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Short communication

Failure to eradicate the lungworm *Dictyocaulus viviparus* on dairy farms by a single mass-treatment before turnout

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ABSTRACT

On two dairy farms it was attempted to eradicate lungworm, *Dictyocaulus viviparus*, by means of a single mass-treatment of all cattle that had been grazed the previous year(s), before turnout in the spring. Both farms experienced two years of lungworm outbreaks in the adult dairy herd prior to this study. Following confirmation that both herds contained lungworm carriers, all animals older than approximately 6 months were treated with eprinomectin in March 2007. One week after treatment none of the animals were shedding lungworm larvae. Subsequently, animals were pastured according to normal farm routine. From August to November all first-calving heifers were coprologically and serologically monitored for lungworm infection. During 2007 both farms remained lungworm-negative and did not report any clinical sign indicative for a lungworm infection. The following year, on one of the farms replacements grazing on cow pastures, started showing signs of parasitic bronchitis which was serologically confirmed. The other herd remained free of parasitic bronchitis until at least the fourth year after the mass treatment, although some coughing was noticed in 2008 among first-lactation heifers. It was concluded that a single mass-treatment before the grazing season may be useful to break a series of annual lungworm outbreaks. However, it is not a secure method to prevent parasitic bronchitis for more than one year.

1. Introduction

Since the early 1990s the incidence of lungworm outbreaks in adult cow herds has increased considerably in the UK (Van Dijk, 2004). Similar observations were made in The Netherlands (Holzhauer et al., 2003). Lungworm, *Dictyocaulus viviparus*, can cause a severe bronchitis with edema, emphysema, and inflammatory responses. Clinical signs involve coughing, increased respiration rates and a more abdominal respiration. The economic losses of a lungworm outbreak may amount to hundreds of Euro's per cow (Woolley, 1997; Holzhauer et al., 2011).

Unlike regions with relatively mild Atlantic winters, such as Ireland and parts of the United Kingdom, year-to-year transmission of infection mainly occurs through carrier animals in The Netherlands (Eysker, 2002). It may be possible that infective lungworm larvae survive on pasture during the winter, but these occasions are extremely rare. Consequently, during the winter stabling period virtually the whole lungworm population on a farm resides inside the host with little or no refuge outdoors. This situation is quite similar to the one for *Haemonchus contortus* in north-western European countries where the entire *H. contortus* population resides in the ewes in spring. Consequently, Waller et al. (2006) concluded that eradication of *H. contortus* by means of mass treatment should be a realistic possibility. The same might apply for *D. viviparus*. Here, we describe the short-term success to eradicate lungworm

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from two farms by mass treating the entire host population shortly before turnout.

2. Farms and procedures

The two farms (A and B) in this study are located south-east of the city of Apeldoorn in the province of Gelderland. Farms owned a total of 160–190 animals (cows and replacements), of which 120–130 were lactating or dry cows. The farms are neighbors and experienced lungworm outbreaks during 3–4 years preceding 2007, the year this study commenced. Outbreaks repeatedly resulted in respiratory symptoms such as coughing, reduced milk yields and loss of body condition. The outbreaks were serologically confirmed by means of an ELISA based on a purified adult lungworm antigen (Ceditest™Lungworm ELISA; Cornelissen et al., 1997) at the Dutch Animal Health Service (GD).

In March 2007, before turnout, all cows and young stock that had been pastured the previous year were individually examined coprologically for presence of lungworm larvae. From each animal at least 30 g of faeces, if present, was used in a standard Baermann procedure (Eysker et al., 1994). From approximately 20% of the animals between 10 and 30 g of faeces was examined. In addition, all heifers that had calved for the first time since housing in the autumn 2006 or should calve before December 2007 were blood sampled. About a week later all animals older than approximately 6 months were treated with eprinomectin (Eprinex®, Merial) at the recommended dose. One week after this treatment, all animals were examined again for lungworm larvae in the faeces as described above.

After the second faecal examination for larvae, animals were turned out following normal farm routine and pasture management practices.

From August until November 2007 each farm was visited monthly to sample the first-calving heifers that were introduced to the cow herd between housing last year until 3–4 weeks before the current visit. This sampling procedure was chosen because first-calving heifers are the best suited to monitor presence or absence of circulating lungworm infection in a cow herd as they are likely the least immune, if not even lungworm-naïve at the start of the grazing season (Eysker, 2002; Ploeger, 2002; Ploeger et al., 2011). The heifers were sampled for both faeces and blood. At each visit the farmers were asked about clinical signs, applied grazing management so far and recent introductions of heifers. At the same time farmers were asked about the calves and yearlings, including if any treatments had been applied for whatever reason. In addition, pastured replacement stock was sampled for blood and faeces at the last visit in November (after housing) and at each of the other visits whenever possible. It was agreed that visits would stop as soon as evidence of lungworm infection would appear.

Following 2007, both farms were visited again the end of March 2008 before the next grazing season. Faeces was collected from all cows and replacement stock >1 year old to check for lungworm larvae. After that, it was left to the veterinarian to keep in touch with both farmers and to report as soon as clinical signs appeared that could point at re-appearance of lungworm infection. In that case, blood samples would be taken for serological examination (Ceditest®Lungworm ELISA) to confirm presence of lungworm. The study was ended in September 2011, i.e. almost 5 complete grazing seasons after the mass treatment in spring 2007.

3. Results

In March 2007, prior to the grazing season, faeces examination confirmed that carriers were present among the cows in both herds. In one herd two cows were shedding lungworm larvae, with 1 and 7 larvae found. In the other herd one cow was positive, with 3 larvae found. These numbers imply counts of <0.25 larvae/g faeces. In both herds no positive samples were found among the replacements (calves and yearlings). Shortly before the grazing season all sampled replacement heifers (lactating or still pregnant) were serologically negative in the lungworm ELISA. A week after the eprinomectin treatment all animals were tested negative with the Baermann.

During the grazing season of 2007 all examined faeces samples were negative on both farms. The same applies for all examined blood samples except one collected in August. For the latter sample The Ceditest™ ELISA returned a weak positive result (optical density ratio just 2% higher than the cut-off percentage). All subsequent monthly samples were negative and well below the cut-off value. The ELISA is considered to be very sensitive in detecting adult worm burdens and once present increases to levels well beyond the cut-off remaining at clearly positive levels for at least more than a month (Cornelissen et al., 1997). Therefore, the isolated single weak positive sample was considered to be false positive and to have been caused by other factors than a circulating lungworm infection. No clinical respiratory symptoms indicative for a lungworm infection were observed on both farms during 2007.

In March 2008, before the next grazing season, none of the examined cows and replacements were found shedding lungworm larvae. During the subsequent grazing season, one farm (farm A) reported that a few first-lactation heifers were coughing in September. However, subsequently collected blood samples were negative for lungworm antibodies and the coughing disappeared without any treatment. The other farm (farm B) reported clinical symptoms in the yearling replacement stock early September 2008. The suspected lungworm infection was serologically confirmed using the Ceditest™Lungworm ELISA at the GD. These yearlings were grazed on cow pastures after the cows. The cows remained free from clinical respiratory symptoms. The farm had not purchased new stock in 2007 or in 2008. It could not be determined where this lungworm infection originated from.

During 2009, on farm A that remained negative in 2008 ten first-lactation heifers were tested for lungworm antibodies in October. All were negative. No clinical respiratory signs were observed during the grazing season. Farm B reported again clear symptoms of lungworm infection. This time the observations were made among the first-lactation heifers. Examination of the faeces for lungworm larvae confirmed symptoms were caused by lungworm infection.