GROWING ECONOMIC BACKLASH DEMANDS LUNGWORM AWARENESS

Author : Hany Elsheikha

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Hany Elsheikha stresses the importance of lungworm prevention in large animals, citing the global repercussions of untreated cases, especially with regards to morbidity, mortality and associated high costs

SOME nematodes use the lower air passages and the lung parenchyma as the final habitat – these are the main focus in this article on lungworm prevention in large animals.

Lungworms are a group of parasitic roundworms (nematodes) that colonise the lower respiratory tract of livestock and companion animals. Globally, they cause high morbidity, mortality and economic costs, especially in temperate regions.

The major diseases caused by lungworms are verminous bronchitis and/or verminous pneumonia “husk”.

Several factors known to affect the development and clinical manifestations of husk include an animal's age, previous immunisation or infection, presence of passively acquired antibodies, and anthelmintic treatment.

Additional factors that may play a role in clinical manifestation include the rate of intake of infectious larvae at exposure, host genetic and acquired factors, and the genotype and virulence of the worm.

Many people believe that the continued global circulation of lungworm infections is due to lack of awareness and inadequate treatment and control.

Husk cases are increasingly and commonly reported in the UK, especially in areas where vaccine use is low.

Clinically, the full spectrum of disease due to lungworm infection is now understood. Signs of lungworm infection range from moderate coughing with slightly increased respiratory rates to severe persistent coughing and respiratory distress and failure.

Moderate lungworm infections are common, elicit partial immunity, and do respond quickly to treatment.

Severe lungworm infections can cause significant distress to the animal, with reduced weight gains, reduced milk yields, unthriftiness and death. Patent subclinical infections can also occur. Anthelmintics are commonly used as a prophylactic against lungworms and other nematode infestations.

The term lungworm is generally used for a variety of different groups of nematodes, some of which use the lower air passages and the lung parenchyma as the final habitat.

Other nematode species, such as ascarid larvae, migrate through the animals' lungs or respiratory tracts, causing degrees of pathological damage according to the nature and intensity of the host-parasite interactions.

Some nematode species, such as *Capillaria aerophila*, live in the lungs, but are not lungworms. Adult worms of this species are found in the mucous membranes of nasal passages, trachea and bronchi of dogs and cats.

Some nematodes that inhabit the right ventricle and pulmonary circulation, such as *Dirofilaria immitis* and *Angiostrongylus vasorum*, are found in dogs in many areas of the world and are also associated with pulmonary disease.

Clinical signs relating to a cardiac or pulmonary syndrome or to a combination of both may occur. Therefore, the lungworm category is more a descriptive than a well-defined taxonomic group.

Taxonomic classification

The major livestock lungworms belong to one of two superfamilies: Trichostrongyloidea or Metastrongyloidea (Figure 1). However, not all the species in these superfamilies are lungworms.

- **Trichostrongyloidea**

This superfamily includes several species that infest hoofed animals including most common domestic species.

Major features of worms belonging to Trichostrongyloidea are their small size, hair-like shape, small mouth, small bursa in males and direct life cycles.

Common species are:

Dictyocaulus viviparus in cattle and deer;

D arnfieldi in donkeys and horses; and *D filaria* in sheep and goats.

• **Metastrongyloidea**

These include species that infest a wider range of mammals, and have indirect life cycles that involve intermediate hosts. Common species are:

Protostrongylus rufescens in sheep and goats;

Muellerius capillaris in sheep and goats; and

Metastrongylus apri in pigs

Other less common genera of metastrongylid lungworms with minor pathogenic importance in sheep and goats include *Cystocaulus, Spiculocaulus*, and *Neostrongylus*.

Diseases caused by the three *Dictyocaulus* species of cattle lungworm are of most economic importance.

D viviparus is common in north-west Europe and is the cause of severe outbreaks of “husk” or “hoose” in young (including yearlings) grazing cattle.

The lungworm of goats and sheep, *D filaria*, is comparatively less pathogenic, but does cause losses, especially in Mediterranean countries. It has, however, also been recognised as a pathogen in Australia, North America, and Europe.

D arnfieldi can cause severe coughing in horses and, because patency is unusual in horses (but not in donkeys), differential diagnoses with disease due to other respiratory diseases can be difficult.

M capillaris is prevalent worldwide and, while usually nonpathogenic in sheep, can cause severe signs in goats.

Lungworms of ruminants

• ***Dictyocaulus viviparus***

– **Morphology**

– Slender white worms, up to 80mm long that live in the lumen of bronchioles.

– **Epidemiology**

– Distribution – seen mainly in temperate areas such as northeastern US and Europe.

– Requires a moist, cool environment.

– Major outbreaks are from July to September, when sensitive (non-immune) calves have been on pasture for two to five months and the parasites have had time to reproduce.

– Pilobolus fungi facilitate the spread of lungworm larvae in pasture. Larvae located on a sporangiophore are shot several feet when the sporangiophore explodes ejecting spores.

– Older animals are usually resistant, but they can act as carriers and spread the infection without showing any symptoms of the disease.

– Under favourable conditions *D viviparus* larvae can overwinter on pasture.

– **Life cycle**

– Direct life cycle, no intermediate host (Figure 1).

– The infective stage is third-stage larvae (L3). Larvae is ingested via contaminated grass; L3 larvae penetrate the gut, moult to fourthstage larvae (L4) and enter the lymphatics; L4 migrate to lungs via circulation; L4 larvae moult to early adult stage (L5) in the alveoli (may arrest at this stage) and L5 migrate through the bronchial tree as they mature; adults are found in the bronchi and bronchioles; infection is patent in 21 days; at 21 days, fertilised females shed eggs, which hatch almost immediately into L1 and are then coughed up, swallowed and passed in the faeces; and on pasture, the L1 larvae moult into L3 within five days.

– **Clinical signs**

– Husk.

– Fog fever – acute pulmonary emphysema associated with heavy infestations of adult cattle

previously exposed to this parasite in endemic areas.

– Clinical signs range from bronchitis to severe consolidating pneumonia as eggs are inhaled to all areas of the lungs and inflammation commences.

– Signs may be correlated with turnout of young cattle into a lush, previously ungrazed pasture in a moist, cool environment.

• ***Dictyocaulus filaria***

– **Morphology**

– Similar to *D viviparus;* infects sheep, goats and wild ruminants. Found in bronchi and bronchioles.

– **Epidemiology**

– Young animals are more susceptible.

– **Life cycle**

– Direct life cycle, no intermediate host.

– **Clinical signs**

– Respiratory signs, cough, dyspnoea.

– Lethargy, weight loss; and catarrhal bronchitis.

• **Protostrongylus rufescens**

– **Morphology**

– Long slender brown worms. Adult worms are found in the terminal bronchioles.

– **Epidemiology**

– Infects sheep, goats and wild ruminants.

– No age-related immunity; adults continue to collect foci of infection throughout life.

– **Life cycle**

– Indirect life cycle, requires a snail or slug as intermediate host.

– **Clinical signs**

– Weight loss.

– Loss of condition.

– Respiratory signs.

– Diarrhoea.

– **Pathology**

– Worms are found in granulomatous foci in the parenchyma and terminal bronchioles.

• **Muellerius capillaris**

– **Morphology**

– Small sized adults.

– Adult worms found in the parenchyma of the lungs.

– **Epidemiology**

– Goat lungworm, but also found in sheep and wild ruminants.

– **Life cycle**

– Indirect life cycle, requires a snail or slug intermediate host.

– **Clinical signs**

– Goats are most susceptible and can die from pneumonia.

– **Pathology**

– Worms are found in granulomatous foci in the parenchyma.

Lungworms of horses

• ***Dictyocaulus arnfieldi***

– **Epidemiology**

– Often seen in horse grazing pasture with donkeys.

– Donkeys are a common natural host and may not show clinical signs; in these cases, horses may have low-level infections with no eggs seen in the faeces, while donkeys will have patent infections (eggs in faeces).

– **Life cycle**

– Direct life cycle, no intermediate host.

– **Clinical signs**

– Horses will have chronic cough, which is not responsive to antibiotics.

Lungworms of pigs

• ***Metastrongylus apri***

– **Epidemiology**

– Common in feral swine and pastured swine.

– Young animals are more susceptible than adults.

– **Life cycle**

– Adults are found in bronchi and bronchioles.

– Indirect life cycle, requires earthworm intermediate host.

– **Clinical signs**

– Verminous bronchitis, parasitic pneumonia.

– Persistent cough that may become paroxysmal.

– Dyspnoea.

– Lethargy.

– Weight loss.

Diagnosis of lungworm infection

• Antemortem diagnosis

Diagnosis of lungworm infection in live animals is based on clinical signs, grazing history and the parasite's epidemiology.

Infection can be confirmed by the demonstration of (L1) in faeces and/or adult nematodes at necropsy of animals in the same herd or flock.

First-stage larvae (L1) can be recovered using most faecal flotation and Baermann techniques with the appropriate salt solutions.

Recovered larvae may be identified to species by their characteristic morphology as follows:

• L1 *D viviparus* larvae measure 310 to 390µm by 19 to 25µm and have a stout body and a conical tail;

• L1 *D filaria* larvae measure 500 to 450µm by 25µm and have a blunt tail and a small knob at the anterior end; and

• L1 *M capillaris* larvae measure 300 to 320µm by 14 to 15µm and have an undulating tail with a small dorsal spine at its base.

Larvae are not shed in the faeces of animals in the prepatent or post-patent phases and usually not in the reinfection phenomenon. In these cases infection may be confirmed by the detection of immature stages in tracheal washes.

Tracheal washes may also provide cytologic evidence of eosinophilic inflammation consistent with parasitic bronchitis or pneumonia. Bronchoscopy and radiography may also be helpful.

Two serological tests for *D viviparus* infection have been developed: an immunoblotbased dipstick test and ELISA.

Diagnosis can be made after failure of antibiotic therapy to ameliorate the condition – especially in horses, which have relatively infrequent infection.

• Postmortem diagnosis

Postmortem diagnosis can be made by the identification of lungworms and characteristic lung pathology at necropsy.

Adult *D viviparus*, found in the bronchi and trachea, are long, thin white worms with small buccal cavities. Males are 17 to 50mm in length and females are 23 to 80mm.

Adult *D filaria* can also be recovered from the bronchi and trachea at necropsy. Adult males are 25 to 80mm long and have a short bursa, and adult females are 43 to 112mm long with a conical, tapered tail.

Adult *M capillaris* are found in the lung parenchyma. Males measure 11 to 14mm by 32 to 35µm and have a posterior end coiled in 11 to 13 spirals. Adult females are 19 to 23mm by 40 to 50µm.

For the veterinarian, husk should be included in the differential diagnoses for persistent respiratory manifestations in animals, especially in enzootic areas.

Treatment of lungworm infection

Animals with slight cough and tachypnoea respond well to treatment, whereas those with dyspnoea, fever, anorexia, depression, and unthriftiness have a poor prognosis (they either die or develop chronic disease form).

Anthelmintic resistance is not yet considered to be a widespread problem in lungworms.

Macrocyclic lactones and benzimidazoles are common treatments (Table 1) and are effective against larval and adult stages of the worm. Decreased efficacy of using macrocyclic lactones has been observed in a Brazilian study (Molento, Depner and Mello, 2006).

Levamisole is used in ruminants, but treatment may need to be repeated two weeks later as it is less effective against larvae during the early stages.

Animals at pasture should be moved inside for treatment, and supportive therapy may be needed for complications that can occur. In severe cases NSAIDs may be helpful.

Table 1 shows the treatment options for lungworm infections by animal species.

Control

In an effort to offer veterinarians, farmers and animal care givers reasonable assistance to help prevent lungworms, this article summarises guidelines that endorse the rationale for prophylaxis.

- **Deworming**

Lungworm infections in herds or flocks are controlled primarily by anthelmintics. Anthelmintic prophylaxis has become feasible with the advent of broad-spectrum, long-acting anthelmintics (for example, ivermectin, doramectin, moxidectin, eprinomectin) and sustained-release intra-regional boluses containing oxfendazole or fenbendazole.

Several strategic deworming programmes have been developed in Europe, which can effectively suppress lungworm infection developing throughout the grazing season.

• **Vaccination**

It is better to prevent than to treat established lungworm infection. Vaccination against husk is a vital component in the lungworm control strategy. The only lungworm vaccine available in Europe is made up of irradiated infective larvae.

The vaccine is effective and primes immune response against any exposure to infection.

Two doses are given four weeks apart, and at least two weeks before the animals can be turned out in order to allow the development of a protective level of immunity.

Used properly, vaccination can prevent clinical disease, but some vaccinated animals may become mildly infected to the extent that larvae are excreted to perpetuate further infection.

• **Management strategies**

Effective control and management of husk should, in addition to parasite management such as chemotherapy and vaccination, provide other supplementary approaches.

– Good sanitation and management practices will help prevent lungworm infection, because pasture herbage is the most common vehicle for transmission of infection.

– Prevent overcrowding and avoid continuous use of the same pasture for youngstock. Pastures known to be contaminated should not be restocked for at least six months.

– Avoid mixing sheep and goats on the same pasture.

– Horses should not be allowed to associate directly or otherwise with donkeys or mules, especially on pastures.

– Pigs should be housed indoors, especially during the most high-risk seasons.

– Pre-turnout removal of adult worms and arrested larval stages from potential carrier yearlings or adult animals is necessary.

– Elimination of intermediate hosts to discontinue the life cycle; for example, preventing access to infected earthworms in pigs.

– If animals are at grass then it is necessary to treat the herd or flock and move on to clean pasture that has not previously had the same animal species on it.

Table 2 shows how to avoid a poor outcome in the management of parasitic pneumonia.

Public health considerations

• Human

The lungworms described in this report are strictly animal parasites and don't infect humans. However, there have been rare reports of human infections with the carnivore lungworm *Eucoleus aerophilus* in Morocco and Russia.

• Animal

Husk is a disease that need not exist if animals are given access to clean pasture and an effective veterinary public health infrastructure is in place. The latter should monitor the water and food supply and curb infections via treatment and elimination of the intermediate and/or reservoir hosts.

Future research

• Pathogenesis of disease

Although much has been accomplished relating to lungworm infections and despite the availability of anthelmintics, as well as a vaccine, lungworm infections can still have high morbidity and mortality rates – especially in risk groups. These include heavily pregnant animals, stressed and immunosuppressed animals, young animals or those with concurrent or underlying disease.

An extensive research effort is needed to better understand the pathophysiology of diseases caused by lungworm, with the aim of developing improved therapeutics and vaccines.

Another area of interest is the possible relationship between lungworm strains and clinical manifestations of disease. Careful analysis of different worm protein variations can be analysed by disease severity.

Furthermore, animal host genetic factors play a role in the presentation and outcome of lungworm infection. Investigation of these genetic determinants has implications beyond defining animal risks for severe infections, as it also offers significant insights about the pathogenesis of these offending parasites and may even identify potential targets for therapeutic intervention.

• **Better vaccines**

The current lungworm vaccine is made of live attenuated L3.

Although effective, the disadvantages of the live vaccine have made development of a more effective recombinant vaccine a priority.

To avoid the side effects associated with the vaccine, scientists and veterinarians should develop newer generations of vaccines generally free of any alarming side effects.

To enable the development and production of such vaccines, identification of potentially protective antigens and knowledge about host mechanisms responsible for the protective immunity are fundamental.

Basic research on known and new virulence factors will greatly enhance our understanding of lungworm pathogenesis and the host response to infection.

• **Emergence of resistance**

Due to increasing levels of anthelmintic resistance, new approaches to lungworm control are warranted, with reduced reliance on chemotherapeutics.

So what can we do to slow or prevent emergence of resistant strains? An important contribution would be to improve monitoring abilities and capacities to detect new anthelmintic-resistant strains when they emerge, and then try to prevent or slow their spread.

Further reading

- Molento M B, Depner R A, Mello M H (2006). Suppressive treatment of abamectin against *Dictyocaulus viviparus* and the occurrence of resistance in first-grazing-season calves, *Vet Parasitol* **141**(3-4): 373-376.
- Taylor M A, Coop R L and Wall R L (2007). *Vet Parasitol* (3rd edn): 80-84.

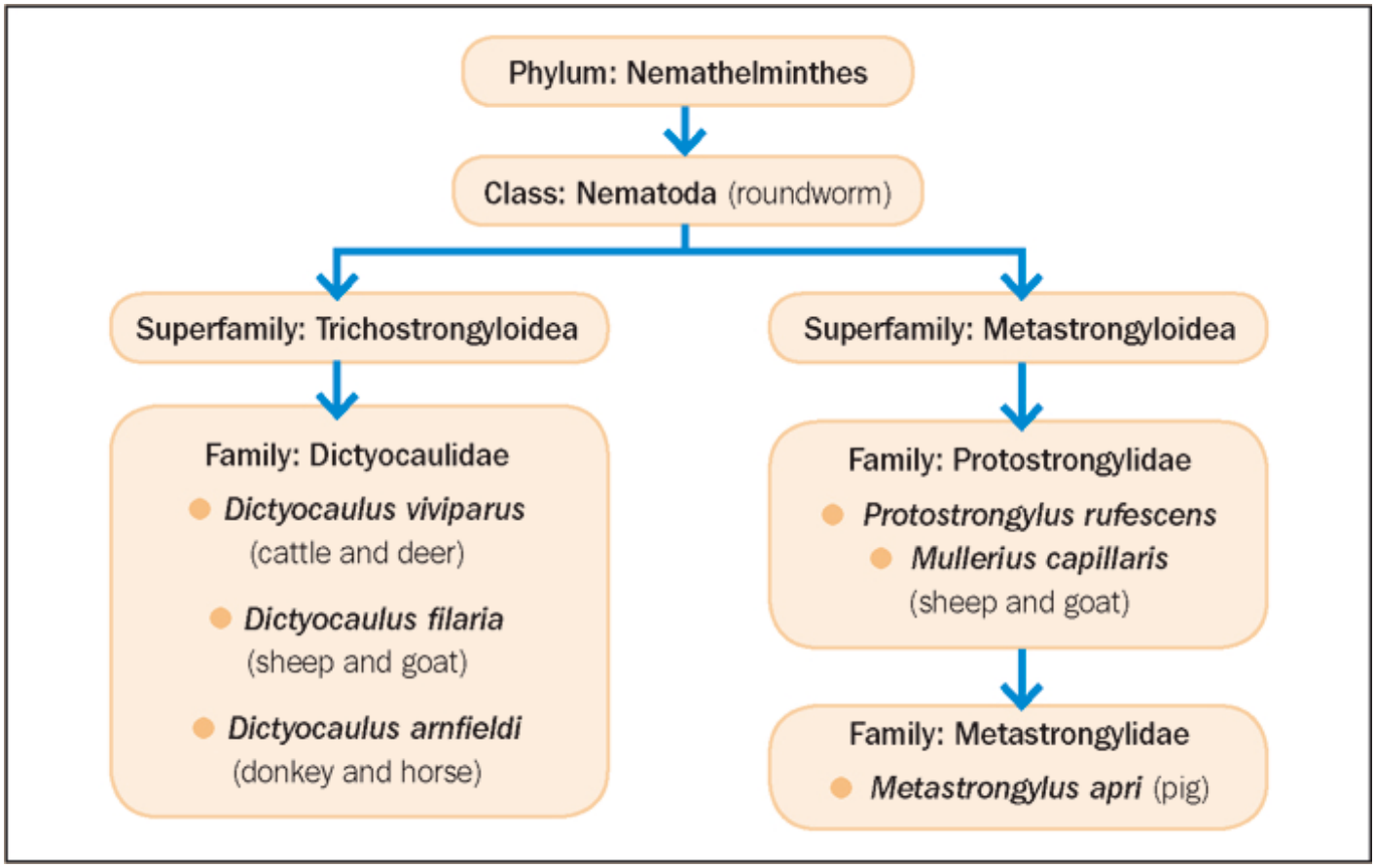

Figure 1. Taxonomic classification of lungworm in ruminants, horses and pigs.

Older animals are usually resistant to lungworm, but they can act as carriers and spread the infection without showing any symptoms of the disease.

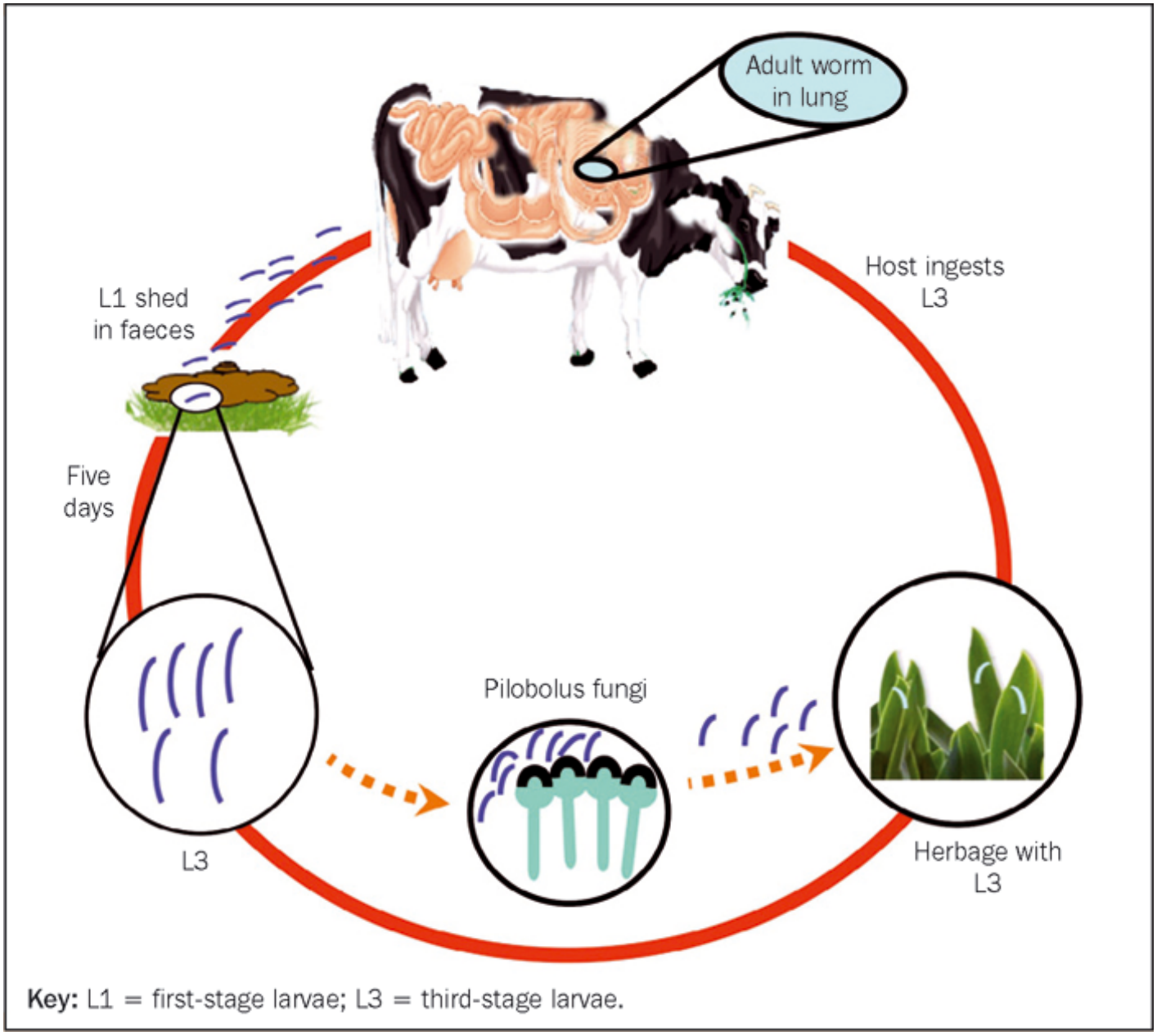

Figure 2. Life cycle of the cattle lungworm **Dictyocaulus viviparus.**

Parasite	Animal species	Treatment
Dictyocaulus viviparus	Cattle	Ivermectin* Doramectin* Moxidectin* Eprinomectin* Fenbendazole† Albendazole† Levamisole†
D filaria	Sheep, goat	As for cattle
D arnfieldi	Horse, donkey	Ivermectin Moxidectin
M apri	Pig	Ivermectin Fenbendazole

* Macrocyclic lactones include ivermectin, doramectin, moxidectin, and eprinomectin.

† Benzimidazoles include fenbendazole, albendazole, and levamisole.

Table 1. Treatment options for lungworm infections

Do not wait for husk to develop to suspect the diagnosis.
Do not exclude the diagnosis because there is no history of lungworm infection.
Do not exclude the diagnosis solely for geographic or seasonal reasons.
Do not withhold therapy if you are clinically suspicious.

Table 2. How to avoid a poor outcome in the management of parasitic pneumonia