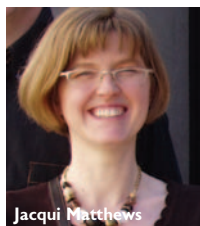# Clinical Forum: Bovine lungworm

Jacqui Matthews BVMS PhD MRCVS
MOREDUN PROFESSOR OF VETERINARY IMMUNOBIOLOGY,
DEPARTMENT OF VETERINARY CLINICAL STUDIES,
ROYAL (DICK) SCHOOL OF VETERINARY STUDIES,
UNIVERSITY OF EDINBURGH, MIDLOTHIAN EH25 9RG AND
DIVISION OF PARASITOLOGY, MOREDUN RESEARCH INSTITUTE,
MIDLOTHIAN, EH26 0PZ, SCOTLAND

Jacqui Matthews

Panel members:

Richard Laven PhD BVetMed MRCVS

Andrew White BVMS CertBR DBR MRCVS

Keith Cutler BSc BVSc MRCVS

James Breen BVSc PhD CertCHP MRCVS

SUMMARY

Parasitic bronchitis, caused by the nematode *Dictyocaulus viviparus*, is a serious disease of cattle. For over 40 years, a radiation-attenuated larval vaccine (Bovilis® Huskvac, Intervet UK Ltd) has been used successfully to control this parasite in the UK. Once vaccinated, animals require further boosting via field challenge to remain immune however there have been virtually no reports of vaccine breakdown. Despite this, sales of the vaccine decreased steadily in the 1980s and 90s; this was probably due to farmers' increased reliance on long-acting anthelmintics to control nematode infections in cattle. This method of lungworm control can be unreliable in stimulating protective immunity, as it may not allow sufficient exposure to the nematode. Such animals remain susceptible to disease when subsequently exposed to *D. viviparus* larval challenge. Evidence of this phenomenon has been provided by the documented increase in the numbers of parasitic bronchitis outbreaks in the UK in the last 20 years, especially in second year grazing animals and adult cattle. This change in disease pattern dynamics is a symptom of the rise in immunologically naïve populations who have either not been vaccinated or exposed to low levels of natural infection during their first grazing season.

LIFE CYCLE

The life cycle of *D. viviparus* is direct. Infection is by ingestion of third stage larvae (L3) from pasture (Fig. 1). The L3 penetrate the intestinal wall and migrate, via the lymphatics and vascular system, to reach the lungs approximately seven days after ingestion. Here, the larvae migrate up the respiratory tree to the larger bronchi and trachea, where they mature to reproducing adult worms about 25 days after ingestion (this is the prepatent period). Animals can harbour 100's-1000's of these white thread-like worms and the adult females produce several thousand eggs that contain first stage larvae (L1). The eggs are coughed up and swallowed with mucus and the L1s hatch out during their passage through the gastrointestinal tract. The L1 are excreted in faeces where development to the infective L3 occurs. L3 subsequently leave the faecal pat via water or on the sporangia of the fungus *Pilobolus*. Infective L3 can develop within seven days of excretion of L1 in faeces, so that, under the appropriate environmental conditions, pathogenic levels of larval challenge can build up relatively quickly.
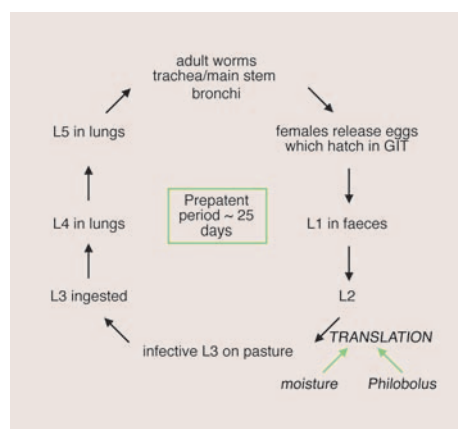

Fig. 1: Dictyocaulus viviparus *life cycle.*

CLINICAL SIGNS

D. viviparus is a highly pathogenic nematode and clinical signs are observed in most immunologically naive individuals. The disease (parasitic bronchitis) is usually observed in the late summer or early autumn, however outbreaks are often reported throughout the year (for example, see Fig. 2a). Affected animals cough, exhibit a rough hair coat and reduced weight gain, the latter being the major economic effect. Severely affected animals become dyspnoeic and can die or need to be culled. In adult animals, the main effects observed are decreased milk yield and reduced fertility with consequent increases in calving period (Holzhauer *et al.* 2003).

Approximately 1-2% of infected animals can develop a hypersensitivity reaction to the nematode and this can lead to acute respiratory distress or sudden death.
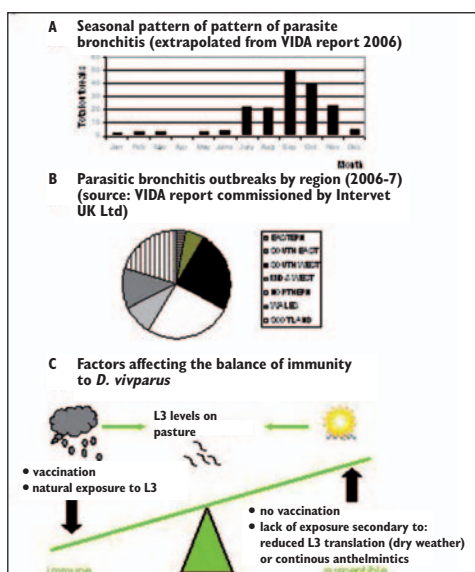

Fig. 2.

DIAGNOSIS

Faecal samples can be submitted for analysis of L1 levels: a minimum of 15 g faeces is required for this. However, it should be noted that false negative results can occur if the infection is still in the prepatent period. Moreover, in adult cattle, which may have a degree of immunity, disease may occur in the absence of a patent infection. For these reasons, laboratory confirmation of parasitic bronchitis by detection of L1 is only successful in a proportion of outbreaks. An alternative to faecal analysis is to detect parasite-specific serum antibodies by enzyme linked immunosorbent assay (ELISA). In practical terms, when investigating an outbreak, it is advisable to analyse faecal and serum samples collected from a group of 6-10 animals that have been showing clinical signs of disease the longest. The ELISA is the preferred option when large numbers of samples are being tested.

Serological diagnosis has been evaluated in naturally and experimentally infected, as well as in vaccinated, animals. These studies have shown that positive ELISA titres are a satisfactory indicator of recent herd exposure, but they are not an accurate means of determining immune status of individual animals (Bos and Beekman 1985). The *D. viviparus* ELISA that is currently commercially available in the UK incorporates an antigen preparation that has been extracted from fifth larvae stage and adult worms (http://www.defra.gov.uk/corporate/vla/servtovet/servtovet-intro.htm). In this test, early larval invasion following vaccination or during the pre-patent period is not detected and parasite specific serum antibody levels do not increase substantially until four to five weeks after initial challenge. Also, seroprevalence rates do not always accurately reflect the presence of clinical disease: for example, in one study where 75% of herds had tested positive by ELISA, only 15% had clinical parasitic bronchitis,

although 51% of farms had experienced disease in the past (Boon, Kloosterman and van der Lende 1986). It should also be noted that cattle convert to seronegative a few months after the adult *D. viviparus* have been expelled so, again, it is difficult to assess the immune status of such animals (Ploeger 2002).

FACTORS AFFECTING THE BALANCE OF IMMUNITY TO *D. VIVIPARUS*

Compared to the gastrointestinal nematodes, immunity to *D. viviparus* develops relatively fast: this is probably the reason why it was relatively 'straightforward' to develop the vaccine back in the 1950s! However, in the absence of vaccination, protective immunity only develops after there has been sufficient exposure of cattle to natural L3 challenge. The level of natural challenge is dependent upon a number of factors and these can determine whether or not an outbreak will occur in a given year. Factors that are important in predisposing to an outbreak of clinical lungworm disease include:

- A history of the presence of *D. viviparus* on a farm as evidenced by previous outbreaks. This is a good indicator that animals are being exposed to the parasite at that site.
- Levels of moisture that affect translation of L3 onto pasture. Sudden changes in environmental circumstances, such as rainfall after a dry spell, may tip the balance between uptake of L3 numbers that result in sub-clinical infection or levels that lead to a full-blown outbreak of severe respiratory disease. This is exacerbated by the fact that not only does rain accelerate larval dispersion from pats but, in the warmth of summer, moist conditions encourage growth of *Pilobolus* which helps to disperse L3. A dry season followed by a wet summer is thought to increase the probability of a disease outbreak because dry weather results in low translation of L3 onto pasture in one year, which creates a 'natural immunity gap' in grazing animals leaving them susceptible to disease in the subsequent grazing season. The importance of rainfall in the epidemiology of parasitic bronchitis is highlighted by UK regional data for outbreaks which shows that the disease is much more prevalent in the wetter, western regions (Fig. 2b).
- Alterations in the dynamics of the immunity status of a herd. The introduction of susceptible stock can destabilize immunity in a herd where a low level of L3 challenge, insufficient to cause disease in existing stock previously, can be sufficient to instigate an outbreak in the introduced animals, which then amplify the levels of infection on pasture (Holzhauer *et al.* 2003). This can occur, for example, when dairy replacement animals are grazed on pasture separate from the main herd and are subjected to extensive worming in their first and second seasons before being introduced to the main herd.

The effect that these factors have on the balance of immunity in a herd in summarized in Fig. 2c.

PATTERNS OF DISEASE OUTBREAKS

Increases in the number of annual parasitic bronchitis outbreaks were recorded by the Veterinary Investigation Diagnosis Analysis (VIDA) throughout the 1990s. These levels peaked in 1997 (McKeand 2000). Since then, the number of outbreaks recorded in the UK by VIDA has reached a relatively steady state (Fig. 3a). It must be noted that the data recorded by VIDA is likely to be a substantial underestimate of the actual prevalence of the disease: many cases are treated by farmers without veterinary consultation or without involvement of the veterinary investigation services.
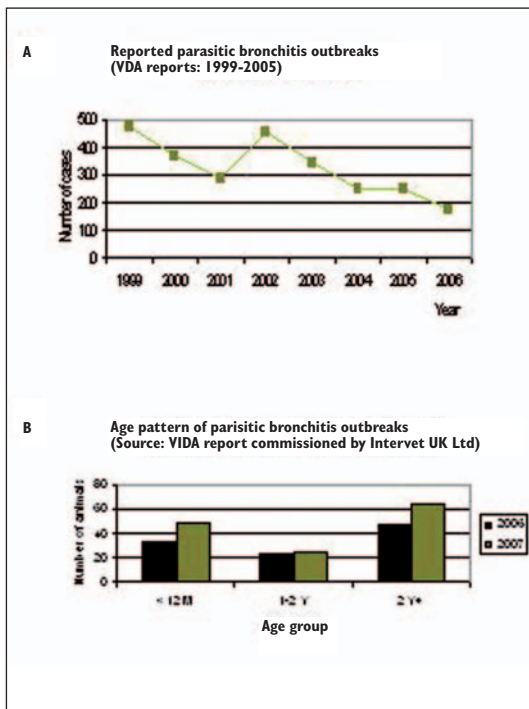

Fig. 3.

The increase in recorded diagnoses of parasitic bronchitis is thought to reflect the increasing commercial involvement and awareness of the disease, as well as the availability of an ELISA for detection of specific antibody. Most importantly though, the rise has probably been due to the fact that many farmers replaced vaccination with highly effective, long-acting anthelmintics in the 1980s (Connan 1993; Mawhinney 1996). For example, sales of the vaccine dropped considerably between the mid-1970s, when the number of vaccinates per annum totalled approximately 750,000 animals, compared with around 250,000 in the 1990s (McKeand 2000). Subsequently, with increased recognition of the disease, the numbers of 'new vaccine users' increased in the late 1990s, with most of these users representing farmers who had experienced an outbreak of parasitic bronchitis in the previous year (source: http://www.huskvac.co.uk/index.asp).

Coincident with the increase in the number of outbreaks, the age pattern of disease within herds also changed: there has been a substantial increase in the proportion of cases recorded in second year grazing animals or adult cows (for example see Fig. 3b: David, 1993, 1996, 1997; Robinson, Jackson & Sarchet, 1993; Williams, 1996). Indeed, parasitic bronchitis is now recognized as the commonest respiratory disease of adult cattle in the UK. In these animals, the disease can have a high morbidity and significant economic consequences via costs of treatment and reductions in milk yield, fertility and body weight (Woolley, 1997). Again, the altered pattern of disease has been attributed to a reduction in usage of the vaccine as farmers increased usage of anthelmintics to control lungworm in addition to the other common (gastrointestinal) nematodes. Although anthelmintics affect lungworm at high efficacy, protection against the nematode in later seasons can be compromised because of the unpredictable degree of immunity that develops following administration of **long-acting drugs**. In a VIDA report (commissioned by Intervet UK in 1999), the data collected indicated that, on average, of farms that stopped vaccinating, 63% had an outbreak of lungworm associated disease in succeeding years (source: http://www.huskvac.co.uk/index.asp). This report also highlighted that, in 1999, around 1000 herds experienced a parasitic bronchitis outbreak, the total number of animals involved being estimated at approximately 40,000.

CONTROL OF *D. VIVIPARUS*

The most effective way to control *D. viviparus* is to vaccinate. This should be performed in combination with strategic anthelmintic treatments to control other nematode species. When selecting anthelmintics, preference should be given to 'pulse release' type versus 'sustained release' formulations as this will assist in the stimulation of immunity not only against *D. viviparus* but also against gastrointestinal species such as *Ostertagia ostertagi* and *Cooperia oncophora*. Moreover, at all times anthelmintics should be used prudently because of the threat of drug resistance. Although the latter is a more recognized phenomenon in sheep (Kaplan 2004) and horses (Kaplan 2002), recent evidence suggests that cattle nematodes are also capable of developing the mutations, and other genetic alterations, that render worms resistant to even the most effective wormers (Pomroy 2006). On farms where vaccination has been stopped or not used previously, the vaccine can be used safely in lactating and pregnant animals with no adverse effects (Holzhauer *et al.* 2005).

TREATMENT OF PARASITIC BRONCHITIS

Benzimidazoles, levamisole and macrocyclic lactones are all effective against *D. viviparus.* Oral administration of drugs should be avoided in dyspnoeic animals. Where there are heavy infection levels (as evidenced by severe clinical disease), anthelmintic treatment can exacerbate clinical signs in some animals. Animals that are anorexic,

dyspnoeic and/or pyrexic should be housed. These animals may benefit from concurrent treatment with a non-steroidal analgesic. If pyrexia persists, antibiotics may be required to treat secondary bacterial infection. It should be noted that administration of anthelmintic early in infection (i.e. in the prepatent period or early patency) may not enable the development of protective immune responses (Hoglund *et al.*, 2003) so such animals should still be considered susceptible to disease in future. Parasitic bronchitis outbreaks have been recorded where up to 50% of lactating cows in a herd showed respiratory distress (Wapenaar et al. 2007). In this case, disease was precipitated by the introduction of susceptible heifers that apparently had amplified pasture contamination to an extent that disease developed in the younger as well as in the adult stock. Here, all animals were treated with a pour-on eprinomectin preparation after which one animal died. However, two weeks later, clinical signs were markedly improved and ten weeks after treatment, milk production improved from 23 to 28 kg/cow/d.

QUESTIONS FOR THE PANEL

1. What treatment regime do you institute for parasitic bronchitis in adult cows?

Richard Laven writes: All the outbreaks I have seen in adult cattle have involved animals showing a variety of signs from mild respiratory disease to severe dyspnoea. My anthelmintic choice is usually based on withhold periods, with products such as eprinomectin, which has no milk withhold, being first choice. I would always treat the whole group and make sure that the farmer or stockman knows that treatment may, at first, make the clinical signs worse in some animals. I have used other anthelmintics in dry cows, e.g. benzimidazoles and levamisole, and they seemed to be effective but the numbers were too small for proper comparison

Andrew White writes: On diagnosing an outbreak of lungworm in adult cattle, there are several considerations to take into account before deciding on a particular treatment.
1. Are these dairy cattle, are they in milk?
2. Are these beef cattle, are they ready for slaughter?
3. Are these animals dyspnoeic or pyrexic?

In this practice, the adult cattle which we see with bovine lungworm are usually in a dairy herd, and often in milk. Treatment therefore is usually pour-on eprinomectin. This is expensive but carries no milk withhold and is also easy to apply. If the affected cattle are beef suckler cows which are not destined for slaughter in the short term, then ivermectin can be used to good effect. However, I would recommend levamisole, repeated in three weeks time. This is because I have seen cattle become much worse and even die when treated with ivermectin. I believe that the sudden and total destruction of all the larval stages in the animal can exacerbate the clinical signs. I have only seen this happen with ivermectin-treated animals, an effect that doesn't seem to occur with levamisole.

If any of the cattle are pyrexic or severely dyspnoeic, I would recommend them to be housed for the rest of the grazing season and given one of the non-steroidal anti-inflammatory preparations at the same time as the anti-parasiticide. In practice however, I find that most farmers will treat with pour-on eprinomectin.

I feel it important to treat the whole group or herd rather than just those clinically affected. Also, having treated the animals, I would recommend a change of pasture, as some of those animals which are still in the very early stages of the disease will not have developed a sufficient immunity to be protected.

Keith Cutler writes: The treatment of husk, in my opinion, can present a huge challenge to the clinician.

Outbreaks of disease usually occur on a group basis with the severity of clinical signs varying between individuals within the group. Despite this individual variation it is reasonable, I believe, to recommend treatment of all the animals in the affected group. Different individuals may, however, require different treatment.

When considering the optimum treatment for a group or individual it is important to remember that, despite the clinical signs attributable to a lung full of live lungworm, the consequences of a lung full of dead lungworm (effectively causing inhalation pneumonia) may be significantly more severe. Treated animals may therefore deteriorate before they improve and some may even die as a consequence of the treatment (although treatment must be given). Owners must be warned of this possibility.

Treating adult cows raises further complications because of the consideration, which must be given to the milk withdrawal periods of products that may be chosen to treat dairy cows. In addition, the effect of the stress caused by handling affected and already debilitated animals in order to treat them must be considered.

Given the above discussion, when treating cases of husk the use of an anthelmintic is inevitable and in lactating dairy cows a single option is available to

avoid discarding large volumes of milk. Where milk withdrawal periods are not an issue (and in more seriously affected dairy cows) my preferred choice of anthelmintic would be for a levamisole-based product which is thought to have the advantage of paralysing the parasites rather than killing them. The aim is to avoid leaving a lung full of dead parasites. By paralysing the worms it is hoped that natural defence mechanisms (for example, the mucociliary escalator and coughing) will clear the bulk of the infection from the lungs. If necessary, treatment using a product from an alternative anthelmintic group can then be given to kill any residual parasite burden a week or two later when, because of the reduced burden, the chance of treatment making the clinical situation worse has also reduced.

In more severely affected animals I would adopt this latter approach to treatment irrespective of any milk withdrawal period which might have to be applied. (In such cases milk yield will already have been severely compromised anyway.) In addition, I would also give broad-spectrum antibiotic cover to address any secondary infection which may be present and non-steroidal anti-inflammatory drugs to reduce inflammation within the lungs and help hasten recovery.

James Breen writes: Anthelmintic treatment for adult cow cases would probably depend on the number of animals affected within the herd. The oral administration of a levamisole-containing product could be considered in individual non-lactating cattle as it has been shown to posses an immunomodulatory effect but may require repeat dosing if the environmental challenge was high. For groups of cattle and/or for animals that are difficult to handle, the use of ivermectin in the form of pour-ons or injections could be indicated; as well as excellent efficacy, the persistence of the drug would protect against potential re-infection. In addition, the use of eprinomectin would have the advantage of a zero withhold period for milk as well as a pour-on preparation when considering treatment for lactating dairy cattle. One disadvantage with the ivermectin is the mass worm death and potential for anaphylactic shock in cattle carrying heavy parasite burdens.

In all cases, I would offer concurrent non-steroidal anti-inflammatory drugs (meloxicam for non-lactating animals or ketofen if a zero milk withdrawal period was desirable) to reduce inflammation in the airways and broad-spectrum antibiotics, for example oxytetracycline, to control secondary infection if the animal(s) are febrile.

2. Where there has been an outbreak of disease on a farm which did not previously vaccinate

or has stopped vaccinating, what is your advice for control in all animals in the following, and subsequent, seasons?

Richard Laven writes: I would always recommend vaccination, as it is safe and effective, ensuring that the anthelmintic programme is designed to fit around the vaccination regime.

Andrew White writes: I believe that the most effective way to prevent an outbreak of bovine lungworm in a herd of cattle is by vaccination. This should be done before turnout for the first grazing season. If an outbreak does occur in an adult herd and the farmer has failed to vaccinate, then my advice would be to vaccinate the whole herd the following year and then all those animals going out for their first grazing season in subsequent years. He may, quite sensibly, argue that the animals clinically affected this year will be immune to further attacks but those herd members which were treated but did not show clinical signs, cannot be guaranteed to have developed sufficient immunity to prevent clinical signs. Having persuaded a farmer to prevent bovine lungworm by the use of vaccine, he should be made aware that this will not protect his herd from other gastro-intestinal parasites. The farmer should also be made aware that he should vaccinate any bought in cattle, even if they are no longer 'youngstock'.

Keith Cutler writes: The advice given varies from farm to farm but always involves vaccination, a review of anthelmintic use and a review of grazing policy.

Youngstock being turned out to grass for the first time should be vaccinated twice prior to turnout with an interval of four weeks between doses according to the manufacturer's data sheet. Care should be taken to treat the vaccine appropriately prior to use (it should be kept in a fridge but not frozen) for best results. Pastures known to be heavily contaminated with lungworm larvae should not be used for youngstock grazing and an anthelmintic treatment regime designed to maximise both parasite control and the development of immunity should be implemented. In situations where compromises have to be made, consideration should be given to re-vaccinating animals being turned out for their second grazing season, either with a full course of vaccine or with a single 'booster' dose,

I consider the regular administration of highly effective anthelmintics on a herd-wide basis to be disadvantageous in the long-term control of lungworm in cattle.

James Breen writes: I would certainly strongly recommend the vaccination of young animals before their first season at grass in the following and subsequent seasons, coupled with a chance to reinforce the unpredictable nature of the disease in terms of epidemiology. The control for other groups

would depend on which group(s) experienced the disease in the previous season. If it were adult animals, then I would advise vaccination of young stock prior to their second season at grass, at least for the subsequent season, as immunity is likely to be very low in this group. I think that vaccination in the first season alongside an anthelmintic program that will allow development of some immunity is essential - if this is achieved then future control should be assured. If the outbreak was associated with animals sourced from outside the herd then biosecurity issues (involving a host of potential pathogens!) need to be discussed, not least of which is the risks to any future stock entering what is now a herd with confirmed lungworm, e.g. the vaccinal status of a hire bull etc.

3. Do you think that the current vaccine could be improved upon? If so, what sort of improvements do you think would increase uptake of a lungworm vaccine by farmers?

Richard Laven writes: The vaccine is an excellent vaccine in terms of providing protection, but it does have two drawbacks.

It is an oral rather than an injectable formulation. It can be interfered with by anthelmintics - vaccine induced immunity can be affected by concurrent/ frequent anthelmintic treatment.

Both of these make vaccination more difficult to fit into modern farming regimes, particularly with the advent of very effective long acting anthelmintics. The main barrier to increased uptake is the fact that most farmers who don't vaccinate get away with it for most of the time even in the west of the UK.

Andrew White writes: The current vaccine has been available for about 50 years now. I have found it to be a very successful product and am not able to suggest any improvements to the vaccine itself. It could, perhaps, be made more user-friendly by introducing a multi-dose bottle and preset dose drenching gun but, because our farmers' requirements are so individual, I am not convinced that there would be a real benefit in this.

I feel that the real flaw in the system is that farmers rely on other gastro-intestinal anti-parasite drugs to control lungworm, and tend to forget that once these animals reach adulthood and enter a milking herd, because they no longer get a regular worming treatment, they are not protected against lungworm. Creating a greater awareness of this fact could, I feel, increase the uptake of the vaccine. We should, of course, impress on the farmers the importance of bio-security, and that stray cattle or common grazing with other herds always pose problems. The farmers, of course, will argue that a substantial price reduction would boost sales!

Keith Cutler writes: The efficacy of the currently available lungworm vaccine is not in question; it has, for years, been used as part of a successful parasite control programme. Vaccine failures are rare and often a perception rather than reality. A failure to prevent an outbreak of husk when vaccine has either not been stored in a fridge or frozen, if it has not been administered at the correct time or if only a single dose has been given, if worming regimes have not been optimal or if poor pasture management has resulted in an excessive challenge cannot really be blamed on vaccine failure!

From a farmer's perspective a single dose vaccine would encourage increased use and a price reduction is also likely to be advantageous!

James Breen writes: In terms of the efficacy of the vaccine then probably not – it must be one of the most cost-effective vaccines on the market for what is a highly unpredictable and sporadic disease with the potential to cause huge losses. The only improvements would be in the delivery of the antigen - an injectable format may improve uptake in those larger herds that are unwilling to administer oral doses to big groups of replacement animals.

REFERENCES
BOON, J. H., KLOOSTERMAN, A., VAN DER LENDE, T. 1984. The incidence of *Dictyocaulus viviparus* infections in cattle in the Netherlands. II. Survey of sera collected in the field. Veterinary Quarterly 6, 13-7.
BOS, H. J., BEEKMAN, J. 1985. Serodiagnosis of lungworm infection in calves using ELISA. Developmental Biology Stand 62, 45-52.
CONNAN, R. M. 1993. Calfhood vaccination for dictyocaulosis. Veterinary Record 133, 554-55.
DAVID, G. P. 1993. Increased prevalence of husk. Veterinary Record 135, 627.
DAVID, G. P. 1996. An epidemiological study of husk in adult cows in 32 UK herds (preliminary findings). Cattle Practice 5, 295-7.
DAVID, G. P. 1997. Survey on lungworm in adult cattle. Veterinary Record 141, 343-4.
HÖGLUND, J., GÅNHEIM, C., ALENIUS, S. 2003. The effect of treatment with eprinomectin on lungworms at early patency on the development of immunity in young cattle. Veterinary Parasitology 114, 205-14.
HOLZHAUER, M., PLOEGER, H. W., VERHOEFF J. 2003. Lungworm disease in dairy cattle: symptoms, diagnosis, and pathogenesis on the basis of four case reports. Tijdschr diergeneeskd. 128, 174-8.
HOLZHAUER, M., HOLLAND, W. G., PLOEGER, H. W. 2005. Preventive vaccination of lactating and pregnant heifers against lungworm: safety and protection in three dairy herds. Tijdschr Diergeneeskd. 130, 74-7.
KAPLAN, R. M. 2004. Drug resistance in nematodes of veterinary importance: a status report. Trends Parasitology 20, 477-81.
KAPLAN, R. M. 2002. Anthelmintic resistance in nematodes of horses. Vet Res. 33, :491-507.
MAWHINNEY, I C. 1996. Control of lungworm. Veterinary Record 138, 263
Mckeand, J. B. 2000. Vaccine development and diagnostics of *Dictyocaulus viviparus*. Parasitology 120 suppl, s17-23.
PLOEGER, H. W. 2002. Dictyocaulus viviparus: re-emerging or never been away? Trends Parasitololoy 18, 329-32.
POMROY, W. E. 2006. Anthelmintic resistance in New Zealand: a perspective on recent findings and options for the future. NZ Veterinary Journal 54, 265-70.
ROBINSON, T. C., JACKSON, E. R., SARCHET, R. W. 1993. Husk in heifers. Veterinary Record 132, 143.
WAPENAAR, W., BARKEMA, H. W., EYSKER, M., O'HANDLEY, R. M. 2007. An outbreak of dictyocaulosis in lactating cows on a dairy farm. Journal of American Veterinary Medicine Association, 231, 1715-8.
WILLIAMS, P. C. 1996. Control of lungworm. Veterinary Record. 138, 263.
WOOLLEY, H. 1997. The economic impact of husk in dairy cattle. Cattle Practice 5, 315-7.

Item code 094942