

# The effect of a natural maedi-visna virus infection on the productivity of South African sheep

B. DUNGU<sup>1</sup>, J. VORSTER<sup>1</sup>, G.F. BATH<sup>2</sup> and D.W. VERWOERD<sup>1</sup>

#### **ABSTRACT**

DUNGU, B. VORSTER, J., BATH, G.F. & VERWOERD, D.W. 2000. The effect of a natural maedivisna virus infection on the productivity of South African sheep. *Onderstepoort Journal of Veterinary Research*, 67:87–96

A cohort study was conducted in order to measure the effect of the chronic indurative lymphocytic mastitis caused by the South African strain of maedi visna virus (MVV) on the pre-weaning growth of lambs born either of naturally infected or uninfected ewes kept under similar conditions. Fifty naturally infected ewes as well as another 40 from a maedi-visna-free source to be used as control animals, were purchased and kept in separate flocks which were managed in a similar way. All the ewes were of the same breed and 3–4 years old. During the adaptation period, and through the mating, pregnancy and lactation periods they were periodically monitored for the presence of MVV serum antibodies. The lambs were weighed at birth and thereafter every 2 weeks until the age of 90 days, when they were weaned. The ewes were then slaughtered, and their udders examined histologically and the number of lymphocytic follicles were counted and assessed. Although the calculated values indicated a correlation between the number of follicles in the udder and the reduction in the growth rate of the lambs, this was not statistically significant. Similarly, despite higher counts of lymphoid follicles in the udders of sero-positive ewes as compared to those that were sero-negative and the lower ewe productivity indexes in infected ewes, no statistically significant differences were found in the indexes of ewes in different follicle categories.

Keywords: Dorper sheep, lamb growth, maedi-visna virus

### INTRODUCTION

Maedi-visna (MV) is a composite Icelandic name for two clinical entities in sheep caused by the same slow virus, a non-oncogenic ovine lentivirus (Dawson 1980; Gudnadottir & Palsson 1967; Palsson 1990). It is also known in the USA as ovine progressive pneumonia. Maedi (meaning dyspnoea) is characterized by chronic progressive pneumonia, and visna (meaning wasting), by meningoencephalitis of adult sheep which leads to weakness and progressive paresis especially of the hind limbs.

Maedi-visna virus (MVV) has a worldwide distribution, with the exception of Australia and New Zealand (Houwers 1990).

Manifestations other than maedi and visna, usually described in ovine lentivirus infections, include a chronic arthritis in mature sheep and a chronic nonfebrile mastitis (Cross, Smith & Moorhead 1975; Van der Molen, Vecht & Houwers 1985; Palsson 1990).

Chronic indurative mastitis with massive lymphoid proliferation in ewes suffering from MVV infection has been described by Dawson (1987) and the association of ovine lentivirus with the lymphocytic mastitis has been demonstrated after natural infection (Olivier, Gorham, Parish, Hadlow & Narayan 1981) as well as in an experimental study (Van der Molen & Houwers 1988). The MVV-associated mastitis may result in a reduction in milk production as a consequence of the compression of the lactiferous sinuses

Accepted for publication 17 January 2000—Editor

Onderstepoort Veterinary Institute, Private Bag X05, Onderstepoort, 0110 South Africa

Faculty of Veterinary Sciences, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa

and loss of glandular tissue (Dawson 1987; Olivier *et al.* 1981; Van der Molen *et al.* 1985; Van der Molen & Houwers 1988).

Houwers *et al.* (1988), studying a flock severely affected with MVV, found lymphocytic lesions in 53% of the mammary glands of the ewes but distinct lung lesions in only 10% of them. These results, and later work by Pekelder *et al.* 1994, suggested that the lymphocytic mastitis and resulting reduction in milk production develops before the lung lesions and is the most important feature of maedi-visna.

Studies on the effect of clinical and subclinical MVV infection on sheep productivity, however, have shown that assessing the damage caused by the infection is a complicated task. Factors such as husbandry, management and normal practices in the local sheep industry have to be taken into account (Campbell et al. 1994; Dohoo et al. 1987; Houwers 1990; Snowder, Gates, Glimp & Gorham 1990; Williams-Fulton & Simard 1989). Although in some reports no statistically significant differences could be found in lamb weights between infected and non infected animals (Dohoo et al. 1987, Snowder et al. 1990), the reduction in milking potential of ewes due to the indurative lymphocytic mastitis has been shown to result in poor pre-weaning growth (Pekelder, Veenink, Akkermans, van Eldik, Elving & Houwers 1994; Smith 1992). There are also indications of an increased mortality rate of the lambs (Anderson, Bulgin, Adams & Duelke 1985; Dawson, Biront & Houwers 1982; Dohoo et al. 1987; Houwers 1990; Snowder et al. 1989). The gross production of an infected flock can thus be affected.

A combination of factors such as the virulence of the virus strain involved and breed susceptibility seem to play a role in the evolution of the infection (Houwers 1990).

Although the first pathological description of a condition fitting the picture of maedi originated from South Africa (Mitchell 1915), followed later by a description by De Kock (1929) of a chronic progressive pneumonia which he called Graaff-Reinet disease, no MVV infection was reported in South Africa until a lentivirus was isolated in 1986 from lungs of sheep suffering from jaagsiekte (Payne, York, De Villiers, Verwoerd, Querat, Barban, Sauze & Vigne 1986), a contagious lung tumour also known as ovine pulmonary adenomatosis. Subsequently, a limited survey indicated a wide distribution of a virus with low pathogenicity (Payne *et al.* 1986).

A significant genetic divergence was demonstrated between the virulent MVV isolated in Iceland, the caprine arthritis-encephalitis virus (CAEV), and the South African ovine MVV (SA-OMV) viruses by means of endonuclease restriction analysis, nucleic acid hybridization techniques and nucleotide

sequence studies (Querat, Barban, Sauze, Vigne, Payne, York, De Villiers & Verwoerd 1987). This study demonstrated that the SA-OMVV was more closely related to MVV than to CAEV, but was significantly distinct to be regarded as a separate subgroup. A phylogenetic history, based on the divergence of nucleotide sequences, suggested that the two ovine lentiviruses have been evolving independently for at least 42 years (Querat *et al.* 1987).

Using the expressed fusion protein p25 as antigen in a Western blot test and in an ELISA test (York, Dungu & Du Plessis 1993), sera from a closed flock of 2400 sheep with a history of lung problems (the Goedemoed prison flock, Free State) were tested. sA seroprevalance for MVV antibodies of 80% was established (York & Dungu, unpublished data 1992). Since then, MVV infection has been confirmed clinically and histologically (Vorster, Dungu, Marais, York, Williams & Boshoff 1996), as well as with commercially available test kits. The results of this pilot study lead to the design of this cohort study on the effects of natural MVV infection on sheep productivity in South Africa.

The aim of the present study was to determine whether the subclinical mastitis, characterized by proliferation of lymphoid follicles in udder tissue due to the infection with SA-OMVV, has a detrimental effect on the growth of lambs born of infected ewes.

# **MATERIALS AND METHODS**

### Sheep

Fifty Dorper ewes aged between 3 and 4 years (six or early eight tooth) were selected from the heavily infected closed flock of Goedemoed prison in the south east Free State, on basis of their consistent sero-positive status for MVV antibodies as revealed by two MVV ELISA assays: the OVI ELISA (Boshoff, Dungu, Williams, Vorster, Conradie, Verwoerd & York 1997; York, Dungu & Du Plessis 1993), and a commercially available test, from Institut Pourquier (KIT ELISA Visna Maedi, Institut Pourquier, Montpellier, France).

The control group consisted of 40 Dorper ewes of the same age range as the previous group. They were selected from a flock with no history of lung problems, and confirmed sero-negative for MVV antibodies by the same two serological tests, conducted on the flock basis as well as on individual basis.

The two sero-groups were brought to the Onderstepoort Veterinary Institute (OVI) and kept in two widely separated groups of pens. Two pens were used for each of the two groups, each pen containing 20 ewes in the control group, and 25 ewes in the sero-positive group. Each ewe was identified with a number by ear-tagging. They were allowed an adaptation period of 2 months, during which they were vaccinated according to the standard schedule in the Onderstepoort area and monitored for internal parasites and any other condition that could affect the study. The mass and the condition score of all ewes were recorded on day 0 of the study and every second week thereafter during the adaptation period. No common facilities were shared by the two serogroups. Each sero-group was fed *ad lib*. with the same type of feed, and attended to by different shepherds throughout the study.

Four Dorper rams of the same genetic background, i.e. the progeny of one sire, were purchased from a flock with no history of lung problems and serologically screened and found negative for MVV antibodies. Two weeks prior to joining, the rams were brought into close proximity of the ewes, in order to generate biostimulation, by the so-called "ram effect" (Chemineau & Cagnie 1992). Two rams were used for each sero-group. During the joining period, they were left with the ewes in one pen for a period of 35 days, and then moved to the other pen of the same sero-group for the same length of time.

Seventy days after joining, a pregnancy diagnosis using trans-abdominal ultra sound (Toshiba® Sonarlayer SAL-32A) was performed on all ewes. All ewes were monitored monthly during the first 3 months of pregnancy for faecal egg count, mass, condition score and serology. A routine clinical examination to assess the general condition of each sheep was carried out weekly, on different days for each groups.

Lambing was closely monitored and a record of the identification of the ewe that had lambed in each 24 h period made. All newborn lambs were weighed and eartagged within 2 days of birth. To monitor the growth of the lambs, their masses were recorded every 2 weeks, using a scale of 0,1 kg accuracy. All lambs or ewes, which developed clinical conditions other than those that could be associated with MVV infection were attended to and, if judged necessary, eliminated from the study.

The lambs were weaned at the age of approximately 90 days, and their masses were recorded. In addition, milk samples were collected from the ewes, and their masses and condition score recorded.

Approximately 3 weeks after their lambs had been weaned the ewes were slaughtered, and tissue specimens from their udders were collected in 10% buffered formalin for histological examination.

Ewes that did not conceive, as well as animals that died during the experimental period, were slaughtered and necropsied. Organ specimens were collected in 10% buffered formalin for histological examination.

# Histological examination

Tissue blocks of mammary tissue for sectioning for histological examination using routine procedures were taken from two sites of each mammary gland of a ewe: one consisted of the tissue from the centre of the gland and the other of tissue adjoining the lactiferous sinus. Sections were stained by the haematoxylin and eosin method (Watt, MacIntyre, Collie, Sargan & McConnell 1992). The extent of the lesions was measured by counting the total number of lymphoid follicles and/or lymphoid aggregates in the four sections from each udder, according to a modification of the method described by Pekelder et al. (1994). Based on these total follicle counts, the udders were divided into three classes: class 1 (0-5 follicles); class 2 (6-15 follicles); and class 3 (more than 15 follicles).

# Microbiological and cytological examinations of milk samples

Milk samples were collected from each ewe in early, mid and late lactation for bacteriological and cytological examinations. Somatic cell counts (SCC) and total bacterial counts were carried out on each milk sample according to the procedure described by the International Dairy Foundation (IDF 1987).

# Serological examination for antibodies to MVV

Blood samples were taken from the ewes at intervals of 4 weeks, except for the last month of pregnancy, and the 90 days lactation period, when no blood samples were taken. At weaning and at slaughtering, blood samples were collected from both lambs and ewes. Vacuum tubes and a new needle for each animal were used throughout the experiment. Serum was collected and tested for antibodies to MVV with the OVI ELISA (Boshoff *et al.* 1997).

# **Analysis of data**

The masses of all experimental ewes and their lambs were recorded and stored on a database for further analysis. For ewes, the mating and weaning masses were used for calculations, although they were weighed several times. The lambs' birth masses and weaning masses were used for calculation of rates described below.

The rates calculated were:

- 1. Conception rate (number of ewes lambed divided by the number of ewes mated).
- 2. Lambing percentage (number of lambs born divided by the number of ewes mated) x 100.
- 3. Fecundity (number of lambs born divided by the number of ewes lambed).

- 4. Perinatal lamb mortality (number of lambs died divided by the number of lambs born) x 100.
- The average daily gain (ADG) of lambs for the pre-weaning period. The pre-weaning ADG was determined by dividing the mass gained from birth to weaning by the number of days for the same period.
- The age and sex corrected weaning mass (ASWM). The corrected weaning mass for 100 days was first determined:

The birth mass plus hundred times the ADG [Birth mass + (ADG x 100)]

The least square mean was used to determine the correction factors for the ram and ewe lambs. These correction factors are then subtracted from the age corrected weaning mass to give the ASWM (Fogarty 1995).

- 7. The ewe productivity index. For each ewe the ASWM of all her weaned lambs are added to get the total weaning mass per ewe (TWM). The ewe productivity index is then calculated as follows:
  - (TWM/average TWM for all ewes) x 100
- The weaning percentage (number of lambs weaned divided by the number of ewes mated) x 100.

A computer programme, obtained from the Grootfontein Agriculture Development Institute, Eastern Cape Province, was used to calculate the lamb age and sex corrected weaning mass, as well as the ewe productivity index.

# Statistical analysis

Differences between the two groups, i.e. sero-positive ewes and their lambs versus sero-negative ewes and their lambs, and the association between different variables and rates were statistically studied by analysis of covariance and *f*-probability, using Gynstat 5, Release 3.2 (Steel & Torrie 1980).

Using regression analysis, the lambs' corrected weaning masses and pre-weaning average daily gains were compared between the infected and the control groups. Mean variates were compared in order to determine if the differences found in the means of these values were statistically significant between the groups.

Regression analysis was also used to determine the relationship between the number of lymphoid follicles in the dam's udder and the lamb's pre-weaning ADG on the one hand, and the ewe's productivity indexes (EPI) and the number of follicles on the other hand. Mean variates were compared in order to determine if the differences found in the means of these values were statistically significant between the follicle categories.

#### **RESULTS**

# Serological monitoring

All ewes maintained their serological status throughout the experiment, i.e. all of the control group remained sero-negative and all of the infected animals remained sero-positive (data not shown).

# Mating and lambing periods

After the adaptation period, 38 sero-negative and 47 sero-positive ewes were mated. The mating and lambing results are given in Table 1. The different reproductive rates are summarized in Table 2.

During the adaptation period, five ewes died or were culled for different reasons, three in the sero-positive

TABLE 1 Mating and lambing results

	Sero- negative ewes	Sero- positive ewes
Ewes at the beginning of the study Ewes mated Average mating mass of ewes Ewes diagnosed pregnant Ewes lambed Lambs born alive Stillborn Single lambs Sets of twins Average birth mass of lambs	40 38 53,83 kg 36 40 1 30 5 4,67 kg	50 47 52,38 kg 43 43 48 1 36 6 4,52 kg

TABLE 2 Reproductive parameters

Rates	Sero- negative ewes	Sero- positive ewes
Conception rate	94,73 %	91,49 %
Lambing percentage	105,26 %	102,12 %
Twinning rate	23,80 %	25,00 %
Fecundity	1,111	1,116

TABLE 3 Summary of serological results at weaning

Group	Total number	Positive	Suspect	Negative
Sero-positive ewes Sero-negative	n = 36	35	1	0
ewes Lambs from sero-positive	n = 29	0	0	29
ewes Lambs from sero-negative	n = 38	0	1	37
ewes	n = 33	0	0	33

group and two in the sero-negative group. Although one of the three sero-positive ewes showed lesions of MV on postmortem evaluation, in none of the five ewes could the cause of death or the reason for culling be associated with MVV infection.

# Lactation and weaning periods

The serological results of lambs and ewes at weaning are given in Table 3, and the productivity data and rates are given in Tables 4 and 5, respectively.

# Monitoring of ewes' milk and correlation with lamb growth

The results obtained after the evaluation of milk samples collected three times during the lactation period from each lactating ewe are summarized in Table 6. Samples from which bacterial growth was obtained could not be associated with high SCC as the bacteria identified were considered to be contaminants. The somatic cell count was therefore interpreted without discriminating between a mechanical or a bacterial cause.

- One ewe in the positive group showed high SCC in both halves for all three examinations.
- One ewe from the negative group showed high SCC in one or both halves in all three examinations.
- Six ewes, three sero-positives and three seronegatives, showed high SCC in one or both halves in two of the examinations.

TABLE 4 Summary of productivity data during lactation

	Sero- negative ewes	Sero- positive ewes
Ewes weaning lambs Lamb losses before weaning Lambs weaned	29 5 33 <sup>a</sup>	36 10 38
ASWM (corrected for age and sex)	26,636 kg	25,176 kg
ALWM (corrected for age and birth status)	26,888 kg	25,029 kg

<sup>&</sup>lt;sup>a</sup> One pair of twin lambs was not monitored further due to an unrelated health problem

TABLE 5 Summary of productivity parameters during lactation

Rates	Sero- negative	Sero- positive
Weaning percentage Pre-weaning lamb mortality Pre-weaning average daily gain Average ewe productivity index	91,660 12,5 % 219,636 112,710	88,370 20,8 % 208,132 93,420

# Histological evaluation of udders and correlation with lamb growth

Depending on the number of lymphoid follicles or aggregates found on histological examination of the mammary glands of the ewes, they were classified in three categories, as illustrated in Table 7. The distribution of lambs in both groups according to the

TABLE 6 Summary of milk test results: mastitis cases according to scc with or without bacteria (number of cases/number of ewes tested in the group)

		Sero-negative ewes	Sero-positive ewes
Early	Both half udders	4/33 (12,1 %)	2/28 (7,1%)
lactation	One half udder	3/33 (9,1 %)	1/28 (3,6%)
Mid	Both half udders	3/32 (9,3 %)	3/38 (7,9 %)
lactation	One half udder	5/32 (15,6 %)	7/38 (18,4 %)
End	Both half udders	2/29 (6,9%)	2/37 (5,4%)
lactation	One half udder	5/29 (17,2%)	2/37 (5,4%)

TABLE 7 Distribution of the ewe groups according to udder classes

Udder class	Sero- negative ewes n = 29	Sero- positive ewes n = 37	Total
Class 1 (0-5 follicles) Class 2	28 (96,6%)	18 (48,6%)	46 (69,7%)
(6–15 follicles) Class 3 (more than 15	1 (3,4%)	8(21,6%)	9 (13,6%)
follicles)	0 (0,0%)	11 (29,7%)	11 (16,7%)

TABLE 8 Distribution of lambs according to udder classes of ewes (number of lambs and the percentage in the sero-group)

Ewe udder class	Lambs in sero-negative group	Lambs in sero-positive group
Class 1 (0–5 follicles) Class 2 (6–15 follicles) Class 3 (more than 15 follicles)	31 (93,9%) 2 (6,0%)	18 (47,36%) 8 (21,05%) 12 (31,57%)

TABLE 9 Average daily gain of lambs for each udder class

Ewe udder class	Average daily gain of lambs (g/day)
Class 1 (0–5 follicles) Class 2 (6–15 follicles) Class 3 (more than 15 follicles)	222,2 199,7 198,3

udder classes is shown in Table 8 and their average daily gain in Table 9. The evaluation of the number of acini lost, or the degree of interstitial fibrosis proved extremely difficult. It was therefore difficult to grade and interpret their contribution to reduced milk production.

# Statistical analysis

Using regression analysis, the lambs' corrected weaning masses and pre-weaning average daily gains were compared between the infected and the control groups. Mean variates were compared in order to determine whether the differences found in the means of these values were statistically significant between the groups.

There were no statistically significant (P < 0.01) differences in the average daily gain and the corrected weaning mass between lambs born from infected and non infected ewes.

Using regression analysis, the relationships were calculated for all animals in the study between the number of lymphoid follicles in the dam's udder and the lamb's pre-weaning ADG on the one hand, and the ewe's productivity indexes (EPI) and the number of follicles on the other. Mean variates were compared in order to determine whether the differences found in the means of these values were statistically significant between the follicle categories (see Table 11).

TABLE 10 Statistical analysis: Comparison of mean variates for the lambs' corrected weaning masses and pre-weaning ADG, between the two sero-groups

Corrected weaning mass	Group	Mean of variate	Standard error	
	Sero-negative Sero-positive	26,331 25,532	0,624 0,589	
F-probability (P < 0,01): 0,17780 not significant				
Average daily gain	Group	Mean of variate	Standard error	
Sero-negative 217,18 6,15 Sero-positive 211,59 5,81				
		211,59		

TABLE 11 Statistical analysis: correlation between udder class, ADG and EPI

Ewe udder class	Mean variates EPI	Standard error	Mean variates ADG	Standard error
Class 1	102,80	2,46	216,49	5,19
Class 2	95,09	5,57	208,61	11,34
Class 3	92,29	5,04	209,88	10,46

F-probability (P < 0,01): EPI, 0,118 ADG, 0,751

Neither the ADG of lambs born of ewes in the various lymphoid follicle categories nor the EPI of the ewes in these categories were statistically different.

#### DISCUSSION

Sheep selected in this study were 3–4 years old. The preclinical period of MV is generally 3–4 years (Palsson 1990), although antibodies to MVV are present earlier. This age group, besides being the most productive in a sheep enterprise, is more likely to be in the critical period of developing the disease, and consequently should be well suited for a study of the effect of the disease on sheep productivity.

A mutton breed, the Dorper, was used for the present study, and therefore its outcome would be more useful to mutton farmers than wool producers. The Dorper breed represents the largest number of purely mutton sheep in South Africa. The Goedemoed flock, which consists exclusively of Dorper sheep, provided an ideal opportunity to select naturally infected ewes since 80% of the 3500 sheep in the flock were positive for antibodies to MVV with the ELISA test.

Differences in breed susceptibility and in clinical manifestation of MVV infection have been reported in other parts of the world (Dawson 1987; De Boer 1975). The occurrence of the infection in South African sheep (Payne 1986; Vorster 1996) without any apparent clinical manifestation suggests a mild effect of SA-OMVV on local breeds.

Genetic factors are known to influence ewe productivity. In order to circumvent or minimize that bias, in this study sheep of the same breed were used in both the infected and the control group. The rams used in the study were also selected for their genetic background, they were all the progeny of one sire. The two-month adaptation period of the two groups of ewes, originating from different parts of the country, enabled them to adapt to similar management and husbandry conditions. Data obtained at the end of this period did not show any discrepancies between the ewes in the two groups (Tables 1 and 2).

The lambs were serologically tested for MVV antibodies at weaning (Table 3), and on the day of slaughsster (data not shown). The negative serological results obtained in lambs born from infected ewes are in accordance with previous observations (Dawson et al. 1982; Houwers 1988). Unlike some lentiviral infections, such as HIV in humans, there is no evidence of in utero transmission of MVV from the ewe to the lamb (Houwers 1988). Transmission from ewe to lamb has been shown to occur mainly through the colostrum, presumably by means of infected monocytes (Houwers 1988), but transmission of the virus

under conditions of close contact via the respiratory route, through aerosol remains one of the most important routes of natural infection. Sero-conversion (or the production of detectable antibodies) did not seem to have occurred in the majority of lambs when they were slaughtered at the age of 3 months. One lamb, however, born from a sero-positive ewe, had a very low titre of antibodies to MVV antigen. The time of sero-conversion has been reported to vary from 5 weeks to 11 months or more (Houwers 1988). It is difficult in our study to assess whether lambs born from infected ewes were lactogenically infected or not, as the majority did not show antibodies at the age of 3 months. Considering results obtained in the Goedemoed flock where the sero-prevalence in lambs born from infected ewes was extremely low under the age of 1 year (unpublished data), further study may be required to determine whether the lactogenic mode of transmission is absent under these conditions, or whether MVV in this group of animals induced "serologic latency" (Johnson, Meyer & Zink 1992; Sihnonen 1981) in infected sheep.

Although five of the 47 ewes mated in the sero-positive group (10,6%) did not conceive, as compared to two out of the 38 in the sero-negative group (5,26%), no evidence of a possible association between these conception rates and the presence of MVV infection could be found. None of the five nonpregnant' sero-positive ewes had signs of MVV infection, either clinically or after histological evaluation. In general, the conception rates in both groups were within the norms of the breed and no statistically significant difference was found, which is in accordance with the results obtained in other studies (Dohoo et al. 1987; Pekelder et al. 1994; Snowder et al. 1990). Furthermore, the course of MVV infection does not suggest a mechanism for a decrease in the conception rate, unless due to a loss of condition caused by clinical disease. These authors also did not find statistically significant differences in the lambing percentage, the twinning rate and the fecundity of the ewes. The low pre-weaning lamb growth associated with MVV infection seems to be the consequence of a decrease in the quantity (volume) of milk secreted, resulting from the lymphocytic mastitis, rather than a compromised ability of the dam to give birth to a healthy lamb. In our study, no statistically significant differences were found in the pre-weaning masses of the lambs born to the two sero-groups of ewes.

Any mortality occurring during the experimental period was recorded and necropsied. The high lamb mortality rate observed especially during the first month of life, could principally be attributed to unforeseen circumstances (Table 12). The lambing season unfortunately coincided with a period when there were abnormally heavy rains. As the animals were kept in open pens, it was not possible for the dams to protect their offspring from the inclement weather. Of the 15 cases of lamb mortality recorded, nine (60%) could be associated with bad weather (Table 12). Management-related conditions could be associated with four of the fatalities (26,6%): three cases of sudden death, and one death associated with tail docking. Two lamb mortalities and one pair of twins were removed from the study for reasons related to the dam: one ewe had a liver abscess and two ewes in the sero-negative group suffered from chronic mass loss and produced very little milk. No definite diagnosis could be made on postmortem examination and histopathological examinations of these two ewes and no antibodies to MVV were detected at the time of their slaughter. In summary, no lamb mortality in either of the groups could be attributed to MVV infection.

Productivity parameters, such as EPI, the lamb ADG and the corrected weaning mass are critical for assessing the viability of a sheep production unit. They were used in this study to assess the possible effect of reduced milk production due to MVV infection on ewe productivity, and were measured, in this study, by assessing lamb growth. Although the observed

TABLE 12 Causes of mortality in lambs during lactation

_	Sero-negative group	Sero-positive group
Environmental factors during lambing period:  • Ewe not bonding with lamb during bad weather  • Trauma  • Other	1 1 (broken leg)	1 2 (1 broken leg, 1 ruptured liver) 4 (1 spinal cord abscess, 1 paralysis, 2 heavy rain)
Sudden death	1	2
Problem with the ewe (sick dam) 2 (1 sick dam, 1 dam with no milk)		milk)
Other	,	1 (during tail docking)

means of most rates tended to be higher in the control group, no statistically significant (P < 0.01) differences were found between the two groups for critical variables, such as the ADG and the corrected weaning mass of lambs. It is important to note that in both groups the mean values were quite close and the calculated standard errors relatively high. This could be due to the small sample sizes as, for practical reasons, it was not possible for larger study groups to be used in the experiment. Although not statistically significant, the differences obtained for these values cannot be summarily ignored, and they are to some extent comparable to the results obtained by Pekelder et al. (1994) in whose study the incubation period was short and the majority of infected animals developed an acute form of MV. The resulting loss in ewe productivity could be directly correlated to the severity of the infection. In South Africa, the lesions observed in a pathological survey conducted on slaughtered sheep from the heavily infected Goedemoed flock were typical of MV (Vorster et al. 1996). The clinical disease in this flock is unequivocally less severe than that observed in the Pekelder et al. study, however, despite the high seroprevalence (80 % of the 3 000 sheep). In this mild South African form of MV there is apparently a less severe reduction in milk production although it is felt that the possible effect of MVV infection on lamb production in South Africa should not be entirely discounted. The mild effect observed in this study, when extrapolated to larger flocks and over longer period of time, could be multiplied and results in a significant economic loss for farmers.

In this study, the effect of MVV infection on mammary tissue could not be evaluated by means of the traditional udder health evaluation procedure, in which somatic cell count of the milk is used. The indurative lymphocytic mastitis caused by MVV infection neither produce a "classical" mastitis in which there are high levels of somatic cells in the milk, nor does it affect the milk quality (Anderson *et al.* 1985; Houwers *et al.* 1988). The main consequence of the udder lesions is the reduction of milk production as a consequence of reduced acinar space.

No statistically significant association could be found between the somatic cell counts in the milk and the respective serological status of the ewes in either of the two serological groups (data not shown).

The histological results of this study (Table 7) indicate that 19 out of 37 sero-positive ewes (51,35%) had more than five lymphoid follicles in their udder tissues, compared to only one out of 29 (3,4%) in the sero-negative group. This latter ewe raised twins and had high somatic cell counts in all three milk samples taken. From none of the samples could bacteria be isolated, and it is concluded that the lymphoid proliferation observed in this sero-negative ewe was

caused by factors other than MVV or bacterial infection.

The mastitis caused by MVV infection is of a chronic nature, and is characterized by inter alia lymphoid proliferation in the udder tissue. Studies by Houwers et al. (1988) have shown that the lymphoid proliferation in the udder of infected ewes appears in the early stage of the infection, before the development of lesions elsewhere in the body. The finding that a high proportion of sero-positive ewes in the present study had udders in which nodules of lymphoid cells were very prominent and numerous, as compared to those in sero-negative ewes, could be linked to MVV infection. An attempt to evaluate and grade the extent of interstitial fibrosis and associated loss of acini proved very difficult. It was therefore not possible objectively to assess its contribution to reduced milk production. These results are in accordance with observations in the pathological survey of the Goedemoed flock, where MV lesions in sero-positive sheep were frequently unaccompanied by clinical signs in seropositive sheep (Vorster et al. 1996).

In our study, the difference in the average daily gains of lambs born from ewes in the various udder classes was not statistically significant, possibly due to the small number of animals used. However, the ADG of a lamb (Table 9) was clearly proportional to the number of follicles found in the udder of its dam. It can be deduced that the correlation in this respect found in the European studies in which highly virulent strains of MVV were used might well be present but to a lesser extent in MV caused by the South African strain. The slower rate of drop growth in lambs could result in significant economic losses in the long run in an infected flock, as MVV infection is life-long.

The present study was a first attempt to evaluate the effect of the SA-OMVV infection on sheep productivity in South Africa. It is considered that the correlation between the incidence of lymphoid proliferation in the MVV infected ewes and the slower growth rate of their lambs, albeit statistically insignificant, is an indication that a further study using larger groups of animals need to be conducted. It would then be possible to assess whether the cumulative effect of the life-long MVV infection results in a more significant amount of milk available to the lambs born from infected ewes. Such a study would realize more information on the possible financial implication of the infection for a farming unit. It would then be possible to assess whether it would be better for a farmer to embark on an MV eradication campaign, or to "live" with the infection, and cull infected ewes prematurely.

The course of MVV infection in South African flocks suggests that the local viral strains are mild, that local

sheep breeds have a lower susceptibility to the infection, or that both of these apply.

#### **ACKNOWLEDGEMENTS**

We thank Drs L. Marais and D. Lategan for their assistance in the selection of animals used in the study. We are grateful to the different sections of the Onderstepoort Veterinary Institute for their contribution. This work was supported by the South African Wool and Meat Board and the South African Agricultural Research Council.

#### REFERENCES

- ANDERSON, B.C., BULGIN, M.S., ADAMS, S. & DUELKE, B. 1985. Firm udder in periparturient ewes with lymphocytic accumulations, retrovirus infection, and milk unavailable at the teat. *Journal of the American Veterinary Medicine Associa*tion, 186:391–393.
- BOSHOFF, C.H., DUNGU, B., WILLIAMS, R., VORSTER, J., CONRADIE, J.D., VERWOERD, D.W. & YORK, D.F. 1997. Detection of Maedi-Visna virus antibodies using a single fusion transmembrane-core p25 recombinant protein ELISA and a modified receiver-operating characteristic analysis to determine cut-off values. *Journal of Virological Methods*, 63:47–56.
- CAMPBELL, J.R., MENZIES, P.I., WALTNER-TOEWS, D., WALTON, J.S., BUCKRELL, B.C. & THORSEN, J. 1994. The seroprevalance of maedi-visna in Ontario sheep flocks and its relationship to flock demographics and management practices. *Canadian Veterinary Journal*, 35:39–44.
- CHEMINEAU, P. & CAGNIÉ, Y. 1992. Training manual on artificial insemination in sheep and goats. Rome: Food and Agriculture Organization of the United Nations Publishers.
- CROSS, R.F., SMITH, C.K. & MOORHEAD, P.D. 1975. Vertical transmission of progressive pneumonia of sheep. *American Journal of Veterinary Research*, 36:465–468.
- DAWSON, M. 1980. Maedi visna: a review. Veterinary Records, 106:212–216.
- DAWSON, M. 1987. Pathogenesis of maedi-visna. *Veterinary Records*, 120:451–454.
- DAWSON, M., BIRONT, P. & HOUWERS, D.J. 1982. Comparison of serological tests used in three State veterinary laboratories to identify Maedi-visna virus infection. *Veterinary Record*, 111:432–434.
- DE BOER, G.F. 1975. Zwoegerziekte virus, the causative agent for progressive interstitial pneumonia (maedi) and meningo-leucoencephalitis (visna) in sheep. *Research in Veterinary Science*, 18:15–25.
- DE KOCK, G. 1929. Are lesions of jaagsiekte in sheep of the nature of neoplasm? Fifteenth Annual Report of the Director of Veterinary Services, Union of South Africa: 611–641.
- DOHOO, J.R., HAENY, D.P., STEVENSON, R.G., SAMAGH, B.S. & RHODES, C.S. 1987. The effects of Maedi-visna infection on productivity in ewes. *Preventive Veterinary Medicine*, 4:471–484.
- FOGARTY, N.M. 1995. Genetic parameters for live weight, fat and muscle measurements, wool production and reproduction in sheep: a review. *Animal Breed Abstract*, 63(3):101–143.
- GUDNADOTTIR, M. & PALSSON, P.A. 1967. Transmission of maedi by inoculation of a virus grown in tissue culture from

- maedi-affected lungs. Journal of Infectious Diseases, 117:1-
- HOUWERS, D.J. 1988. Ovine lentivirus infections: aspects of immunology, pathology, epidemiology and control. Ph.D. Thesis. University of Utrecht.
- HOUWERS, D.J. 1990. Economic importance, epidemiology and control of MVV infection, in *Maedi-visna and related diseases*. Dordrecht: Kluwer Academic Publishers.
- HOUWERS, D.J., PEKELDER, J.J., AKKERMANS, J.P.W.M., VAN DER MOLEN, E.J. & SCHREUDER, B.E.C. 1988. Incidence of indurative lymphocytic mastitis in a flock of sheep infected with maedi-visna virus. *Veterinary Record*, 122:435– 437.
- IDF 1987. Bovine mastitis: definition and guidelines for diagnosis. *Bulletin of the International Dairy Federation*, 211
- JOHNSON, L.K., MEYER, A.L. & ZINK, M.C. 1992. Detection of ovine lentivirus in sero-positive sheep by in situ hybridization, PCR, and cocultivation with susceptible cells. Clinical Immunological Immunopathology, 65:254–260.
- MITCHELL, D.T. 1915. Investigation into Jaagsiekte or chronic catarrhal-pneumonia of sheep. *Third and Fourth Reports of the Director of Veterinary Research, Union of South Africa*: 585–614
- OLIVIER, R.E., GORHAM, J.R., PARISH, S.F., HADLOW, W.J. & NARAYAN, O. 1981. Ovine progressive pneumonia: pathologic and virologic studies on the naturally occuring disease. *American Journal of Veterinary Research*, 42:1554–1559.
- PALSSON, P.A. 1990. Maedi-visna. History and clinical description, in Maedi-visna and related diseases. Dordrecht: Kluwer Academic Publishers.
- PAYNE, A.L., YORK, D.F., DE VILLIERS, E.M., VERWOERD, D.W., QUERAT, G., BARBAN, V., SAUZE, N. & VIGNE, R. 1986. Isolation and identification of a South African Lentivirus from Jaagsiekte lungs. *Onderstepoort Journal of Veterinary Research*, 53:55–62.
- PEKELDER, J.J., VEENINK, G.J., AKKERMANS, J.P.W.M., VAN ELDIK, P., ELVING, L. & HOUWERS, D.J. 1994. Ovine lentivirus induced indurative lymphocytic mastitis and its effect on the growth of lambs. *Veterinary Record*,134:348–350.
- QUERAT, G., BARBAN, V., SAUZE, N., VIGNE, R., PAYNE, A., YORK, D.F., DE VILLIERS, E. & VERWOERD, D.W. 1987. Characteristics of a novel lentivirus from South African sheep with pulmonary adenocarcinoma (jaagsiekte). *Virology,* 158: 158–167.
- SIHVONEN, L. 1981. Early immune response in experimental maedi. *Research in Veterinary Science*, 30:217–222.
- SMITH, C. 1992. Ovine Lentivirus: A Real or Imaginary threat? *Journal of the American Veterinary Medicine Association*, 200: 139–143
- SNOWDER, G.D. & GLIMP, H.A. 1989. Effect of ovine progressive pneumonia on ewe milk production. *Journal of Animal Science*, 67:171.
- SNOWDER, G.D., GATES, N.L., GLIMP, H.A., GORHAM, J.R. 1990. Prevalence and effect of subclinical ovine progressive pneumonia virus infection on ewe wool and lamb production. *Journal of the American Veterinary Medicine Association*, 197: 475–479.
- STEEL, R.G.D. & TORRIE, J.H. 1980. Principles and Procedures of statistics, 2<sup>nd</sup> ed. McGraw-Hill Book Company, New York.
- TUSTIN, R.C. 1969. Ovine jaagsiekte. Journal of the South African Veterinary Medicine Association, 1:3–23.
- VAN DER MOLEN, E.J. & HOUWERS, D.J. 1988. Indurative lymphocytic mastitis in sheep after experimental infection with maedi-visna virus. *Veterinary Quarterly*, 9:193–202.

- VAN DER MOLEN, E.J., VECHT, U. & HOUWERS, D.J. 1985. A chronic indurative mastitis in sheep, associated with maedi/ visna virus infection. *Veterinary Quarterly*, 7:112–119.
- VORSTER J.H., DUNGU, B., MARAIS, L.C., YORK, D.F., WILLIAMS, R. & BOSHOFF, C.H. 1996. A perspective of Maedi-visna in South Africa. *Journal of the South African Veterinary Association*, 67(1):2–3.
- WATT, N.J., MacINTYRE, N., COLLIE, D., SARGAN, D. & McCONNELL, I. 1992. Phenotypic analysis of lymphocyte
- populations in the lungs and regional lymphoid tissue of sheep naturally infected with maedi-visna virus. *Clinical and Experimental Immunology*, 90:204–208.
- WILLIAMS-FULTON, N.R. & SIMARD, C.L. 1989. Evaluation of two management procedures for the control of Maedi-visna. Canadian Journal of Veterinary Research, 53:419–423.
- YORK, D., DUNGU, B. & DU PLESSIS, D. 1993. The cloning and expression of a maedi-visna virus cross reacting antigen and its use in a sensitive diagnostic assay to detect MVV affected sheep. *Proceeding: Biotech SA '93, poster 6.*