Impact of sub-clinical coccidiosis in cattle

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Coccidiosis in cattle is a ubiquitous disease that is known to cause moderate to severe and sometimes lethal, enteritis with fever, loss of appetite and haemorrhagic diarrhoea, especially in young calves between the ages of three weeks and 10 months.

Adult cattle are usually the asymptomatic carriers of the disease and 16-27% are said to excrete low levels of oocysts which contaminate the environment for calves to become infected. Calves show higher levels of oocyst excretion when infected and contaminate the environment further for new calves to become infected.

This environmental contamination results in almost all calves becoming infected by 10 months of age. Even on severely infected farms only about 20% of the calves that become infected show actual clinical symptoms of the disease.

This means that 80% or more of the calves are subclinically infected and it has been stated in many publications that this subclinical disease will cause severe production losses in calves, but this has seldom, if ever, been quantified. Impaired growth rates and reduced feed conversion have been stated in many publications that this subclinical disease in young calves when given before the suspected clinical symptoms develop.

The timing of this dose is based upon previous history of the disease on a particular farm and the stress factors involved in the development of clinical disease.

This multicentre study on six separate sites in three different countries studied the effect of subclinical coccidiosis in both beef and dairy calves on farms where clinical coccidiosis was known to occur.

Materials and methods

Six separate farms where clinical coccidiosis was suspected were identified in Belgium (two farms), France (three farms) and Germany (one farm) and the presence of E. bovis and/or E. zuernii was proven by means of faecal isolation and identification on each of the six farms prior to the start of the study.

Both male and female calves between the ages of three weeks and four months were included in the study and were of different breeds (Belgian Blue Whites, Charolais, Salers and Holstein-Friesians). Body weights varied between 67-126kg at the start of the study.

At each site the calves were allocated to two groups of similar size and one group was treated orally with 0.25% diclazuril (Vecoxan, Janssen Animal Health) at a dose level of 1mg/kg body weight. The other group was left as controls and not treated at all or else given a placebo treatment of water or skimmed milk.

No calves had received any anticoccidial treatment prior to the trial and they were weighed at the time of treatment and again three weeks later.

Faecal samples were collected daily for three weeks on one site in Germany but every second day on the other trial sites and the number of oocysts per gram of faeces (OPG) was determined on each sample as well as identification of the species of Eimeria present.

A total of 231 calves were involved in the study, of which 116 were in the treated group and 115 in the control group.

Results

Faecal OPG counts.

No difference in oocyst counts was present between the groups at the start of the trial but by day four after treatment the treated group showed a statistical difference (P<0.0001) in OPG compared to the control group. The species of coccidiosis present at all the sites except one were predominantly E. bovis. The one site was mainly due to E. zuernii.

The long term effect of treatment, such as, the mean area under the curve for the sum of E. bovis and E. zuernii for the 21 days of treatment, varied between 67-126kg at the start of the study.

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Table 1. Oocyst count per gram of faeces at day one and five.

<table>
<thead>
<tr>
<th>Group</th>
<th>Day one Geometric mean (range)</th>
<th>Day five Geometric mean (range)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>22.2 (0.97-2)</td>
<td>0.2 (0.150)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Control</td>
<td>13.7 (0.16-2)</td>
<td>9.8 (0.110.4)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Body weights at day one and 21.

<table>
<thead>
<tr>
<th>Group</th>
<th>Day one Mean weight (kg)</th>
<th>Day 21 Mean weight (kg)</th>
<th>Mean weight gain</th>
<th>Difference (kg)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>91.5</td>
<td>107.8</td>
<td>16.4</td>
<td>+2.7</td>
<td>0.003</td>
</tr>
<tr>
<td>Control</td>
<td>94.0</td>
<td>107.6</td>
<td>13.7</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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ages of calves and so weight gains were considered to be more important than the initial weights.

At all but one of the trial sites the treated calves had a better weight gain than the control calves.

When the data of the six farms is pooled, the treated calves had a significantly higher average weight gain of 2.7kg compared to the untreated calves which represents a 20% increase.

Haemorrhagic diarrhoea was only seen in 2.2% of the calves.

A wider definition of calves suffering from clinical disease was taken as any calf that showed some diarrhoea for three consecutive days and at least a positive faecal oocyst count during an eight day span in the same period. The rest of the calves were considered to be suffering from subclinical coccidiosis.

According to this definition 15.7% (18 of 115) of the calves in the control group were considered to be suffering from clinical coccidiosis and the remaining 84.3% (97 of 115) of the calves were suffering from subclinical coccidiosis.

The weight gains in these clinically and subclinically affected calves compared to the weight gains in the treated calves (Table 3).

### Effective treatment

The metaphylactic treatment of the diclazuril was effective in reducing the OPG count considerably within four days of treatment and also over the full 21 days of the trial.

Clinical coccidiosis, as can be expected, results in greater weight losses (6.8kg per calf) than those seen in calves suffering from subclinical coccidiosis (2.0kg per calf).

The effect of subclinical coccidiosis, when defined as calves shedding oocysts in the faeces for three consecutive days, on weight gains, however, is considerable, when we consider that a far greater number of calves are suffering from subclinical coccidiosis than the number that suffer from clinical disease (84.3% versus 15.7% respectively). The total weight losses due to the subclinical disease is calculated in Table 4.

From Table 4 it can be clearly seen that the majority of the weight loss seen in a calf herd suffering from coccidiosis is due to the high number of calves (84.3%) suffering from subclinical coccidiosis, namely 61% of the losses and although clinical disease results in higher individual calf weight losses, the total weight loss due to the clinical cases (15.7%) is only 39% of the total weight lost in the herd.

### Conclusion

In calf herds where clinical coccidiosis is seen it is usually only the clinical cases that are treated, by which stage severe damage to the intestine has already occurred resulting in poor performance and often stunted calves.

Because the total weight loss in the herd is greater in the subclinical cases of the disease and because the numbers suffering from this latter form of coccidiosis is always greater than the clinical cases, more attention should be paid to treatment of subclinical cases than has been done in the past. Some 0.25% diclazuril when used as a metaphylactic treatment reduces the losses due to clinical and subclinical coccidiosis in the herd and will result in far lower economic losses than when only clinical cases are treated.

The timing of this metaphylactic treatment is important and should be given as close to the expected outbreak as possible.

This, in practice, is often difficult to achieve but should be based on the previous history of the farm and is often linked to periods of stress in the farm management.

If the metaphylactic treatment is not given on time then it is suggested that when the first cases of coccidiosis are seen the clinically affected calves should be treated and at the same time all the remaining calves in the group are given a metaphylactic treatment to reduce the severe losses that could occur from the subsequent clinical and subclinical disease.

### Table 3. Weight gains in clinical and subclinical calves.

<table>
<thead>
<tr>
<th>Group</th>
<th>Status</th>
<th>Mean weight gain per head (kg)</th>
<th>Weight gain loss per calf (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>Reference</td>
<td>16.4</td>
<td>14.4</td>
</tr>
<tr>
<td>Control</td>
<td>Sub-clinical</td>
<td>14.4</td>
<td>-2.0</td>
</tr>
<tr>
<td>Control</td>
<td>Clinical</td>
<td>9.6</td>
<td>-6.8</td>
</tr>
</tbody>
</table>

### Table 4. Weight losses in all animals.

<table>
<thead>
<tr>
<th>Group</th>
<th>Status</th>
<th>Number of calves</th>
<th>Weight loss per calf (kg)</th>
<th>Lack of weight gain (kg)</th>
<th>Losses (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treated</td>
<td>Reference</td>
<td>115</td>
<td>0.000.0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Sub-clinical</td>
<td>97</td>
<td>-2.0-194</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Clinical</td>
<td>18</td>
<td>-6.8-122</td>
<td>39</td>
<td></td>
</tr>
</tbody>
</table>