The possible role of *Ostertagia circumcincta*, coccidiosis and dietary protein level in the development of swelling disease in Angora goat kids

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ABSTRACT
A trial was carried out to investigate the possible role of *Ostertagia circumcincta*, coccidiosis and dietary protein level in the diet in the development of swelling disease in Angora goat kids. Eighty Angora goat kids were bought from 5 producers whose flocks had a history of swelling disease. These kids were kept in enclosures at Grootfontein Agricultural Development Institute near Middelburg (Eastern Cape) for the duration of the experiment. The animals received a combination of the following treatments: a) high protein vs normal protein diet; b) *Ostertagia circumcincta* infection or no *O. circumcincta* infection; c) coccidial infection or no coccidial infection. Data recorded included weekly body weight, weekly total plasma protein levels (TPP), weekly faecal egg counts, weekly coccidial oocyst counts and haematology at Weeks 1, 5, 9, 13 and 16. The goats were also monitored daily for any clinical symptoms. There was no specific trend in any of the parameters measured among the different treatment groups at any stage during the experimental period. The goats were shorn during Week 10 of the experiment. On Monday 6 September 2004 (Week 12 of the study), 19 of the goats developed some subcutaneous oedema. The Saturday (4 September 2004) was rather hot (30 °C), followed by very cold rainy conditions (11 °C) on Sunday (5 September 2004). Twelve of the goats developed what can be described as little oedema, while 7 developed moderate oedema. The number of goats that developed oedema was fairly evenly distributed among the various treatment groups. As far as the specific treatments are concerned, more goats on the normal protein diet developed moderate oedema than the goats on the high protein diet. Body weights of goats that developed moderate oedema were lower throughout the experimental period than body weights of goats that developed little or no oedema, while TPP of goats that developed moderate oedema were lower from Week 5 of the study onwards. There were also no significant differences at any stage throughout the experimental period in faecal egg counts, faecal coccidial oocyst counts or any of the blood parameters between goats that developed moderate oedema, little oedema and those that did not develop any oedema. No goats developed full-blown swelling disease during the course of the experiment. It is possible that the treatments applied in this study are not inductive of the disease, or the effects of the treatments were not severe enough to induce swelling disease.

Key words: blood parameters, body weight, faecal egg counts, faecal oocyst counts, total plasma protein.


INTRODUCTION
During the early 1970s, Angora goat farmers reported a condition in their goats, characterised by the sudden onset of severe subcutaneous oedema of the lower body parts. Some goats died of the condition, while others seemed to recuperate spontaneously. According to various internal reports of the Provincial Veterinary Laboratory at Middelburg (Eastern Cape), numerous trials were conducted to investigate the cause of this disease. However, no conclusive evidence was found as far as the cause of the disease was concerned. Therefore, results of these trials were not published. Other experiments conducted during 1977 and 1978 (11 trials in laboratory animals and 45 trials in goats), using different organs inoculated by various routes, failed to give any indication that the disease can be transmitted. A survey of swelling disease was also conducted among Angora goat producers in 1985.

Outbreaks of swelling disease still occur periodically and some losses are still experienced by Angora goat producers. No definite cure is known; various farmers practice different treatments, not all of which are always successful. As a result, delegates at the 60th Annual General Congress of the Mohair Growers’ Association of South Africa unanimously accepted a resolution that purposeful research should be carried out on the cause, treatment and prevention of swelling disease in various areas among all age groups of Angora goats in South Africa. Cases of swelling disease in Angora goats have also been reported in Britain and New Zealand.

Swelling disease in Angora goats is characterised by the sudden onset of severe subcutaneous oedema of the lower body parts, namely ventral neck, chest and abdomen, front and hind legs, especially over the joints and sometimes the lower jaw. The swelling is cold, pitted, painless and variable in intensity in different parts of the body. Fluid flows freely if the skin is cut or punctured. The fluid is clear, copious and non-clotting. There usually are no symptoms such as fever, heart or pulse irregularities or respiratory distress. Other symptoms include swollen lymph nodes, diarrhoea, slight depression and loss of appetite with consequent weight loss. No other clinical signs are apparent.

Various factors have been mentioned that possibly act as predisposing factors for the development of swelling disease:
- Internal parasites (*Ostertagia circumcincta*, *Haemonchus contortus*, other roundworms, coccidiosis and *Paramphistomum*)
- Blood parasites
- Other pathological conditions
- Any stress-inducing condition (weaning, shearing, dipping, castrating, inclement weather, competition for food or change of feed).

There are no indications that swelling disease is correlated with the following: gender, season, climate, weather, topography, veld type, specific plants, licks or external parasites.

Taking into account all factors mentioned above, as well as the lack of any scientific proof for or against any of the possible
Experimental animals and location

Sixteen 8-month-old castrated male Angora goat kids were bought in May 2004 from each of 5 producers whose flocks had a history of swelling disease. These 80 kids were kept in enclosures at Grootfontein Agricultural Development Institute (GADI) near Middelburg (EC) for the duration of the experiment. On arrival at GADI, all kids were vaccinated against pulpy kidney disease, drenched with a broad-spectrum anthelmintic (alendazole, closantel; Prodose Orange Virbac, Halfway House) at the recommended dosage and treated for coccidiosis and dietary protein level in the development of swelling disease in Angora goat kids.

Experimental design

The experiment had a 2 × 2 factorial design. Animals were divided into 8 treatment groups (Table 1) on the basis of 2 goats from each farmer per group. Body weight of goats from the different producers differed considerably at the start of the adaptation period. The goats were divided among the treatment groups in such a way that the average body weight of the different groups was the same at the start of the adaptation period. The average body weight of the various groups at the start of the adaptation period was either 14.6 kg or 14.7 kg. All animals were adapted to their new environment on their respective diets for a period of 7 weeks. Eleven of the goats died during the adaptation period, prior to the commencement of the experiment. The cause of death in most cases was general ill thrift and pneumonia.

Experimental procedures

Experimental treatments. The animals received a combination of the following treatments:

- Diet (high protein vs normal protein level). Goats in the relevant groups received the 2 diets indicated in Table 2 ad libitum.

Table 2: Composition of experimental diets.

<table>
<thead>
<tr>
<th></th>
<th>High protein diet (%)</th>
<th>Normal protein diet (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lucerne</td>
<td>70</td>
<td>65</td>
</tr>
<tr>
<td>Molasses meal</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Maize stover</td>
<td></td>
<td>14.5</td>
</tr>
<tr>
<td>HPC 36</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Di-calcium phosphate</td>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>17.2</td>
<td>11.9</td>
</tr>
<tr>
<td>Energy (TDN)</td>
<td>56.5</td>
<td>56.3</td>
</tr>
<tr>
<td>Ca</td>
<td>1.08</td>
<td>1.11</td>
</tr>
<tr>
<td>P</td>
<td>0.26</td>
<td>0.25</td>
</tr>
</tbody>
</table>

Infection with Ostertagia circumcincta

Third-stage *O. circumcincta* larvae were obtained from the Onderstepoort Veterinary Institute (OVI). Five donor Dorper wethers, kept under conditions that preclude unintentional infection with worms, were drenched daily with levamisole (Rippercol, Janssen Pharmaceuticals, Halfway House) at 15 mg/kg live weight for 2 consecutive days and faecal egg counts were carried out to ensure that the animals were as far as possible free of internal parasites. These 5 donor animals were infected with the larvae obtained from OVI, and faeces were collected for culturing of larvae to be used for the artificial infection of experimental animals. One week prior to the start of the experiment, the animals were treated with a Levamisole anthelmintic (15 mg/kg live weight) to ensure they were free from internal parasites at the start of the experiment. EPG recorded at the start of the experiment (Week 1) showed that all goats still had some roundworm infections. Therefore, goats in Groups 1 and 5 were again treated with a levamisole anthelmintic, but Groups 3 and 7 were not treated, to avoid interfering with the coccidial infections. The goats in the various groups were infected weekly with 10 000 3rd-stage *O. circumcincta* larvae during Weeks 1 to 4.

Infection with coccidia

Faecal samples from Angora goat kids with coccidiosis were used to obtain cultures of coccidial oocysts. The faecal samples were mixed with water and left to stand for 24 hours. The supernatant...
fluid was discarded and the suspension containing the oocysts was again diluted with water. The process was repeated until a relatively clean solution, containing the oocysts, was obtained. The oocysts were left at room temperature until they sporulated.

One week prior to the start of the experiment, faecal coccidial oocyst counts were high in goats of all groups. The animals were subsequently treated for coccidiosis (Diclazuril – 2 m/kg per 5 kg body weight) at the recommended dosage. However, at the start of the experiment, the animals still had high faecal coccidial oocyst counts.

Groups 1, 2, 5 and 6 were again treated for coccidiosis with sulfa-methazine. Owing to still high OPG, goats in Groups 1 and 5 were treated with Diclazuril after Week 1, but Groups 2 and 6 were not, to avoid interfering with the O. circumcincta infections. The goats in the various groups were dosed twice with approximately 20 000 sporulated coccidial oocysts after data collection in Week 1 and Week 2.

**Data collection**

All kids were weighed on arrival at GADI and weekly thereafter. The goats were also monitored daily for any clinical symptoms. Blood samples were collected every week from each goat into heparinised vacutainer tubes. The samples were centrifuged at 2000 G immediately after collection for determination of total plasma protein concentration (TPP). A 2nd blood sample was collected for haematology in Weeks 1, 5, 9, 13 and 16 of the experiment. This whole blood sample from each goat was sent to Golden Vetlab for analyses of the following:

- Red blood cell count
- White blood cell count
- Packed cell volume
- Haemoglobin concentration
- Mean cell volume
- Mean cell haemoglobin concentration

Individual faecal samples were collected weekly for determination of eggs per gram of faeces (EPG) and oocysts per gram of faeces (OPG) at the Middelburg PVL, employing the McMaster technique. No samples were collected during Week 7, as the technician responsible for faecal egg counts was not available that week. Larval cultures were done during Week 5 for identification of roundworm species present. The goats were shorn on 20 August 2004 (Week 10 of the study).

**Statistical analysis**

Data on body weights, TPP, EPG, OPG and blood parameters were analysed using the GLM-procedure of SAS, fitting experimental group as a fixed effect in order to evaluate possible differences between treatment groups. The effect of dietary protein level on body weight and plasma protein levels of the animals over the experimental period were evaluated by fitting diet as a fixed effect in a linear model. Differences in EPG between goats receiving O. circumcincta larvae and goats which did not receive any artificial O. circumcincta infection, were tested for significance by including infection status in the model. The same was done for OPG.

Pooling the data on body weight and TPP collected over the 16-week experimental period, the effects of EPG and OPG on body weight and TPP were determined. Furthermore, recorded data of goats that developed signs of oedema were compared with those of the goats that did not develop any oedema, by fitting oedema status (moderate, little or none) as a fixed effect.

**RESULTS**

There were no significant differences in body weight among the different treatment groups at any stage during the experimental period. Body weights of goats from the different breeders differed considerably at the start of the adaptation period, when the goats from each breeder were divided among all the treatment groups in such a way that the average body weight of the different groups was the same at the start of the adaptation period. For the rest of the experimental period, differences within groups were much larger than differences among groups, therefore differences in body weights that were recorded over the experimental period were not statistically significant.

Total plasma protein concentration (TPP) of the goats over the first 6 weeks of the experiment, did not differ significantly among the treatment groups. From Week 7 onwards, animals in Group 8 had significantly ($P < 0.05$) lower TPP than those in the other groups, while TPP of animals in Groups 5 and 6, were lower than TPP of Groups 1, 2, 3 and 4 from Weeks 8 and 9 onwards. During Weeks 15 and 16, TPP of animals in the normal protein diet groups (Groups 5, 6, 7, 8), was lower than that of animals in the high protein diet groups (Groups 1, 2, 3, 4).

Group mean faecal egg counts and coccidial oocyst counts over the experimental period also showed no specific trend among the different groups. Generally, differences among animals within the specific groups were larger than the differences among the groups themselves.

Goats artificially infected with O. circumcincta larvae had higher faecal egg counts during Weeks 6 to 10, and Weeks 13 and 15 ($P < 0.05$) than goats that did not receive any artificial O. circumcincta infection (Fig. 1). Larval cultures done during Week 5 indicated that the majority of roundworms were indeed O. circumcincta. Goats artificially infected with coccidial oocysts had higher faecal oocyst counts only during Weeks 3 and 12 ($P < 0.05$), compared to goats that received no artificial coccidial infection (Fig. 2). As is evident from the OPG recorded during Weeks 1 and 2, the goats in all groups had a natural infection of coccidial oocysts, therefore there where no differences in OPG between those groups that were artificially infected with coccidial oocysts, and those that were not subjected to artificial infection.

The effects of dietary protein level on body weight and TPP of goats over the experimental period are illustrated in Figs 3 and 4, respectively. The positive effect of a higher dietary protein level was reflected in the higher body weights of the goats that received the high protein diet. Goats on the high protein diet had a larger decrease in body weight after shearing, but were less affected by the
adverse weather conditions between Week 11 and Week 12, when some goats developed oedema. They also recovered more quickly (Week 13) than goats on the normal protein diet. From Week 8 onwards, TPP of the goats receiving the high protein diet was higher than that of the goats that received the normal protein diet.

On Monday 6 September 2004 (Week 12), 19 of the goats developed some subcutaneous oedema. The Saturday (4 September 2004) was rather hot (30 °C), followed by very cold, rainy conditions (11 °C) on Sunday (5 September 2004). Twelve of the goats developed what can be described as little oedema, while 7 developed moderate oedema. Goats that developed oedema were distributed over all groups, with the exception of Group 2. After a week, all signs of oedema disappeared in all the affected goats.

The number of goats in each group that developed oedema is summarised in Table 3. Results indicate that the number of goats that developed oedema was fairly evenly distributed among the various treatment groups. As far as the specific treatments are concerned, more goats on the normal protein diet developed moderate oedema, compared with the goats on the high protein diet. Furthermore, more goats that received coccidial infection also developed moderate oedema compared with the ones that did not receive artificial infection.

Mean body weight and TPP of the animals that developed moderate, little or no oedema are presented in Figs 5 and 6, respectively. It is obvious that body weights of goats that developed moderate oedema were lower throughout the experimental period than body weights of goats that developed little or no oedema. It is also evident that TPP of goats that developed moderate oedema was lower from Week 5 ($P < 0.05$), than TPP of goats that developed little oedema and those that did not develop any oedema.

There were no significant differences at any stage throughout the experimental period in EPG between goats that developed moderate oedema, goats that developed little oedema and those that did not develop any oedema.

There were also no significant differences at any stage throughout the experimental period in the red blood cell count, haemoglobin concentration, haematocrit, mean cell volume, mean cell haemoglobin concentration or white blood cell count (Table 4) between goats that developed moderate oedema, goats that developed little oedema and those that did not develop any oedema.

**DISCUSSION**

The main observation in this study was that no goat developed full-blown swelling disease during the course of the experiment. It is possible that the treatments applied in this study in an attempt to induce swelling disease, are not inductive of swelling disease, or the effect of the applied infections was not severe enough to induce swelling disease, and that higher infection levels of *O. circumcincta* or coccidial oocysts may induce the disease.

Similar results were obtained in 3 experiments conducted at GADI during 1980 to 1984, in which 64 goats (4-tooth and older – Trial 1), 54 goats (majority 12 months old, some were 4- to 6-tooth – Trial 2) and 20 goats (4- to 6-tooth – Trial 3) respectively, were infected with *O. circumcincta* larvae at varying doses$^{13}$. No goats developed swelling symptoms in Trial 1, while 2 goats from Trial 2 and 1 from Trial 3 developed distinct oedema. A further 10 goats from Trial 2 developed little oedema.
only on the chest area. It was concluded that it seemed as if *O. circumcincta* could be an underlying cause for the development of swelling disease. However, some or other trigger mechanism (any stress-inducing condition) is necessary for the goat to develop full-blown swelling disease.13

According to the available literature, full helminth counts of affected goats in other studies showed a variable picture of low to moderate parasite burdens1,4,11,13. Most commonly *Ostertagia circumcincta* was found, alone or with *Trichostrongylus colubriformis* and/or *Nematodirus spathiger*. In some cases, no parasites were found even in undosed animals. Other worm species were also found, but in general no clear pattern emerged. Faecal egg counts were generally low (<1000 eggs/g), but occasionally very high (>10 000 eggs/g).11 Similar results were obtained in 2003 and 2004 at the Middelburg PVL on goats with severe swelling disease.8 In the latter cases, coccidial egg counts varied between 9000 and 35 3000 oocysts/gram, with most recorded OPG higher than 20 000 oocysts/gram.

In the present study, more goats that received artificial coccidial oocysts developed moderate oedema compared with those that were not artificially infected. As there were no significant differences in OPG at any stage of the experiment between goats developing moderate, little or no oedema, as well as the fact that goats artificially infected with coccidial oocysts had higher faecal oocyst counts only during Weeks 3 and 12, compared to goats that received no artificial coccidial infection, no overall conclusion with regard to the effect of coccidial infection on swelling disease can be drawn. However, when comparing infection status of only those animals that developed moderate oedema, it was found that those goats that received artificial coccidial infection had higher OPG from Week 2 to Week 10. The higher coccidial burden in the weeks preceding the development of oedema could have contributed to these goats developing oedema. This should, however, be investigated further.

In the present study, the goats that did develop moderate oedema were smaller than those that developed little or no oedema. This is in accordance with the results of other work13, where the goats that developed swelling disease were also the smallest in their respective groups. Stress caused during competition for food could probably have contributed to these goats developing oedema symptoms.

Furthermore, goats that did develop moderate oedema had lower TPP levels throughout the experimental period. According to other available reports, the plasma of oedematous goats have a lower total protein concentration, lower plasma albumin levels and a lower albumin:globulin ratio than that of normal goats1,4,11,13. Similar results were obtained from swelling disease cases investigated during 2003 at the Middelburg PVL.8 In another study where goats were infected with various levels of *O. circumcincta* it was found that all the infected goats had reduced plasma protein levels13. However, in the present study, neither EPG nor OPG had any significant effect on body weight or TPP.
Table 4: Blood parameters (±SE) of experimental animals.

<table>
<thead>
<tr>
<th></th>
<th>Week 1</th>
<th>Week 5</th>
<th>Week 9</th>
<th>Week 12</th>
<th>Week 13</th>
<th>Week 16</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Red blood cell count</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Moderate (×1012/l)</td>
<td>12.0 ± 0.3</td>
<td>12.8 ± 0.3</td>
<td>12.6 ± 0.3</td>
<td>12.4 ± 0.1</td>
<td>12.7 ± 0.4</td>
<td>12.3 ± 0.4</td>
</tr>
<tr>
<td>Little</td>
<td>12.3 ± 0.2</td>
<td>12.8 ± 0.2</td>
<td>12.8 ± 0.2</td>
<td>12.8 ± 0.1</td>
<td>12.7 ± 0.3</td>
<td>12.8 ± 0.2</td>
</tr>
<tr>
<td>No</td>
<td>12.3 ± 0.1</td>
<td>13.0 ± 0.1</td>
<td>13.0 ± 0.1</td>
<td>–</td>
<td>12.8 ± 0.1</td>
<td>13.0 ± 0.1</td>
</tr>
<tr>
<td><strong>Haemoglobin concentration (g/dl)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>8.7 ± 0.4</td>
<td>10.2 ± 0.4</td>
<td>10.0 ± 0.4</td>
<td>9.7 ± 0.2</td>
<td>9.9 ± 0.4</td>
<td>9.1 ± 0.5</td>
</tr>
<tr>
<td>Little</td>
<td>9.1 ± 0.2</td>
<td>9.8 ± 0.2</td>
<td>10.0 ± 0.2</td>
<td>10.0 ± 0.1</td>
<td>9.5 ± 0.3</td>
<td>9.8 ± 0.3</td>
</tr>
<tr>
<td>No</td>
<td>9.1 ± 0.1</td>
<td>10.0 ± 0.1</td>
<td>10.2 ± 0.1</td>
<td>–</td>
<td>9.7 ± 0.1</td>
<td>9.7 ± 0.2</td>
</tr>
<tr>
<td><strong>Haematocrit (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>28.8 ± 1.2</td>
<td>31.6 ± 1.3</td>
<td>30.6 ± 1.3</td>
<td>30.0 ± 0.6</td>
<td>28.6 ± 1.4</td>
<td>29.3 ± 1.5</td>
</tr>
<tr>
<td>Little</td>
<td>30.7 ± 0.8</td>
<td>32.1 ± 0.9</td>
<td>31.6 ± 0.8</td>
<td>31.3 ± 0.4</td>
<td>30.8 ± 1.0</td>
<td>31.6 ± 1.0</td>
</tr>
<tr>
<td>No</td>
<td>30.9 ± 0.4</td>
<td>32.9 ± 0.4</td>
<td>32.6 ± 0.4</td>
<td>–</td>
<td>31.7 ± 0.4</td>
<td>32.2 ± 0.5</td>
</tr>
<tr>
<td><strong>Mean cell volume (fl)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>24.0 ± 0.5</td>
<td>24.6 ± 0.6</td>
<td>24.3 ± 0.5</td>
<td>24.4 ± 0.2</td>
<td>24.3 ± 0.5</td>
<td>23.8 ± 0.5</td>
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<tr>
<td>Little</td>
<td>24.8 ± 0.4</td>
<td>25.2 ± 0.4</td>
<td>24.8 ± 0.3</td>
<td>24.3 ± 0.1</td>
<td>24.5 ± 0.4</td>
<td>24.6 ± 0.3</td>
</tr>
<tr>
<td>No</td>
<td>25.12 ± 0.2</td>
<td>25.3 ± 0.2</td>
<td>25.1 ± 0.1</td>
<td>–</td>
<td>24.8 ± 0.2</td>
<td>24.7 ± 0.2</td>
</tr>
<tr>
<td><strong>Mean cell haemoglobin concentration (g/dl)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>30.2 ± 1.0</td>
<td>32.4 ± 1.0</td>
<td>32.6 ± 1.2</td>
<td>32.2 ± 0.5</td>
<td>31.7 ± 1.2</td>
<td>31.0 ± 1.2</td>
</tr>
<tr>
<td>Little</td>
<td>29.7 ± 0.7</td>
<td>30.8 ± 0.6</td>
<td>31.8 ± 0.8</td>
<td>31.9 ± 0.3</td>
<td>30.6 ± 0.8</td>
<td>31.0 ± 0.8</td>
</tr>
<tr>
<td>No</td>
<td>29.7 ± 0.3</td>
<td>30.7 ± 0.3</td>
<td>31.4 ± 0.4</td>
<td>–</td>
<td>30.7 ± 0.3</td>
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</tr>
<tr>
<td><strong>White blood cell count (×109/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>14.3 ± 1.4</td>
<td>12.4 ± 1.7</td>
<td>13.8 ± 1.7</td>
<td>15.7 ± 0.7</td>
<td>14.0 ± 2.2</td>
<td>13.8 ± 2.0</td>
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<tr>
<td>Little</td>
<td>11.2 ± 0.9</td>
<td>12.6 ± 1.1</td>
<td>13.2 ± 1.1</td>
<td>16.5 ± 0.5</td>
<td>17.1 ± 1.6</td>
<td>14.2 ± 1.3</td>
</tr>
<tr>
<td>No</td>
<td>12.9 ± 0.5</td>
<td>13.1 ± 0.5</td>
<td>13.8 ± 0.5</td>
<td>–</td>
<td>15.7 ± 0.7</td>
<td>14.3 ± 0.6</td>
</tr>
</tbody>
</table>

Blood parameters recorded for goats that developed moderate oedema during the present study, are in accordance with those for red blood cell count, haemocrit, mean cell volume and mean cell haemoglobin concentration reported elsewhere. However, in swelling disease cases investigated in 2003 and 2004 at the Middelburg PVL, it was found that haemoglobin concentration was lower in goats affected with swelling disease, while white blood cell counts of affected goats were elevated. Segmented neutrophils, lymphocytes and monocytes are the types that were significantly elevated in the latter cases.

The susceptibility of Angora goats to cold stress and their subsequent physiological reactions have been well documented. Therefore the stress due to the adverse weather conditions during the experimental period most probably precipitated the disease in the goats already predisposed as a result of the other factors.

**CONCLUSIONS**

From all the information available on swelling disease in Angora goats, it is obvious that it is a complex condition, of which little is known regarding the mechanisms involved in the initiation and course of the disease. Although *O. circumcincta* infection has been listed as the probable cause, predisposing factors and effective treatment of swelling disease in Angora goats, Grootfontein ADI; Animal Production Progress report.

The possibility that there are different clinical forms of the disease should also be borne in mind, as reported cases differed considerably in the extent of oedema, as well as with regard to other associated parameters and the duration and outcome of the disease.

**ACKNOWLEDGEMENTS**

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