

## Evaluation of Control Program of Maedi-Visna by Foster Feeding with Cow Colostrum and Other Measures

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**Abstract:** A serological and clinical case control study of Maedi-visna virus (MVV) infection in sheep was carried out at the sheep ranch of Sheno Agricultural Research Center from November 2004 to March 2005 with the objective of characterization the clinical and pathological features of the MVV disease in the center and to evaluate the effectiveness of the control program that practiced in the ranch to produce MVV free flocks. A total of 250, Yearlings (125) and Ewes (125) serum samples were collected and Agar Gel Immunodiffusion (AGID) test was used to identify the presence of antibodies against MVV infection. All sampled sheep were clinically examined whereas few seropositive sheep were examined for gross and histopathological lesions. The overall seroprevalence of MVV infection in the station was 176(70.4%). There was statistically significant difference ( $P < 0.000$ ) between the two feeding types in the mean seroprevalence of the yearling sheep that were fed on their dam colostrum and milk was 79(79%) and that were fed with cow's colostrum and milk 0 (0%). The seroprevalence of MVV infection varied significantly ( $P < 0.05$ ) between Awassi 19(38%), crosses 33(66%) and local Menze 124(82.7%). MVV infection increased with increasing age of sheep from 80(64%) in yearlings with out considering the rearing type to 100% in seventh years old and hence, the seroprevalence was also significantly different ( $P < 0.05$ ) with the variation in age. 21(11.9%) MVV seropositive sheep were found to express the clinical disease. The serological and clinical findings suggested that MVV infection is a major health problem in the center. Based on the above result, artificial rearing by isolation of ovine colostrum deprived lambs offers an effective method of maedi-visna control that could be successfully applied in station.

**Key words:** Age • AGID • Breed • Control • Maedi-Visna virus • Management • Ranch • Sheep

### INTRODUCTION

Sheep production plays a great economic role in the small holder farmers of Ethiopian highlands. Ethiopia has about 25.02 million heads of sheep of which 75% is found in the highlands and the rest in the lowlands of the country [1]. The sheep population in the Amhara regional state is about 8,987,694. This contributes 23% to the total population of the country [1].

Sheep plays a major role in the economy of the country. However, the potential of sheep production has not been efficiently exploited by the Amhara region as well as the country due to different problems. But, in the past decades various attempts were made by the government to realize the benefits from the sub sector.

Hence two sheep breeding and multiplication centers were established with the major objectives of genetic conservation for some attractive traits, cross breeding of phenotypically selected menz breed with superior exotic genotypes and distribution of superior genotypes to the users. However, the appearances of respiratory diseases like ovine progressive pneumonia (Maedi-visna) in the centers have prevented them to meet their objectives and to stop ram distribution and further breeding activities [2-4].

MV is a chronic disease of sheep produced by ovine lentivirus (OVLV), a member of a family of Retroviridae [5,6]. Maedi-visna comprises two Icelandic words describing the clinical presentation of two apparently different syndromes Maedi or labour breathing; a fatal,

progressive pneumonia of mature sheep, Visna or wasting which is a meningoencephalitis, which causes progressive paralysis and death [7,8].

The occurrence of MVV in Ethiopia was first detected in imported breed in 1986 at Agarfa sheep ranch (Bale province). AGID test was conducted and has found 30% seropositivity for MVV infection. Hence, the detection of MV was resulted in the destruction of the flocks with final closure of the ranch [9]. Therefore, the objectives of this study were to assess the effectiveness of the control program (cow colostrum and milk feeding) and complete separating of the lambs from their dam after lambing applied on the sheno sheep ranch and to identify the risk factors associated with disease in the farm/ ranch.

## MATERIALS AND METHODS

**Study Area:** The study was conducted at Sheno agricultural research center, Amhara regional state, North Showa. The study area is a plateau, located about 75km North East of Addis Ababa in the cool high-lands of North-Showa, at an altitude of about 2800m above sea level (a.s.l), latitude 9°36'N and longitude 39°38'E with annual rainfall ranging between 800-1000 mm with bimodal rainfall in pattern consisting of along rainy season that extends from June to September and a short rainy season from February to May/March. Average monthly minimum air temperature is between 5°C and 6°C and frost usually occurs in the months of November and December that reaches an average minimum air temperature of 0.3 to-2.7°C, while the maximum ambient (air) temperature is ranging between 18 to 23.3°C. Mean relative humidity is 68.2%.

**Study Animals:** The study population was included Menz, Awasi-Menz cross and Awassi breeds, Yearling sheep and their dams in the ranch. The study population considered was artificially reared progeny (nipping) and their dams and naturally reared progeny and their dams.

**Study Type and Sampling Procedure:** The study type was case control study design. The study population was stratified in to two strata based on the rearing type of progeny as sheep progeny reared with natural method and artificially reared sheep. The stratification was required because the study population is kept under different management type, which could influence the seropositivity of MVV. Stratification with a variable sampling fraction was used and the proportional allocation was 4:1 for naturally over artificially reared

sheep. Simple random sampling technique was used to select individual study animal in each strata and the desire sample size was calculated according to the formula given by [10] and it was 250.

**Data Collection:** Whole blood samples were collected from selected sheep flocks with each protocol using non-heparinized 10 ml Vacutainer tubes and needles from Jugular vein to produce serum. Serum samples from the study population were tested with AGID (Institut Pourquier, Montpllier-France) according to the protocols recommended by OIE [11] to determine the presence of specific antibodies against maedi-visna virus and to estimate the seroprevalence in the study population. Clinical examination was also made in sheep both in artificially reared and naturally reared progenies and their dams during sera sample collection.

**Data Management and Analysis:** Data collected during sampling and laboratory results were entered in Ms-Excel spread sheet. Descriptive statistics such as mean, median, standard errors, prevalence and confidence intervals of means were used to approximate the seroprevalence for MVV antibodies in the ranch. Risk factors such as breed, age, sex, contact between sheep in the ranch and management were considered and their difference with seropositivity was analyzed by chi square.

## RESULTS

The overall mean seroprevalence MVV infection in both intervention and non-intervention group in the study population was 176 (70.4%) (Table1). There was significant difference ( $P < 0.05$ ) in seroprevalence among three breeds of sheep which was 38% in Awassi and 82.67% in Indigenous Menz sheep (Table 1). The study showed that there was significant difference ( $P < 0.05$ ) between mean seroprevalence among yearling breeds which were artificially reared; Menz-Awassi cross (0%) and those fed on their dam colostrums and milk, Menz (92%) (Table1). The study found that sex did not result significant difference in the mean seroprevalence of MVV (Table 1).

It was found that age could result significantly difference ( $P < 0.05$ ) in the seropositivity of MVV infection (Table 2).

The mean seroprevalence of maedi-visna in treatment and control lambs (control intervention and non-intervention groups) was significantly different ( $P < 0.00$ ). The seroprevalence of MVV increased as type of rearing ranging from 0% to 79 % (Table 3).

Table 1: Mean seroprevalence of maedi-visna virus infection at different risk factors

Types of risk factor	Total tested	Seroprevalence (%)	CI (95%)	$\chi^2$ (P-value)
Menz	150	124(82.7)	77-89	12.4(0.012)
Awassi	50	19(38)	25-51.5	
Cross Breed	50	33(66)	53-79	
Total	250	176(70.4)	65-87	
Menz lamb (yearling)	50	46(92)	85-99	18.74(0.00)
Awassi lamb (yearling)	50	33(66)	53-79	
Cross lamb (yearling)	25	0(0)	0	
Total	125	79(63.2)	55-72	
Male	42	31(73.8)	60-87	6.96(0.137)
Female	208	145(69.7)	63-75	
Total	250	176(70.4)	64-76	

Table 2: Mean seroprevalence of maedi-visna infection at different age groups

Age (years)	Test	No Positive	Seroprevalence (95%CI)	$\chi^2$ (P-value)
1.4-2	125	80	64(55-72)	10.06(0.039)
3	21	17	81.0(64-97)	
4	15	14	93.3(80-106)	
5	45	31	68.9(55-82)	
6	9	8	88.9(68-109)	
7	4	4	100.0(100)	
8	6	3	50(43-57)	
9	25	19	76(59-92)	
Total	250	176	70.4(64-76)	

Table 3: Comparison of ewe colostrum and milk feeding and cow colostrum and milk feeding on the mean seroprevalence of maedi-visna in the yearling

Feeding type	Total Tested	Positive	seroprevalence(95%CI)	$\chi^2$ (P-value)
ewe colostrum and milk until weaned	100	79	79 (71-86.9)	72.16(0.032)
Cow colostrum and milk until weaned	25	0	0(0)	
Total	125	79	63.2(54.75-72.0)	

Table 4: Relation between the clinical disease and maedi-visna seropositivity in sheep

Serological result	Apparently healthy	Clinical disease (%)	Total
Positive	155	21(11.9)	176
Negative	66	8(10.8)	74
Total	221	29(11.6)	250

Table 5: The proportion of clinically diseased sheep in relation to breeds and clinical syndrome

Breed type	No clinical cases	No positive (%)
Menz	18	15(83.3)
Awassi	5	2(40)
Awassi-Menz Crossed	6	4(66.7)
Total	29	21(72.4)
Type of clinical syndromes	No clinically diseased	No seropositive(%)
Ill-thrift	6	4(19.04)
Respiratory distress	2	2(9.52)
Nervous system problem and lameness	3	2(9.52)
Alopecia and Ill-thrift	13	10(47.62)
Ill-thrift and Respiratory distress	5	3(14.286)
Total	29	21(72.414)

Upon clinical evaluation, among the twenty nine clinical cases, 21(72.4%) were seropositive where as 8(27.6%) were seronegative.

Among 21 seropositive animals with overt clinical disease, 15 (71.4%) were Indigenous Menz breed, 4 (19.1%) were Awassi x Menz cross breed and 2 (9.5%) from Awassi breed.

This result indicated that Awassi breeds could stay a long period of time without any clinical sign (Table 5). Among the clinical signs that were observed, emaciation (ill-thrift) and alopecia was dominant (47.62%) where as respiratory distress (9.52%) and nervous system problems and lameness (9.52%) were the least (Table 5).

## DISCUSSIONS

The overall mean seroprevalence of antibodies in both intervention and non-intervention group to MVV infection in the study population was 176(70.4%). The mean seroprevalence in the non-intervention group of sheep was 79(79%) and none of the yearlings in the intervention group i.e. sheep that were fed on cow's colostrum and milk were isolated from any contact with any flock starting from birth had no any serological evidence of infection. This indicates the effectiveness of prevention of MVV infection by foster feeding the newborn and avoiding contact with infected flocks. In this finding the risk of losing exotic breeds of sheep is avoided. This is inline with the study done by Woldemeskel *et al.* [3] and Garedeew *et al.* [12] who reported 74% (78/105) and 61.3%, respectively, but lower than Ayelet *et al.* [9] and Tsegaw [4] observed lower seroprevalence of 5.4% (11/203) and 6.04% (9/149), respectively showing the disease has not shown too much change despite the ranch has implemented a slaughter policy of clinically sick animals since then.

In this study, the breed related seroprevalence varied considerably from 38% in Awassi sheep to 82.67% in Indigenous Menz sheep. Woldemeskel *et al.* [3] also reported prevalence difference between in Awassi (48%) and Indigenous Menz sheep (92%) in a study conducted on morbid cases. The higher mean seroprevalence observed in this study Indigenous Menz sheep suggest that this breed is particularly susceptible to MVV infection. Differences in breed susceptibility to MVV have been reported by Houwers *et al.* [13] and de la Concha-Bermejillo, [14]. Schaller *et al.* [15] also reported a prevalence that varied between 0.4% and 36% on breeds. Simard and Morley [16] also found a prevalence that varied from 5.6% to 39.3% in Canadian sheep. The Awassi breed, although susceptible to infection do not develop the ovine lentivirus-associated diseases [17].

The study found that there was significant difference age-related mean seroprevalence of MVV infection. The seropositivity increased starting from 3 yrs to older sheep and this could probably be explained by the longer exposure of animals to horizontal transmission and the delay of seroconversion following infection. Seroprevalence reduction in sheep was seen in sheep older than 7.7 years could be explained by the losses through either culling of morbid animals or death of sheep having clinical MV following an infection earlier in their life. This finding agrees with the findings of Woldemeskel *et al.* [3], Tsegaw [4], Ayelet *et al.* [9] and

De la Concha-Bermejillo [14] who reported the infection rate increases with increasing age from 56.25% in young to 100% in older. Garedeew *et al.* [12] reported 12.25% young animals 87.75% in adults.

The mean seroprevalence of MVV in male sheep was 31(73.8%) and 145(69.7%) in female but there was no statistically significant difference in seroprevalence between sexes. Similarly, Woldemeskel *et al.* [3] reported the seroprevalence was not significantly different among sexes. In contrast to the above findings, Tsegaw [4] and Simard and Morley [16] reported a highly significant difference in seroprevalence between sexes, higher in male (24.1%) than female sheep (14.4%) and higher in female (18.9%) and 14.5% in male sheep, respectively. Even though the seroprevalence rate of MVV infection in male and female differs statistically, no one justified so far why this happens.

The mean seroprevalence rate of MV in sheep that fed on their dam's colostrums and milk till weaning stage is 79% where as in sheep that were fed on cow colostrums and milk was nil. There was significant difference in the mean seroprevalence between the two rearing systems. In the fostering lambs there was no any means of contact between their dams and also their house is far up to 1.2 km from their dams' and is hygienic. The result showed that this controlling program can prevent horizontal transmission of the disease. Similarly, Deboer *et al.* [18] reported that none of the lambs that were feed on cow's colostrum showed signs of infection where as 81% of lambs fed on their mother colostrums and which lived together with their dams for one year did not. Cutlip *et al.* [19] have also suggested that preventing colostrum transfer and early contact with infected dams are effective means of obtaining MV free progeny. Similar results were also reported by Houwers *et al.* [13], Light *et al.* [20] and Scheer-czechowsk [21] and others indicated high risk of lactogenic transmission in severely affected flocks. In their study, lambs from seropositive ewes had 7.6 times higher risk to seroconvert within their first two years of life compared to those seronegative ewes. Deboer *et al.* [18] also indicated a contact of 10 hours between the infected dam and her progeny has a 28% probability of cross infection. This finding could be due to the prevention of the lambs from feeding on their dam colostrums and milk and due to avoiding contact between the lamb and dam.

In this study, 29(11.6%) of animals from the total sampled animals were clinically diseased and from the seropositive animals 21(11.9%) had overt clinical disease. Although 8(10.81%) of animals had overt clinical disease,

they were seronegative. This could be explained that those animals may not be infected and/or the signs were due to some other diseases.

Petursson *et al.* [22] reported the incidence of clinical disease depends on how wide spread the MVV infection is in the flock and it will take long period (years) to be detected following the introduction of infection. Sihvonon *et al.* [23] also suggested a wide spread of MVV infection before clinical cases/signs are detected when introduced to free areas.

The seroprevalence of MVV infected sheep that developed overt clinical disease increased as the age increased. This could be due to chronic nature of the disease. Because of the chronic nature of the infection, the clinical signs are mostly seen in animals greater than 3 yrs of age. According to Cutlip *et al.* [19], MVV infections are characterized by a long incubation period and life-long viral persistence and clinical signs are rarely seen in sheep less than 3 yrs old. Constable *et al.* [24] and Verwoerd *et al.* [25] also reported the antibody response confers no resistance to disease and the clinical course of disease is progressive.

The clinical disease was different among breeds and it was higher in Indigenous Menz sheep 15 (83.3%), followed by Awassi-Menz cross, 4 (66.7%). This could be due to the resistance difference among breeds of sheep. Similarly, Tsegaw [4] has suggested that even though all breeds of sheep appear to be susceptible to MVV infection, the low resistance observed in Indigenous Menz sheep.

In the seropositive animals with overt clinical signs, the higher proportion was due to combination of ill-thrift with hair loss followed by ill-thrift, combination of ill-thrift, domination of ill-thrift and respiratory distress. Cutlip *et al.* [19] reported a chronic pneumonia with signs of progressive respiratory failure and progressive emaciation as the most common clinical manifestation of MVV infection. Houwers [7] also indicated increased number of progressively emaciated sheep and increased losses due to pneumonia were associated with the MVV infection.

In conclusion, serological, clinical and gross pathological as well as histopathological findings suggested that the MVV infection is a major health problem in the station. This study also gives a clue what has to be done in the future to control the disease spreading from different breeding and multiplication centers to the farmers and then the centers distribute genetically improved and infection free cross-breed rams to the farmers. As the result of the study indicated, the

main route of transmission of the MVV from infected to non-infected sheep in the ranch could be via ingestion of colostrum and milk from their mothers and contact with infected sheep. In light of this the following recommendations are forwarded.

- Prevention of lambs from feeding their dam colostrum and continuous sera test as well as identification of rams those have genital tract lesions can reduce the transmission rate of the disease from infected to the non-infected in the sheep flock.
- Large flock size, breed difference, older aged sheep presence and management type could be the main risk factors associated with the occurrence and spread of MVV infection in the ranch.
- unless and otherwise a sheep is a valuable progeny, all seropositive animals should be culled and the area should be free from sheep flock for certain period of time to make free the area
- Seronegative animals should be isolated and quarantined to avoid contact with infected or suspected flock and frequent testing of the animals should be practiced until a flock is free from infection and new born lambs from MV seropositive flocks should be isolated and fostered with cow's colostrum and /or milk.

## REFERENCES

1. Central Statistical Authority (CSA), 2009. Federal Democratic Republic of Ethiopia, Central Statistical Authority (CSA), Agricultural Sample Survey 2008/2009 [2001E.C.], Report on Livestock and Livestock Characteristics (Privet Peasant Holdings), Addis Ababa, pp: 120.
2. Tibbo, M., M. Woldemeskel and A. Gopilo, 2001. An outbreak of respiratory disease complex in sheep in central Ethiopia. *Trop. Animal Health Prod.*, 33: 355-365.
3. Woldemeskel, M., M. Tibbo and L.N. Potgieter, 2002. Ovine progressive pneumonia (*Maedi-Visna*): an emerging respiratory disease of sheep in Ethiopia. *Dtsch Tiera. Wochen*, 109: 486-488.
4. Tsegaw, F., 2004. Epidemiological and clinico-pathological study of Maedi-visna virus infection in sheep in selected districts of the Amhara National Regional state, Ethiopia. MSc. thesis, AAU, FVM.
5. De la Concha-Bermejillo, A., 2002. Ovine progressive pneumonia research at the Texas Agricultural Exp. Station: What we have learnt in the last decades. In: *Sheep and Goat, Wool and Mohair CPR*. 129: 129-138.

6. Radostitis, O.M., D.C. Blood and C.C. Gay, 2007. Ovine progressive pneumonia (Maedi-Visna). In: Veterinary Medicine. A Textbook of Cattle, Sheep, Goats, Pigs and Horses, 9<sup>th</sup> ed. London, Bailliere Tindall, pp: 1071-1075.
7. Houwers, D.J., 1990. Economic Importance, epidemiology and control. In: Maedi-Visna and related diseases. G. Petursson and R. Hoff-Jorgensen, (eds.) Kluwer Academic Publishers, pp: 83-117.
8. Pritchard, G.C. and M. Dawson, 2000. Maedi-visna. In Diseases of sheep, 3rd ed. In Martin, W.B and Aitken, I.D (eds). Black well science Ltd. Comp, registered at the United Kingdom, pp: 187-191.
9. Ayelet, G., F. Roger, M. Tibbo and S. Tembley, 2001. Survey of Maedi-Visna in the Ethiopian Highland Sheep. Vet. Rec., 161: 208-210.
10. Thrusfield, M., 2005. Veterinary Epidemiology. 2<sup>nd</sup> ed. Black well science Ltd., pp: 182-198.
11. Office International Des Epizootics (OIE), 2004. medivisna virus in manual of diagnostic tests and vaccine for terrestrial animals, 5<sup>th</sup> ed., Paris: office international des epizootics, pp: 1-21. Philadelphia: W.B. saunders, pp: 582-583.
12. Garedew, L., G. Ayelet, R. Yilma, A. Zeleke and E. Gelaye, 2010. Isolation of diverse bacterial species associated with maedi-visna infection of sheep in Ethiopia. African J. Microbio. Res., 4: 014-021.
13. Houwers, D.J., D.W. Konig, M.J. DeBoer, J.J. Pekelder, P. Vallenia and G. deVaries, 1987. MV control in sheep III. Results and evaluations of a voluntary control program in the Netherlands over a period of four years, Vet. Q., 9: 29-36.
14. De la Concha-Bermejillo, A., 1997. Maedi-Visna and Ovine progressive pneumonia. Vet. Clin. North Am. Food Anim. Pract., 13: 13-33.
15. Schaller, P., H.R. Vogt, M. Strasser, P.F. Nettleton, E. Peterhans and R. Zanoni, 2000. sero prevalence of Maedi-Visna and Border disease in Switzerland. Schw. Arch. Teirh., 42: 145-153.
16. Simard, C.I. and R.S. Morley, 1991. Seroprevalence of Maedi-Visna in Canadian sheep. Can. J. Vet. Res., 55: 269-273.
17. Perk, K., 1995. Characteristics of ovine and caprine lentivirus infections Leukemia-9 (suppl.1): 98-100.
18. DeBoer, G.F., C. Terpestra, J. Hendrinks and D.J. Houwers, 1979. Studies in epidemiology of maedi-visna in sheep. Res. Vet. Sc., 26: 202-208.
19. Cutlip, R.C., H.D. Lehmkuhl, K.A. Brogden and M.J. Schmerr, 1991. Seroprevalence of Ovine progressive pneumonia virus in various domestic and wild animal species and species susceptible to the virus. J. Am. Vet. Res., 52: 189-191.
20. Light, M.R., I.A. Schipper, T.W. Molitor, J.E. Tilton and W.D. Slanger, 1979. Progressive Pneumonia in sheep: incidence of natural infection and establishment of clean flocks. J. Anim. Sc., 49: 1157-1160.
21. Scheer-Czechowski, P., H.R. Vogt, A. Tontis, E. Peterhans and R. Zanoni, 2000. Pilot project for Eradicating Maedi-Visn in Walliser Black nose sheep. Apr., 142: 155-164.(Article in Germany).
22. Petursson, G., U. Andersdottir, O.S. Anderson, G. Georgsson, R.A. Palsson, B. Rafnar and S. Torsteinsdottir, 1992. Lentivirus diseases of sheep and goats: Maedi-visna and caprine Arthritis-Encephalitis. In: speedy, A.W. (ed). Progress in sheep and goat research. C.A.B. International Redwood, U.K., pp: 85-107.
23. Sihvonene, L., V. Hirvela-Koski, L. Nuotio and U.M. Kokkonen, 1999. Serological survey and Epidemiological investigation of Maedi-visna in Finland. Vet. Microbiol., 65: 265-270.
24. Constable, P.D., W.A. Meier, G.L. Foley, D. Morin, R.C. Cutlip and J.F. Zachary, 1996. Visna-like disease in a ram with chronic demyelinating encephalomyelitis. J. Am. Vet. Med. Assoc., 208: 117-120.
25. Verwoerd, D.W., R.C. Tustin and A.L. Williamson, 1994. Maedi-Visna. In Infectious Diseases of Livestock with special reference to Southern Africa. Coetzer, J.A.W., Thomson, G.R., Tustin, R.C. (eds). Oxford University press, Southern Africa, Capetwon, 2: 792-796.