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Short communication

A FIVE-YEAR SURVEY (2002–2007) ON OVINE PULMONARY ADENOMATOSIS IN A MIXED-BREED SHEEP FLOCK

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Summary

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This study was conducted to identify and eradicate ovine pulmonary adenomatosis (OPA) in a sheep flock of different breeds in Iran. The animals included three imported types of Merinos, Chios, and Suffolk and two native types of Mogani and Shal. The adult sheep were monthly examined to find the suspected cases. For final diagnosis, the affected lungs were sent to pathological laboratory to confirm the diseases. By selecting and slaughtering the clinically positive cases, the disease occurrence was dramatically reduced during the 5 years of study and no positive case was diagnosed afterward. Out of 650 sheep examined during the five years, 132 (20.3%) were affected by OPA. There was a trend toward significant difference in susceptibility to OPA (P=0.058) between the sheep breeds. This study suggests that selecting and slaughtering positive cases of OPA are useful means for eradicating the diseases in sheep flocks.

Key words: control, diagnosis, ovine pulmonary jaagsiekte, sheep

Ovine pulmonary adenomatosis is a contagious, viral, neoplastic disease of the lungs of sheep, which has been reported from Europe, Asia, Africa, and America. It is a contagious tumour of sheep causing a progressive respiratory disease and principally affecting adult animals (Querat *et al*., 1987). The etiological agent of OPA, jaagsiekte sheep retrovirus (JSRV), is the only virus known to cause a naturally occurring lung adenocarcinoma (Palmarini *et al*., 1999). There is no laboratory test for diagnosing OPA in the live animal. The period between infection and the appearance of clinical signs may be several months or years and many JSRV-infected sheep do not exhibit clinical signs during their lifespan. This allows the spread of OPA into new flocks through contact with infected but apparently normal animals (Griffiths *et al*., 2010). Clinical signs are shown when the tumours become sufficiently large, or numerous enough to interfere with respiratory function. Diagnosis mostly relies on clinical history and examination of the animal, as well as on necropsy findings and histopathological examination (Palmarini *et al*., 1996). Affected sheep lose weight and show respiratory symptoms such as moist rales heard without stethoscope and frothy mucus

discharge from nostrils with head forced down (Gonzalez *et al*., 1993).

The incidence of the disease is usually 2–5% but in some flocks can reach 10% (Sharp & De Martini, 2003). Two recent studies on sheep lungs obtained from a slaughterhouse in central Iran reported an OPA prevalence of 0.22–3.0% (Kojouri & Karimi, 2002; Khodakaram-Tafti & Razavi, 2010), whereas a JSRV-specific PCR study with blood-extracted DNA from the live sheep in north-west of Iran showed a prevalence of 18% (Rezazadeh *et al.*, 2012). No applied eradication plan for controlling OPA in Iran has been reported. The objective of this study was to identify and control OPA in a sheep flock of five different breeds in Iran during five successive years.

A sheep flock composed of different sex and age groups with ewes, rams and lambs of different breeds kept in a stationary flock from the Animal Science Research Institute of Iran with variable number of sheep throughout the years was used to study OPA occurrence from 2002 to 2007. The breeds included imported Merinos, Chios, and Suffolk and native Mogani and Shal (Table 1). The number of sheep in this flock during the 5 years was around 5200 with culling rate of about 14%. This study was done on adult sheep (more than 2–3 years old), most of them (98%) ewes. They were culled due to different causes such as infections, metabolic, parasitic diseases and physical injuries. The culled sheep were examined to find the suspected cases of adenomatosis. Those which showed difficult breathing, tachypnea, abdominal breathing, emaciation and excess fluid discharge from the nostrils were suspected as OPA cases and slaughtered. In carcass examination, the lungs which were double or triple their normal size, with grey-yellow colour, non-collapsibility with rubbery consistency, and a meaty appearance mostly in ventral part of the lung cross section, were considered as OPA cases (Hecht *et al*., 1994). The mediastinal lymph nodes enlargement resembling a small cucumber was also another sign for identifying OPA cases. Affected lungs were sent to the pathology laboratory of Iranian Razi Institute for histological examination. The laboratory confirmed the disease as OPA. The data were analysed by chi-square test at $P<0.05$ and at $P<0.1$ as levels of statis-

Table 1. Occurrence of ovine pulmonary adenomatosis (OPA) in a sheep flock of different breeds from 2002 to 2007[1]

Breed	Total number of examined (culled) sheep during 5 years	Number (%) of OPA-infected sheep
Merino and its crosses[2]	231	33 (14.3)
Chios and its crosses[3]	125	32 (25.6)
Suffolk and its crosses[3]	195	47 (24.1)
Mogani[4]	33	7 (21.2)
Shal[4]	66	13 (19.6)

[1] Total number of sheep during the 5 years was around 5200 with an average culling rate of 14%; [2] Imported breed crossed with fat-tailed Mogani breed; [3] Imported breed crossed with fat-tailed Gezel breed; [4] Native fat-tailed breeds.

tical significance of differences and trend, respectively (Kaps & Lamberson, 2004).

Out of 650 culled sheep which were examined during the 5 years of study, 132 (20.3%) were affected by OPA (Table 1). With regard to the 14% culling rate, the prevalence of OPA in the flock was 2.8%. By selecting clinically positive cases identified within the flock during the present study and slaughtering them, the OPA occurrence was reduced and the disease came under control after five years of the plan with no positive case diagnosed afterward. Diagnosis and slaughtering at the early stages of the disease can be useful to protect sheep flock against OPA infection and as a result, help improve animal welfare and performance. Previous studies showed variable percentages of the disease incidence in different flocks. Kojouri & Karimi (2002) reported OPA occurrence of about 3% in sheep more than 3 years old. Khodakaram-Tafti & Razavi (2010) reported that the incidence of the disease is usually 2–5% and in contrast of the present study did not observe any metastatic lesions in lymph nodes.

The statistical analysis revealed a trend toward significant (P=0.058) difference in OPA susceptibility between sheep breeds as shown in Table 1. This response suggests a genetic factor contributing to OPA susceptibility. Merino sheep and its crosses with positive case numbers of 33 out of 231 (14.3%) had the lowest susceptibility, whereas Chios and Suffolk breeds and their crosses with positive case numbers of 32 out of 125 (25.6%) and 47 out of 195 (24.1%), respectively, had the highest susceptibility. Native Mogani and Shal breeds with positive case numbers of 7 out of 33 (21.2%) and 13 out of 66 (19.6%), respectively, showed susceptibility around the average of imported breeds. No other study was found reporting genetic susceptibility to OPA. Higher susceptibility of milk- and meat-producing breeds of Chios and Suffolk compared to wool-producing Merino and native breeds may be related to stress due to higher nutrient demands of milk- or meat-producing sheep leading to more OPA incidence although it needs further research.

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REFERENCES

Gonzalez, L., R. A. Juste, R. A. Cuervo, I. Idigoras & C. Saez de Ocariz, 1993. Pathological and epidemiological aspects of the coexistence of maedi-visna and sheep pulmonary adenomatosis. *Research in Veterinary Science*, **54**, 140–146.

Griffiths, D. J., H. M. Martineau & C. Cousens, 2010. Pathology and pathogenesis of ovine pulmonary adenocarcinoma. *Journal of Comparative Pathology*, **142**, 260–283.

Hecht, S. J., J. O. Carlson & J. C. De Martini, 1994. Analysis of a type D retroviral capsid gene expressed in ovine pulmonary carcinoma and present in both affected and unaffected sheep genomes. *Virology*, **202**, 480–484.

Kaps, M. & W. R. Lamberson, 2004. Biostatistics for Animal Science. CABI Publishing, Wallingford, Oxfordshire, UK, pp. 84–90.

Khodakaram-Tafti, A. & Z. Razavi, 2010. Morphopathological study of naturally occurring ovine pulmonary adenocarcinoma in sheep in Fars province, Iran. *Iranian Journal of Veterinary Research*, **11**, 134–138.

Kojouri, G. A. & I. Karimi, 2002. Sheep pulmonary adenomatosis: A study on prevalence and pathological findings. *Pajouhesh-va-Sazandegi*, **53**, 64–67.

Palmarini, M., C. Cousens, R. G. Dalziel, J. Bai, K. Stedman, J. C. De Martini & J. M. Sharp, 1996. The exogenous form of jaagsiekte retrovirus is specifically associated with a contagious lung cancer of sheep. *Journal of Virology*, **70**, 1618–1623.

Palmarini, M., J. M. Sharp, M. De las Heras & H. Fan, 1999. Jaagsiekte sheep retrovirus is necessary and sufficient to induce a contagious lung cancer in sheep. *Journal of Virology*, **73**, 6964–6972.

Querat, G., V. Barban, N. Sauze, R. Vigne, A. Payne, D. York, E. M. De Villiers & D. W. Verwoerd, 1987. Characteristics of a novel lentivirus derived from South African sheep with pulmonary adenomatosis (jaagsiekte). *Virology*, **158**, 158–167.

Rezazadeh, F., G. H. Zarrini, C. H. Cousens & N. Attarilar, 2012. Prevalence of jaagsiekte sheep retrovirus infection in North-West Iran. *Global Veterinaria*, **9**, 535–540.

Sharp, J. M. & A. J. De Martini, 2003. Natural history of JSRV in sheep. *Current Topics in Microbiology and Immunology*, **275**, 55–97.

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