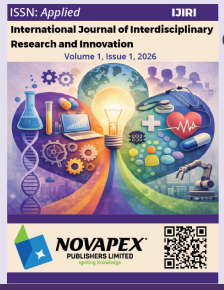

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Bovine lungworm

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ABSTRACT

Bovine lungworm, Dictyocaulus viviparus, is the disease-causing organism which affects cattle of all ages and on top of its self-evident negative effect on the welfare of the animals, may have significant economic effect on the farm level, even in cases of subclinical infections. Although the majority of clinical incidents are observed in cattle in pasture between July and housing, there are rare outbreaks in housed previously grass-infected cattle or sometimes due to forage or low level cycling in straw yards. Although the overall seasonal trend of infection remains the same across extensive time and space, the number of cases observed at both regional and farm levels varies significantly every year and within season. Such comparative inability to predict may create uncertainties in control decisions. To be in a position to offer valid, evidence based guidance on treatment and management to the farmers, it is essential that clinicians understand key, practically important, biology, pathology, immunity and epidemiology of the lungworm.

KEYWORDS: cattle parasites, Dictyocaulus viviparus, epidemiology, pathology, immunology

Introduction

Dictyocaulus viviparus is the causative agent of a severe and occasionally lethal respiratory illness of cattle of all ages, parasitic bronchitis (PB) or verminous pneumonia, and informally. Despite the presence of the parasite in Europe, North America, and Australasia, its effect seems to be most apparent where the prevalence of infection in dairy cattle, using bulk tank milk *D. viviparus* antibody concentrations, is between 3% in Switzerland (Frey *et al*., 2018) and 17% in Germany, 20% (Schunn *et al*., 2013) in Belgium (Bennema *et al*., 2009), 63% in Ireland (Bloemhoff *et al*., 2015 First grazing season (FGS) sero-epidemiological surveys of dairy calves in Germany and dairy and beef calves in Sweden revealed farm prevalence of about 40 percent (Hoglund *et al*., 2004; Schnieder *et al*., 1993). Even though the incidence of parasitic bronchitis occurs in the season, the majority of cases are reported during the late summer and autumn (APHA, 2016), the incidence of infection is highly variable on a regional and temporal scale, and the risk of disease outbreak is far less predictable compared to parasitic gastroenteritis, which also takes place in cattle at the same time of the year at pasture (Al-Gharban, 2016). There have been occasional reports of cases in calves who have never been at grass, as well as in housed cattle, perhaps due to the acquisition of infection shortly after birth or by means of cyclic transmission of infection in the bedding (Crawshaw and Smith, 2003; Al-Eodawee et al., 2023). The first in a series of two papers is based on the premise of completing and extending the review paper published earlier in Livestock (Tilling, 2014), at first with an epidemiology, pathology and immunobiology emphasis, and relying on some of the hugely important studies previously done in the field in the past 65 years, in association with the outcomes of some recent research on lungworm.

Larval ecology and epidemiology

The infection of *Dictyocaulus viviparus* also continues every year in farms in two principal channels (Jarrett *et al*., 1957a):

- During pasture (herbage and soil) as infectious third stage larvae (L3).
- In carrier animals, usually as inhibited L4, hatch to adult lungworms during spring.

In spring when animals are turned out again they can ingest L3 on grazed pasture that was grazed by infected cattle the year before or, in case of grazing with carrier animals, ingest larvae that have been deposited on the herbage after re-activation of dormant larvae to develop into egg-laying adults at the end of winter (Eysker *et al*., 1994). This is because the free- living stages of *D. viviparus* do not take food and grow into infective stages (L3) at a very short period; less than a week in optimum conditions (Rose, 1956). The lack of feeding can be used to explain the fact that they seem to be rather inactive as presumably they must use the little energy resources available to them however, despite the fact that only a few weeks of feeding on pasture can support the infective larvae in warm, dry climate, somewhat paradoxical is the fact that they can live more than a year; despite the fact that the fields used have been used to harvest hay during the period in between (Duncan *et al*., 1979; Nelson, 1977; Oakley, 1977).

An example of this is also the larval listlessness, which is why indirect routes of the dung pat translocation onto the nearby vegetation are so vital such as those mediated by dung-related fungus of the genus *Pilobolus* (Doncaster, 1981; Robinson, 1962). *Pilobolus* species are typical coprophilous fungi; fungi spores travel to the small intestine of herbivores in the dung, where they germinate and then give rise to sporangiophores within a period of about 1 week. The sporangia are expelled by the sporangiophores in the middle of the day, together with any larval lungworms that may have been climbing on the sporangiophores on to the sporangium (Doncaster, 1981). Not only is the interaction between lungworm and *Pilobolus* spp. not simply a pleasant piece of interesting biology, but it is also of significance in the epidemiology of dictyocaulosis since it has been found that translocation of L3 across distances of 100 cm or more between the dung pat and the surrounding herbage was minimized by more than 90% in the absence of the fungus (Jorgensen *et al*., 1982). Furthermore, larval transport over long

distances within and between fields due to passive feces on farm implements and farm implements has also been shown (Rose and Michel, 1957; Spedding and Michel, 1957).

The carrier animal relative significance to the epidemiology of dictyocaulosis is also geographically variable over-winter larval survival plays a significant role in the year-to-year maintenance of infectivity of pastures and contains small numbers of adult lungworms in 31% of yearlings (stirks) and 4% of cows sampled in late winter (Cunningham *et al*., 1956). In the Netherlands, carrier animals are those assumed to play a key role in the epidemiology of lungworm (Saatkamp *et al*., 1994), over-wintering on pasture in the Netherlands being thought to be inconsequential (Al-Hassani et al., 2018). Carrier animal significance has been exploited in the efforts to eradicate *D. viviparus* in the dairy farms by treating the herd before turnout to eradicate all of the lungworm in the older cattle (Ploeger and Holzhauer, 2012). Although this paper is entitled differently it was shown that over a year on one farm and at least four years on the other no infection or disease took place. Regardless of the regional differences, it must be borne in mind that mixed-age cattle groups of animals represent another risk factor related to disease (Forbes, 2017) due to the possibility of mixed carrier animals with their naive youngstock (Gupta and Gibbs, 1970; Gharban et al., 2022).

D. viviparus takes 3-4 weeks to complete the pre-patent phase and female lungworms are highly fecund; most records on adult egg production rates and therefore larvae (L1) output are between 1000 and 25,000/female/day (Ploeger and Eysker, 2000), which is useful as an average in theorizing. In this way even relatively small (Less than 50) numbers of cattle bearing adult worms may be seeding the pastures with millions of L1 per day. The result is that the increase of pasture larval population can occur very fast because L3 development can be obtained within less than a week. Although L3 can survive longer and survive over-winter in warmer and drier conditions on short-swarmed pastures, survival may be very brief, only a matter of weeks, and this can cause great variations in the larval populations of the pastures (Jorgensen, 1980b). Assuming a 250 kg, FGS calf is used as an example, the amount of forage it consumes is of the order of 5 kg dry matter (DM) /day (Forbes *et al*., 2000), hence, when grazing a pasture with 1600 L3/kg DM (Jorgensen, 1980b) it may be ingesting 8000 *D. viviparus* L3/day. They have a rate of establishment at 30% (Ploeger and Eysker, 2000) in the lungs of naïve animals, and thus a burden of 2400 worms in an adult could be obtained in a single day of L3 ingestion; calamities associated with such an adult burden include clinical disease and death (Jarrett *et al*., 1957a; Michel and Parfitt, 1956; Gharban et al., 2022).

Pulmonary stages and pathogenesis

After ingestion, infectious larvae become more active in the presence of bile in a microaerophilic environment (Jorgensen, 1980a) and penetrate the wall of the small intestine after approximately four days and thence via the thoracic duct through the circulation to the lungs where the infection reaches the lungs within a week after being infected (Jarrett *et al*., 1957b). In this case, the larvae infest the alveoli, then the bronchioles where they grow up to be adults in the major bronchi or at the base of trachea. This is when the parasites have a remarkably fast period of growth; larvae begin to enter the lungs a few millimeters long, and it will transform into adult worms a number of 3-8 centimeters long 3-4 weeks later (Laabs *et al*., 2012). In the cases of colonization of the lungs, inflammatory changes are significant in both the alveoli and the bronchioles in which there are mucus and debris of parasites, full of eosinophil (Schnieder *et al*., 1991). The alveolar pathology involves epithelialization whereby the normal cell structure is substituted by undifferentiated cells that cannot carry out gaseous exchange making the affected alveoli incompetent (Jarrett *et al*., 1954).

It seems that alveolar epithelialization is irreversible and can explain some of the failures in treatment where the elimination of the parasites still does not result in a clinical manifestation clearance (Jarrett *et al*., 1957b). A loss of ciliated epithelial cells in the bronchi (Schnieder *et al*., 1991) is also another aspect of the pathology and this would also be likely to make the host more susceptible to

other respiratory pathogens. This is often presumed by practitioners and possibly the grounds on which precautionary administration of antibiotics is undertaken under some situations, but very little and mixed scientific evidence exists to support such an approach. Clinical manifestations were more intense in co-infected calf in case of *D. viviparus* infection, and subsequent infection with Respiratory Syncytial Virus (RSV) (Verhoeff *et al*., 1988). Recrudescence of infection occurred in relatively high challenge (50 L3/kg) with *D. viviparus* in animals latently infected with bovine herpes virus I (IBR) (Msolla *et al*., 1983). Contrastingly, there was no aggravation of bacterial isolation frequency and severity of pathology in the calves infected with *Pasteurella haemolytica* by subsequent infection with *D. viviparus*, despite severe PB developing in these calves (Shoo *et al*., 1990).

Host-parasite interactions

Immune responses are central in dictyocaulosis with regards to host susceptibility as well as disease manifestation. The immune response is fundamentally biphasic (Kooyman *et al*., 2007; Michel, 1962; Michel and Mackenzie, 1965) and can be summarized elsewhere (Forbes, 2012) as well as below:

- Phase 1: Once the ingested larvae are out of the intestinal tract they travel to the mesenteric lymph glands before reaching the lungs. An immune response is triggered in there and most of the larvae are destroyed in place and they do not reach the respiratory tract. Activation occurs fast (within 2 weeks of a primary infection) but is brief and declines significantly during the next 6 months unless it proceeds with additional exposure.
- Phase 2: Those larvae that manage to avoid the immune reaction in the mesenteric glands enter the respiratory system a secondary immune reaction is activated leading to the dismantling and eradication of parasites in the lungs. Unlike the phase 1 response, the 2 nd phase response takes place over a period of two years or longer in the lungs, even in the absence of any challenge.

The magnitude of the response is dose-dependent, and in the case of cattle, it is possible to obtain patent infections without the necessary development of a fully protective immunity and therefore succumb to a subsequent infection when back on the same pasture by ingesting high contents of larvae produced by the first generation of parasites (Jorgensen, 1980b) and the induction of protective immunity after only 10 days development in naive hosts, but the magnitude of the response increases as the animals develop more (Ploeger and Eysker, 200

Re-infection syndrome is experienced when the phase 1 response has been diminished by absence of exposure. Larvae re-exposed to it do so at the lungs where they are exposed to the Phase 2 response. Should large numbers of larvae get to the lungs they will be killed and killed, although as a consequence, the resultant immunopathology may result in respiratory symptoms that can mimic those of patent *D. viviparus* infection (Michel and Coates, 1958). This is the reason why the re-infection syndrome is frequently observed 10-14 days post a change to a new pasture and why the larvae of lungworm are rarely present in fecal samples in adult cows at pasture except in cases of naïve and immunologically naïve larvae which are undergoing a primary lungworm infection or in carriers where inhibited larvae has re-initiated growth.

Lungworm in adult cows

The fact that the percentage of the parasite in adult cattle has been increasing over the past years or not is not a recent quote as some people would think but these are the first lines in an article published in 1955 (Michel and Shand, 1955). However, even though there are no precise estimates of the prevalence of PB in adult cattle, even passive surveillance of its diagnosis, as depicted in a recent review (McLeonard and Van Dyk, 2017), includes an upsurge in the rate of submissions being done on adult cattle (David, 1993). In case of actual increase in the prevalence in cows what could be the probable cause? One of the widely spread beliefs is that this is caused by a decrease in the use of lungworm vaccination and

increased dependence on anthelmintics in the treatment of PB (McLeonard and Van Dyk, 2017), but there is limited scientific evidence to substantiate this. It is rather possible that a smaller number of animals in the national herd with a predictable immunity to *D. viviparus* with the help of vaccination implies that the enzootic instability has become more widespread and hence the outbursts of patent lungworm infection or the re-infection syndrome in cows when the host immunity and larval challenge no longer match (David, 1997).

Many of the studies of relevancy in the field could be found during the decade between 1988 and 1998, where the immunity to lungworm was evaluated, following the strategic application of anthelmintics (including boluses); one of the more recent papers is referenced because it lists some of these studies (Schnieder *et al*., 1996). The clinical and serological monitoring of the immune status of anthelmintic-treated populations was conducted in eight studies by artificial infection by *D. viviparus* infective larvae following housing at the end of the grazing season. Post challenge lungworm burden in anthelmintic treated animals in one study (Taylor *et al*., 1997) was always significantly lower than in naïve controls but was usually comparable across peer groups that received different strategic regimes or in untreated controls. The effectiveness of the anthelmintic regime in controlling lungworm infection was either inversely related to the level of protective immunity or not (Borgsteede *et al*., 1998). Of interest, also, is that cattle exposed to natural lungworm challenge (Forbes and Rice, 2000) or experimentally infected (Taylor *et al*., 2000) can seroconvert to *D. viviparus* in an environment containing effective concentrations of anthelmintic.

The non-evidence of strategic anthelmintic usage during the initial grazing season as a significant risk factor of PB in elderly cattle is furthered by certain epidemiological reports which did not identify any significant and consistent relationships between anthelmintic use in FGS cattle and dictyocaulosis in cows (David, 1997; Ploeger *et al*., 2000). Although there are scanty supporting evidence of their contribution to the risk factors, there have been several changes in the management of dairy herd within the past 25 years or so, which may have contributed to the epidemiology of dictyocaulosis, increasing the outcome, especially in dairy cows. These include:

- Heifers calving at two years
- Reduced time in grass prior to calving; it reduces exposure to *D. viviparus*;
- Heifers kept off pasture were not close to the mature dairyman herd (David, 1997; Michel and Ollerenshaw, 1963).
- On their farms of birth
- Rearing on other farms on contract basis.
- Heifers raised indoors to the time they become pregnant and turned out to graze at about 18-months of age.
- Year-round calving (AYR) herds
- Large proportion of young stock of mixed ages.
- Various turnout periods and number of days/months which grass is spent until the first calving, depending upon the month of birth (Michel, 1957)
- Vaccination regime can solely implement in certain groups of FGS calves (autumn/winter-born).
- FGS calves can only be subjected to strategic anthelmintic programs when they are grouped into a select few cohorts.
- Intensively grazed grass based spring-calving dairy herds.
- Young calves sent to grass, or before they are weaned.
- Majority of calves are too young to be put in vaccination program (before turnout)
- In FGS not a perfect match with the strategic use of anthelmintics.
- High frequency rotational grazing of milking herd that had returned to grazed pasture in a few weeks.

Using information on the biology, epidemiology and immunobiology of *D. viviparus*, it can be inferred

that the above changes in husbandry may cause each or all of them to result in enzootic instability and, therefore, outbreaks of PB, especially in older animals. Besides, deficiency of relevant quarantine may lead to:

* Introduction of naive animals in an endemically infected herd.
* Introduction of infected carrier animals into naïve herd.

The two of these were identified to be associated with outbreaks of PB, and both of them are related to the introduction of infected (carrier) animals into a naïve herd (David, 1997).

Monitoring and diagnosis

Without any proactive control measures against lungworm, one of the most popular and suggesting strategies is that of vigilance and treatment (Michel, 1969). This practice entails that the farmer or stockperson should be able to detect the clinical signs and either treatment or notify the veterinary clinician promptly in case he/she suspects that disease is influencing his/her cattle.

Young stock

Coughing, tachypnea (≥50 breaths/minute) and ill thriving in (naive) calves or yearlings in the second half of the grazing season can be considered near-pathogenomic of disease (Jarrett *et al*., 1957a), making clinical dictyocaulosis highly suspected when one or more of the following follow criteria are met:

- July to October
- Permanent pasture that once was grazed by (older) cattle.
- Combined grazing with the aged cattle.
- None of the anthelmintic strategic use or lungworm vaccines.
- Loss of appetite and retarded growth (Boon *et al*., 1984; Kroonen *et al*., 1986)
- Coughing and / or tachypnea in some of them, especially under disturbance, in a group of animals.
- Death (Downey, 1965)

Their presence in any fecal samples of a sample of about 10 animals is confirmatory, but negative result does not exclude the disease as still may be infected in pre-patent phase: re-sampling of 7-10 days later may confirm. In case PB is not suspected, the different diagnosis of other respiratory pathogens should also be conducted.

Adult dairy cows

Under circumstances such as the following, lungworm would be a concern as far as adult cattle are concerned:

- July to October
- Younger cattle mixed grazing.
- Animals that are added to herd during grazing season including heifers and bought-in animals.
- Recent move to a new field
- No lungworm booster vaccinations.
- There is coughing and tachypnea in a few people (Wapenaar *et al*., 2007).
- Unresponsive, depressed, lack of appetite (Michel and Shand, 1955)
- Acute decline of individual and/or bulk tank milk daily yield.
- Apparent sudden death (SAC, 2018)

Despite the fact that some PB cows are in severe respiratory distress, the diagnosis may be surprisingly difficult unless the cows are experiencing primary infection, when fecal samples may demonstrate the presence of *D. viviparus* larvae, but in young animals - one to two months - and the cow only (Fiedor

et al., 2009) - one to two months. Seropositive samples of blood or milk will also be observed with a primary infection, as long as it is adequate: >25 larvae (Strube *et al*., 2017). Antibodies to *D. viviparus* have little diagnostic potential other than in cases of new infections in naïve animals as they may be detected at least 6 months following initial exposure (Cornelissen *et al*., 1997; Fiedor *et al*., 2009), even without challenge, which means that a positive result only indicates that the animal/herd is infected at some time in the past but not necessarily at the time of sampling. More so, re-infections in dairy cattle may lead to the low or absence of antibody responses and hence seropositivity may be a temporary phenomenon (Strube *et al*., 2017).

According to recent studies, no significant relations between faecal lungworm larva presence, *D. viviparus* seropositivity, and PB clinical manifestation (coughing) were observed in dairy cows (May *et al*., 2018). It was however found that the cows with a patent lungworm infection had a significant ($P<0.05$) association with the daily milk yield, and was lower by 1.6 kg/cow/day when compared with the controls. A very low percentage (5 percent) of coughing cows were infected with patent lungworm and hence the main focus is also on the differential diagnosis of respiratory disease between groups of adult cattle and is specifically on infections of bacteria, mycoplasma or viral infections especially infectious bovine rhinotracheitis (IBR). But with that said, as opposed to respiratory symptoms, a drastic decrease in daily milk production was found to be correlated with patent PB, it might be suggested that an unexpected drop in the milk production at the individual cow or the herd level, in particular, in the latter half of the grazing period, are indicative of lungworm, unless any other factors of poor yield can be identified.

Helminth co-infections

The common practice is to consider parasite infections species-by-species, however, due to the prevalence of parasitic gastroenteritis (PGE) in cattle of all ages and PGE in combination with *Cooperia oncophora* in FGS animals, lungworm infections are usually superimposed on PGE. The tendency is observed in the survey of antibodies on milk tanks based on the mass milk tank testing on the antibodies to different helminths in dairy cattle (Bennema *et al*., 2009; Bloemhoff *et al*., 2015; Frey *et al*., 2018). This complicates the ability to pinpoint what or who is causing resultant decreases in milk yield to lungworm or *O. ostertagi* or a combination of the two (Dank *et al*., 2015). In naive calves under experimental conditions, co-infestations of *Ostertagia ostertagi* and *Cooperia oncophora* produce a more significant effect of dictyocaulosis which is reflected in the degree to which lungworm burden and larval shedding occurs than when the same is done in the absence of interaction with *Cooperia oncophora* (Kloosterman *et al*., 1989). This is another possible PGE role in dictyocaulosis, transmission of *D. viviparus* has been found to increase when feces are in semi-liquid state and scattered sparsely across pasture (Rose and Michel, 1957); diarrhea is usually disruptively linked to PGE. These findings apply to the PB management in the field, where concurrent control PGE would appear to be desirable, especially in young cattle (Michel and Parfitt, 1956).

Dictyocaulosis in cattle, in all its manifestations, has a profound impact on infected individuals, the herd and the farm with self-evident losses through mortality, reduced growth rates and milk yield, costs associated with treatment of clinical cases and control measures (Holzhauer *et al*., 2011). An understanding of the underlying pathology and immune responses to *D. viviparus,* considered within an epidemiological framework, can help clinicians understand disease processes and squeal so that recommendations for therapy and prevention can be logical and evidence-based; this will be the subject of the second article on lungworm.

Conclusion

The pulmonary biology has the potential to cause increased changes in the pasture larval population and therefore an eventual risk of infection and disease during the same grazing season. The result of

the infection exposure will be determined by the size of the pasture larval challenge and the immune condition of the cattle grazing in the pasture. Alterations in dairy systems and rearing in the recent past (approximately 30 years) could have led to the destabilisation of the acquisition and maintenance of immunity to *D. viviparus*, either due to natural exposure or vaccination. The best way of treating and controlling lungworm, based on understanding the main features of immunobiology and epidemiology is best discussed with farmers who know specifics about their farms every year.

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