae on contaminated grass. Protective immunity develops after infection; therefore, the disease primarily affects young animals during their first grazing season. Major outbreaks are seen from mid-summer to early autumn, when sensitive calves have been on pasture two to five months and the parasites have had time to reproduce. Older animals that have been exposed in previous grazing seasons are usually resistant, but they can act as carriers and spread the infection without showing any symptoms of the disease. Moreover, under favourable conditions *D. viviparus* larvae can overwinter on pasture.

After ingestion of infective stage three larvae (L3), the parasite penetrates the intestinal wall and nearby lymph nodes where they moult into stage four larvae (L4). The L4 larvae then continue to the lungs via lymph and blood, where they appear approximately one week after ingestion with moderate infection doses, and as early as after 24 hours at very high infective doses. While in the lungs the larvae develop into young adults, which migrate up through the airways as they mature. Mature adult worms are quite large, measuring up to 8 cm long. Females living in the main airways produce eggs that hatch almost immediately into first stage larvae (L1). These L1 larvae which are coughed up from the lungs, swallowed down into the rumen, are then excreted and can be detected in faeces from approximately day 25.[4] On pasture, the larvae moult into the second stage (L2) and further to L3. The time needed for the development of the L1 to L3 stages is dependent on weather conditions but under optimal conditions can be reached within five days during mild (>15°C) damp weather.

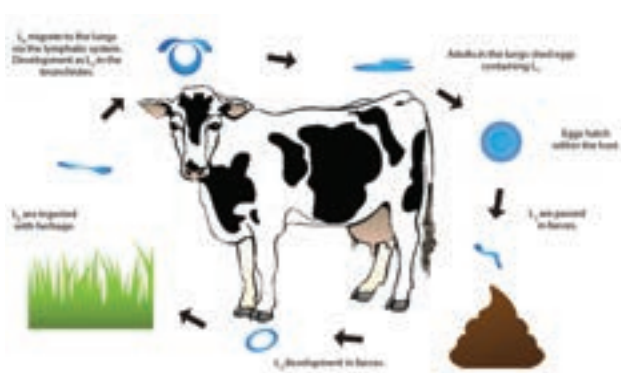

Figure 5: Life cycle of *Dictyocaulus viviparus*.[5]

IMMUNITY

Immunity against lungworm larvae is stimulated by exposure. Larval or pre-lung immunity against the infective stage larvae (L3) only lasts for a few months, depending on the level of challenge.[4] Larval immunity is reliable but requires a persistent challenge to be maintained. Vaccination prior to turnout stimulates larval immunity before exposure allowing animals to have appropriate protection for the season.

Immunity against adult lungworm lasts for two to three years depending on the level of infection. Adult or lung-phase immunity is strong and prevents maturation of larvae into the adult stages and survival in the lungs.

While immune protection is very useful, the strong inflammatory response initiated by lung phase when immunity against larval stages has waned can be severe. Re-infection syndrome occurs when older animals are introduced to a pasture with a heavy lungworm burden and become infected with L3 larvae around the time larval immunity has waned, a severe inflammatory response can occur even though a firm adult immunity has developed.[4] The resultant effect can lead to severe dyspnoea and ill-thrift in affected adults as well as reduced milk yield in dairy animals.

RISK FACTORS

Failure to allow calves and adult cattle controlled and continual exposure to either a low or moderate level of infection to develop and maintain immunity to the lungworm parasite is the main predisposing factor for disease. Young animals (especially first grazing season calves) if exposed to a very high challenge early in life will succumb to disease. Older animals may also show signs of disease. The increased use of long acting broad-spectrum dosing products in young stock has been associated with leaving older cattle vulnerable to infection which was previously only seen during the first grazing season.

The risk of exposing calves to a high challenge is increased if they are entered on to pasture contaminated by animals that were in their first grazing season during the previous year. Overwintering of L2 larvae can occur if optimum weather conditions prevail. Mixing groups of old and young cattle as well as introducing bought-in animals with unknown lungworm status has the potential to cause a problem during the season.[5]

In Irish grazing systems, maximising the grazing season is key to profitability. On some farms, young calves receive a long acting anthelmintic product during the first grazing season as a labour-saving tool. Unfortunately, this can delay those animals from acquiring immunity against larval and adult stages of lungworm until the second grazing season or later.

Upon arrival into the adult milking herd, heifers may become exposed to lungworm challenge and suffer clinical signs of infection during lactation. The resultant increase in pasture contamination can cause adults to suffer re-infection syndrome. The outcome of a disease outbreak in adults can be huge due to reduced feed intake, milk-drop, infertility and deaths.

It is estimated that around 10% of adults in the herd can be asymptomatic carriers of lungworm infection. This acts to provide adult grazing platforms with a low-level challenge to stimulate ongoing pre-lung phase immunity. The use of zero milk withdrawal dosing products has increased in dairy herds. The disadvantage is that the use of anthelmintics can clear

out asymptomatic carrier cows, thus removing the ongoing challenge that they would have provided. In turn, larval immunity wanes quickly leaving the herd with only lung phase immunity for protection in the face of a challenge.

IDENTIFYING THE PROBLEM

In the acute, pre-patent phase, parasitic bronchopneumonia can be difficult to diagnose. Egg production has not commenced, there are no adult larvae in the lungs, antibody production against the L5 stage has not begun and a plasma eosinophilia is not specific to the condition. Clinical signs and ruling out other causes may be the most appropriate approach. Sudden onset of coughing, milk drop in dairy cows, difficulty breathing and ill-thrift all potentially indicate a heavy parasitic burden amongst other causes.[5]

During the patent phase, diagnostic sampling is often more valuable. To identify a lungworm infection within the group, faecal samples can be sent to a veterinary laboratory to be examined using the modified Baermann technique for the presence of L1 larvae. Samples should be sent to the lab soon after collection and kept cool as the larvae can die rendering the sample useless. First stage larvae will only be found if egg-producing adults are present in the lungs. In early cases, adults may not have commenced egg production therefore a negative result does not rule out *D. viviparus* as a cause of clinical signs. In animals that have been previously exposed, a strong lung phase immunity will prevent adult development so egg production is unlikely to occur. To support a diagnosis, bronchiolar lavage can be useful to demonstrate the presence of larvae in the lungs.

Blood samples can be used to detect antibodies against lungworm indicating recent exposure. Antibodies are usually found in blood three to four weeks post infection and can last for up to six months. There is often a characteristic plasma eosinophilia though this is not diagnostic or uniformly present. Gross post-mortem examination of the respiratory tract often reveals a high number of adult lungworm larvae within the lumen of the trachea and bronchi. Lung consolidation adjacent to congested airways are a common feature.[5]

CONTROL

Due to the unpredictable nature of timing an outbreak of lungworm infection, it is vital to focus on a useful strategy of boosting animal immunity while reducing the overall challenge on pasture. The three most important components of controlling lungworm involve vaccinating prior to turnout to boost immunity, good pasture management and implementing a strategic worming program. This will ensure that animals have reliable immune protection, be exposed to a low level of challenge throughout the grazing season to maintain immunity as well as benefit from appropriate anthelmintic products that allow exposure to occur and are effective at treating infections of both lung and gut worms.[5]

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2. Holzhauer et al. Lungworm outbreaks in adult dairy cows, estimating economic losses and lessons to be learned, Veterinary Record (2011), 169:494-499.
3. McLeonard C, Van Dijk J. Controlling lungworm disease (husk) in dairy cattle. In Practice (2017) Volume 39:408-419
4. Van Dijk J. The Epidemiology of *Dictyocaulosis* in Cattle, Cattle Practice (2004) Vol 12 Part 2, 137-138.
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READER QUESTIONS AND ANSWERS

1. THE INFECTIVE STAGE OF LUNGWORM IS:
- **A.** L1
- **B.** L5
- **C.** L3
- **D.** L2

2. THE PREPATENT PERIOD IS GENERALLY ASSOCIATED WITH:
- **A.** No clinical signs
- **B.** A negative Baermann faecal analysis
- **C.** Egg production commencing
- **D.** L1 larvae in the trachea

3. OUTBREAKS OF LUNGWORM ARE MORE LIKELY IN FIRST GRAZING ANIMALS WHEN:
- **A.** They graze on infected pasture
- **B.** There is a period of wet weather to facilitate dispersal of infective larvae
- **C.** Most likely during late Summer to Autumn
- **D.** All of the above

4. OUTBREAKS OF LUNGWORM ARE MORE LIKELY IN ADULTS WHEN:
- **A.** Young stock have received long acting anthelmintic products in their first grazing season
- **B.** Naïve animals enter the adult group
- **C.** Pre-lung phase immunity has waned in adults
- **D.** Anthelmintic products are administered to the entire adult group
- **E.** All of the above

5. MANAGING LUNGWORM ON INFECTED FARMS REQUIRES:
- **A.** Pasture management
- **B.** Vaccination
- **C.** Strategic worming
- **D.** All of the above

6. WHICH STATEMENT IS NOT TRUE?
- **A.** Lungworm can overwinter on pasture if the conditions allow
- **B.** Adult lungworm can lay 1,000 eggs per day
- **C.** Eggs are passed out in faeces
- **D.** Antibodies are usually found in blood three to four weeks post infection and can last for up to six months

ANSWERS: 1:C; 2:B; 3:D; 4:E; 5:D; 6:C.