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References


Effect of three sustained-release devices on parasitic bronchitis in first year calves

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The effect of three intraruminal sustained-release devices (SRD) against Dictyocaulus viviparus infection was tested in five groups of six calves. Group 1 served as untreated controls, and groups 2, 3 and 4 were dosed with a levamisole SRD, a fenbendazole SRD, and an ivermectin SRD, respectively. Group 5 was vaccinated against lungworm and received a levamisole SRD. The calves were turned out on May 28 and the devices given seven days later. All the calves received trickle infections with a total of 200 lungworm larvae between 9 and 34 days after turnout. They were housed on October 28, challenged with 5000 lungworm larvae and slaughtered three weeks later. No clinical signs of parasitic bronchitis were observed during the study. The treated groups gained significantly more weight (P<0.05) than the controls, but did not differ among themselves. Larvae were first detected in the faeces of the control group between 25 and 32 days after the infection, and there was a group mean of 21 larvae per gram (lpg) after 60 to 80 days, after which the lpg gradually decreased. In group 2, larvae were detected near the end of the grazing season and never exceeded a group mean of 1.5 lpg. In group 3, a very low larval output was observed after housing (group mean 0.1 lpg). Groups 4 and 5 never became patent. The results of an ELISA followed the pattern of larval output; optical densities above the cut-off value were recorded in groups 1, 2 and 3. On the basis of worm recoveries after challenge, group 1 was immune. Group 4 had significantly more lungworms than group 2. There were no significant differences in worm numbers between groups 2, 3 and 5, but the worms in group 5 were retarded in growth (P<0.05).


Materials and methods

Experimental design

Five adjacent permanent pastures of 1 hectare each, divided into two parts of 0.5 hectare were available (pastures 1a, b to 3a, b). They had been grazed by sheep in 1995, but not by cattle for several years. They were mown and fertilised before the start of the trial. The adjacent pastures were 4 m apart and had electric...
The Veterinary Record, June 20, 1998

Fig 1: Mean larval output (larvae per gram of faeces) by the calves of the five groups (group 1, controls; group 2, levamisole SRD; group 3, fenbendazole SRD; group 4, ivermectin SRD; group 5, vaccination and levamisole SRD)

Fig 2: Mean ELISA optical densities at 450 nm of the calves of the five groups (group 1, controls; group 2, levamisole SRD; group 3, fenbendazole SRD; group 4, ivermectin SRD; group 5, vaccination and levamisole SRD)

fences to prevent contamination from pasture to pasture (Borgsteede and others 1988, 1990). Thirty female Friesian Holstein calves, born in the first half of February 1996, were reared indoors free of parasites. They were randomly allocated to five groups. The six calves in group 5 were vaccinated against lungworm on April 16 and May 14 (Bovilis; Intervet). On May 28 they were turned out on to pasture 5b, and the other 24 calves were turned out on to pasture 1b. On June 4, these 24 calves were removed from pasture 1b, divided into four groups (1 to 4) and put out on to pastures 1a to 4a. On the same day, the calves of groups 2 to 5 were dosed with an intraruminal device. The resultant groups were:

1) Pasture 1, an untreated control group;
2) Pasture 2, a levamisole SRD group (Chronomintic bolus; Virbac);
3) Pasture 3, a fenbendazole SRD group (Panacur SR bolus; Hoechst);
4) Pasture 4, an ivermectin SRD group (Ivomec SR bolus; MSD AgVet);
5) Pasture 5, the vaccinated group treated with a levamisole SRD (Chronomintic bolus; Virbac).

All the calves were infected orally with 200 lungworm larvae according to the following schedule: 40 larvae on June 13 and 17; 30 larvae on June 20 and 24; 20 larvae on June 27 and July 1; and 10 larvae on July 4 and 8. Each group of calves was moved according to the following regimen: on July 16 (a→b), July 29 (b→a), August 19 (a→b) and September 9 (b→a); from September 20 until they were housed on October 28 both halves of each pasture were available.

During the grazing season, the animals were inspected daily for clinical signs. The course of the lungworm infection in each calf was monitored weekly by faecalysis of 10 g of faeces taken from the rectum. Every four weeks, the samples were also used to detect eggs of gastrointestinal helminths by a modified McMaster technique, followed by the culture of 25 g faeces at 27°C for the identification of infective nematode larvae. Starting on April 16, a blood sample was collected weekly from the calves of group 5, and from May 28 onwards from all the calves. From eight weeks after the first infective dose blood samples were taken once every two weeks. The antibody response against a specific lungworm antigen was monitored with an ELISA (de Leeuw and Cornelissen 1993). The calves were weighed at turn-out (May 28) and on the day they were housed (October 28).

Challenge infection

After housing, three calves of each group were challenged on October 30 with 5000 lungworm larvae less than two weeks old, and the remaining 15 calves were challenged on October 31. The calves were slaughtered on November 20 and 21. Lungworms were collected by the perfusion technique described by Andrews and James (1994). The perfused lungs were chopped into small pieces and weighed. A sample of 10 per cent of the lungs was baermannised for the detection of larval stages. The worms collected were measured and classified according to Eysker and others (1990).

Statistics

The differences in weight gain and numbers of worms between the five groups were analysed statistically by calculating the least significant difference (LSD) at the 5 per cent level.

Results

Clinical observations

The sustained release devices were administered without problems. Coughing was heard occasionally during the grazing season, but it occurred regularly only in calves of group 1, particularly in August; no treatment was necessary. After the challenge infection, no coughing was heard. While it was housed one calf of group 2 regurgitated its bolus.

Counts of lungworm larvae in the faeces

The results of the larval lungworm counts are shown in Fig 1. All the control calves became patent and larvae were detected 32 days after the initial infection. Between day 56 and 63, a second increase in larval output was observed. The highest group mean was 21 lpg. Five calves of group 2 became patent 123 days after the first infection but at a much lower level; the highest group mean was 1.5 lpg. In group 3, five calves became patent. One calf had larvae in the faeces at 32 days and 39 days, but then became negative. Larval output in the other animals of group 3 started after 137 days, later than in the group 2 calves, with the highest group mean of 0-1 lpg. No larvae were detected in the faeces of the calves of groups 4 and 5 throughout the study.

Counts of gastrointestinal nematodes

Strongylo-type eggs were occasionally found in the faeces of all the groups but not in all the calves in a group. The faecal egg count never exceeded 25 eggs per gram (epg). An egg of Nematodirus battus was found in a calf of group 1 and one egg of N helvetianus was found in a calf of group 5. Larval cultures revealed the presence of Haemonchus contortus in calves of groups 1, 3 and 5. Ostertagia species larvae were occasionally

![Graph showing larval output](https://via.placeholder.com/150)

![Graph showing ELISA optical densities](https://via.placeholder.com/150)
found in cultures from groups 2 and 3, and Cooperia oncophora larvae were observed in cultures of group 3 only. No larvae were found in the cultures from group 4.

**ELISA results**

After vaccination, a small increase in optical density (OD) was observed in the antibody response of samples from the calves of group 3 (Fig 2). However, this increase did not reach the cut-off value (0.25). Samples from the calves of the control group showed a sharp increase in OD between 25 and 39 days after the first infection, and the OD then decreased slowly until the end of the study. The OD in group 2 reached the cut-off value after 137 days, and in group 3 after 151 days. In groups 4 and 5 the cut-off value was never reached.

**Weight gain**

The calves in the treated groups grew significantly better than the control calves during the period on grass (P<0.05). There were no significant differences between the groups of treated calves. The control calves had a mean weight gain of 808 g per day, but the calves of groups 2, 3, 4 and 5 gained on average 962 g, 926 g, 914 g and 948 g per day, respectively.

**Postmortem lungworm counts**

The numbers of lungworms and their developmental stage are shown in Table 1.

After challenge, the calves were kept for 21 days before they were slaughtered. This period is too short for the development of fully grown adult worms. Thus, the adult worms which were found must have grown from larvae on the pasture before the challenge infection was given. The total number of worms in group 4 was significantly higher than in groups 1 and 2 (P<0.05), but not significantly higher than in groups 3 and 5. The numbers of adult worms found in groups 1, 2 and 3 were significantly lower (P<0.05) than in groups 4 and 5. The number of young adult worms was significantly higher in the calves of group 4 (P<0.05). The numbers of juvenile worms and early 5th stage worms were significantly higher in the calves of group 5.

**Discussion**

The pattern of lungworm infestation which is characteristic in calves on the majority of Dutch farms was successfully imitated. The low level of infection, representing a small overwintered infection or an infection brought on to pasture by carrier animals, caused a patent infection in the control calves which contaminated the pasture. This contamination resulted in a second generation of lungworms with a high larval output in the faeces. The fact that the larval output then decreased indicates that the pasture contamination did not result in a third generation of lungworms in the calves, probably because a strong immunity had developed.

This natural build-up of immunity occurred without obvious clinical signs of parasitic bronchitis. However, the control calves gained significantly less weight than the treated calves, although their mean weight gain of 808 g per day is quite acceptable in practice. The results of the faecal examinations showed clearly that only the lungworm infection could have been the cause of the difference, because in all the groups the infections with gastrointestinal nematodes were negligible. This was not surprising considering that the pastures had not been grazed by cattle for several years. The absence of nematodes in the lungworm larvae in the levamisole SRD-treated group after 123 days was also not surprising, because it was observed by Borgsteede and others (1988, 1990) that small numbers of larvae were excreted by animals treated with this SRD.

The lungworm infections in the calves of groups 2 and 3, which were detected by faecal examinations and confirmed postmortem, were also indicated by the ELISA results. This observation supports the hypothesis that this ELISA demonstrates the presence of adult worms in the lungs (de Leeuw and Cornelissen 1993), and explains why the OD in the sera of the vaccinated calves (group 5) did not reach the cut-off value. The small increase in OD observed after the second vaccination could have been due to the presence of a few insufficiently irritated larvae in the vaccine and their development to the adult, but not sexually mature, stage. It also explains why the OD in the sera of the calves of groups 4 and 5 did not reach the cut-off value after challenge, because the time between challenge and slaughter was too short to induce a measurable increase in OD.

Although no parasite-naive controls were used to confirm the infectivity of the challenge the authors are confident that these larvae were fully infective. A donor calf, used to maintain the strain was infected at the same time with the same batch of larvae, and showed a normal infection pattern with patency starting at 24 days after infection. The postmortem worm counts therefore clearly indicated the stronger immunity built up by the control calves during their period on pasture. In terms of worm counts and worm development, immunity at the time of challenge was weakest in the calves of group 4, in which no lungworm larvae were found in the faeces during the whole study, the ELISA titres did not reach the cut-off value and no adult lungworms were found postmortem. The results suggest that the 200 larvae which the calves were challenged with during their first period on pasture were probably the only contact with lungworm for these calves and that after challenge the growth of the worms was not delayed owing to the lack of an immune response. As a result they carried a significantly larger number of young adult worms. These worms also showed a remarkable homogeneity in length compared with the worms from the calves in groups 2, 3 and 5. This homogeneity of development was particularly absent in the calves of group 5. A possible explanation is that these calves had been vaccinated they still had some immunity which was not impaired by the levamisole SRD. It is known that the immunity which is induced by vaccination declines in the absence of a challenge (Düwel 1963). The vaccinated calves were, like the other calves, infected experimentally after turn-out, but it is likely that the levamisole SRD prevented these larvae from presenting an immune stimulus. This likelihood is supported by the results of the ELISA, in which the OD did not rise above the cut-off value. Because there was no larval output in the vaccinated calves, there was no boost to the immunity induced by vaccination. It is therefore recommended that this levamisole SRD should not be used in combination with vaccination; the bolus alone is sufficient to prevent lungworm infection under the conditions which prevail in the Netherlands.

Although there were differences between the parasitological and immunological effects induced by the three SRDs, it is not clear what these imply for the second grazing season. Even if calves do not develop a solid immunity to lungworm in their first grazing season, they will not necessarily develop problems in the second grazing season or later. If lungworm-naive animals are exposed to small infections in the second grazing season, they may well be able to build up immunity in a natural way. The build-up and maintenance of immunity to lungworm still depends on factors such as the number of lungworm larvae ingested and the pattern of ingestion.

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**TABLE 1: Mean numbers of Dicyocaulus viviparus recovered from each group after challenge with 5000 lungworm larvae**

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>Adult</th>
<th>Young adult</th>
<th>Juvenile</th>
<th>EL-5</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control</td>
<td>1-2a</td>
<td>Oa</td>
<td>Oa</td>
<td>0a</td>
<td>1-2a</td>
</tr>
<tr>
<td>2</td>
<td>SRD with LEV</td>
<td>0.8a</td>
<td>18.0a</td>
<td>5.7a</td>
<td>0a</td>
<td>24.5a</td>
</tr>
<tr>
<td>3</td>
<td>SRD with FBZ</td>
<td>7.0a</td>
<td>34.2a</td>
<td>22.6a</td>
<td>0a</td>
<td>64.2a</td>
</tr>
<tr>
<td>4</td>
<td>SRD with IVM</td>
<td>0a</td>
<td>114.2a</td>
<td>43.3a</td>
<td>7.2a</td>
<td>164.7a</td>
</tr>
<tr>
<td>5</td>
<td>SRD with LEV</td>
<td>0a</td>
<td>24.0a</td>
<td>60.7-21.3a</td>
<td>126.0a</td>
<td></td>
</tr>
</tbody>
</table>

SRD Sustained release device, LEV Levamisole, FBZ Fenbendazole, IVM Ivermectin

In each column mean numbers with different superscripts are significantly different (P<0.05)
Thoracic oesophageal abscess in a pony

S. L. Freeman, I. M. Bowen, C. M. Marr

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OESOPHAGEAL obstruction or "choke" is a common condition in horses. It is usually caused by intraluminal impaction of ingested material; however, strictures, diverticula, neoplasia, abscesses and cysts can also lead to partial or complete oesophageal obstruction (Moore and Kintner 1976, Scott and others 1977, Roberts and Kelly 1979, Freeman 1982, Green and others 1986, Ford and others 1987, Orsini and others 1988, Boy and others 1992, Campbell-Beggs and others 1993). Strictures and diverticula can occur as congenital conditions, but most frequently are the sequelae of mucosal necrosis caused by impaction of ingested material (Freeman 1982, Stick 1987). Neoplasia of the equine oesophagus is usually malignant and therefore associated with other clinical signs, such as weight loss (Moore and Kintner 1976, Green and others 1986, Ford and others 1987, Boy and others 1992, Campbell-Beggs and others 1993). There are no reports of oesophageal abscesses in the horse. This case report describes the clinical findings, diagnosis and treatment of an oesophageal abscess in a pony.

A 32-year-old pony gelding was presented with partial oesophageal obstruction of 48 hours duration. The referring veterinary surgeon had been able to pass a nasogastric tube with some difficulty into the stomach; however dysphagia and the regurgitation of food material persisted despite repeated lavage with water via a nasogastric tube.

On presentation, the horse was bright and in good bodily condition. Heart rate, respiratory rate and temperature were normal. Thoracic auscultation was also normal but there was a dry cough. The right mandibular lymph node was enlarged. Oesophageal endoscopy revealed a soft tissue mass in the thoracic oesophagus at a distance of 140 cm from the external nares. The mass was yellowish in appearance, with a smooth surface and regular margins. It was spherical, approximately 4 cm in diameter, and projected from the left dorsal oesophageal wall into the oesophageal lumen. The distal oesophagus and stomach appeared grossly normal.

Lateral thoracic radiography revealed normal lung fields. An ovoid radiopacity was present in the region of the thoracic oesophagus, caudal to the heart base. There was gas dilation of the adjacent oesophagus. Contrast radiography of the oesophagus was performed using a mixture of barium powder (E-Z-Paque; E-Zem) and molasses administered orally. The mass was outlined as a discrete, regularly margined ovoid filling defect (5 x 6 cm) within the contrast material (Fig 1). The oesophagus immediately proximal and distal to the mass was dilated, but the oesophageal wall was smooth and regular.

In view of the age of the horse it was considered important to identify or exclude the possibility of neoplasia. Other differential diagnoses included an oesophageal cyst, granuloma or abscess. Further investigation was therefore performed with the aim of identifying any neoplastic process and the extent of involvement of other organs.

There was mild anaemia (haematocrit 25 per cent), increased serum globulin and fibrinogen (80 g/litre and 3-4 g/litre, respectively) and a marked lymphopenia (total white cell count 5.52 x 10^9 cells/litre, lymphocyte count 0.565 x 10^9 cells/litre), which was consistent with inflammation and sepsis. Ultrasonographic examination of the thorax and abdomen was unremarkable. Cytological examination of peritoneal fluid was normal. The pleural fluid had an increased total nucleated cell count (229 x 10^9 cells/litre), which were predominantly neutrophils (90 per cent), and was compatible with sepsis. No neoplastic cells were identified. Nasal and tracheal aspirates of the mass were obtained under endoscopic guidance, using an 18 gauge needle attached to a catheter. The tissue recovered contained bacteria, neutrophils, macrophages, squamous cells and some plant material.

The findings were consistent with a diagnosis of a penetrating oesophageal foreign body which had resulted in a septic pleuritis and secondary oesophageal abscess formation. The horse was treated with 15,000 iu/kg procaine penicillin (Duphane; Solvay-Duphar) intramuscularly once daily, 7.5 mg/kg neomycin and 15,000 iu/kg penicillin (Neomycin Penicillin; Intervet) intramuscularly once daily, and 2 mg/kg phenylbutazone intravenously twice daily (Equipalazone; Arnolds), for seven days. During this period food was gradually reintroduced. The pony progressed to eating concentrates and grass, although a degree of dysphagia remained. After seven days of treatment, the mass was no longer endoscopically visible and the mass area of inflammation was the only abnormality identified in this region. Similarly, radiographic examination showed no evidence of the intraluminal mass or oesophageal dilatation (Fig 2). The peripheral white cell count had increased to 8.67 x 10^9 cells/litre (neutrophils, 6.63 x 10^9 cells/litre; lymphocytes, 1.43 x 10^9 cells/litre).

After seven days, the horse was discharged from the hospital, and treatment continued with 30 mg/kg potentiated sulphonamides (Equisure; Boehringer Ingelheim) administered orally once daily for two weeks and 2 mg/kg phenylbutazone administered orally once
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