

# Emergence of *Peste des Petits Ruminants* in Sheep and Goats in Eastern Saudi Arabia

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## Key words

Sheep – Goat – Pest of small ruminants virus – Antigen antibody reaction – Epidemiology – Saudi Arabia.

## Summary

By early April 2002, a severe outbreak of a disease was reported in sheep and goats in Al-Hasa province, Hofuf township (lat. 25° N, long. 47° E) of the eastern region of Saudi Arabia. The involved flock was composed of 70 adult sheep and goats. The onset of the disease was sudden. The clinical manifestations were high fever (41°C), lachrymation, nasal discharge, salivation, profuse diarrhea, followed by recumbency and death. The course of the disease in the affected animals took three to four days. Thirty out of seventy animals in the herd were affected (43% morbidity rate). The case mortality rate was 100%. A virus was isolated in Vero cell culture and was reacted in cross virus neutralization tests, using reference hyperimmune sera against *peste des petits ruminants* (PPR) and rinderpest virus. The virus was eventually identified as the PPR virus. The epidemiology of the disease in Saudi Arabia and the Arabian peninsula is discussed.

## ■ INTRODUCTION

The pest of small ruminants, *peste des petits ruminants* (PPR), is known as an acute or peracute disease of sheep, goats and small wildlife ruminants. The disease was first recorded in Côte d'Ivoire in 1942 (11). Since then, it has been reported in African countries between the equator and the Sahara (8, 14). In later years, it has been identified in the Arabian peninsula (2, 10, 24), the Middle East (15, 16) and the Indian subcontinent (20).

PPR is caused by a *Morbillivirus* of the family Paramyxoviridae (4, 6, 12). It is closely related to the rinderpest (RP) virus in physico-chemical and serological characters. However, both viruses can be differentiated by the virus neutralization test (VNT) (14, 19, 21) and competition ELISA (3).

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Based on clinical observations, the PPR virus disease was suspected in Saudi Arabia in sheep between 1977-1979 (5) and in gazelles (13). In both occasions no virus was isolated. In 1990 successful virus isolation was performed from diseased goats (2). The present paper gives evidence for the recrudescence of the disease in sheep and goats in Saudi Arabia.

## ■ MATERIALS AND METHODS

### *Disease outbreak*

During March and April 2002 a disease outbreak was reported in a mixed flock of sheep and goats in a suburb 30 km west of Al-Hasa province (lat. 25° N, long. 47° E). The herd consisted of 70 adult animals. The morbidity rate was 43%, while the mortality rate was 100%. Upon examination, there was fever that reached 41°C. The animals were anorexic and dull. They showed mucopurulent nasal discharge, lachrymation, salivation, profuse diarrhea, recumbency and death. The sick animals were treated with broad-spectrum antibiotics, antipyretics and anti-inflammatory drugs. None of the sick animals responded to the treatments. Epidemiological investigations were carried out.

### *Postmortem examination and sample collection*

Necropsy was performed on moribund animals and samples were presented for virological investigations. These were whole blood in ethylene diamine tetra acetic acid (EDTA), spleen, lymph nodes, liver and lungs. Tissue samples were also collected in 10% formol saline for immunohistochemical examinations.

### *Virus isolation*

The tissue samples were ground and made in 30% (w/v) suspension in F-12 medium without serum (Sigma Chemical). The suspension was centrifuged at 1000 g for 15 min. The supernatant was collected and antibiotics were added (penicillin 10,000 units/ml, streptomycin 10 mg/ml, and amphotericin B 25 mg/ml). Supernatants were used to inoculate Vero and baby hamster kidney (BHK-2) cell cultures, maintained in F-12 minimum essential medium (MEM) supplemented with 5% fetal bovine serum. The cultures were incubated rotating at 37°C and daily examined for appearance of the cytopathic effect (CPE).

### *Virus identification*

#### *Reference sera*

The reference goat anti-PPR-virus hyperimmune serum (PE-26) was kindly supplied by Dr Euan Anderson who was then at AVRI Pirbright, UK. The anti-RP-virus antiserum was a hyperimmune serum produced in rabbits as described by Abu Elzein et al. (2).

#### *Reference virus*

The reference virus was the Kabete 'O' rinderpest vaccine strain. It was used in the agar gel immunodiffusion tests (AGID) and in cross-neutralization tests, as described below.

#### *Agar gel immunodiffusion test*

The AGID method, as specified by OIE (17), was followed to detect the *Morbillivirus* antigen in the suspected tissues (spleen, lymph nodes, lungs and kidneys). Each of these tissues was made into 50% (w/v) suspension in phosphate buffered saline (PBS) pH 7.2. The reference virus was used as positive control antigen. Each of the tissue homogenates and the reference virus antigen were

reacted against the rabbit anti-rinderpest hyperimmune serum in a layout similar to that described by OIE (17).

#### *Virus neutralization test*

The micro VNT using Vero cell culture was used to examine the antigenic relationship between the suspected isolated PPR virus and the reference RP virus. Reciprocal VNTs were performed between the PPR suspected virus and each of the reference PPR and RP sera. The same procedure was repeated for the RP vaccine virus against the two reference sera (2, 23). The log<sub>10</sub> end point titers were calculated as described by Reed and Muench (18).

#### *Fluorescent antibody test*

The indirect fluorescent antibody test (FAT) was used as described by Durojaiye (7) to detect the virus antigen in tissues of the infected animals using an anti-rabbit conjugate. All diluents of the reactants contained 1% ovalbumin, and 0.01% Tween 20 was added to the washing buffer (1).

## ■ RESULTS

### *Postmortem findings*

A sheep and a goat that were moribund were sacrificed for postmortem examination. Both animals showed varying degrees of erosion of the mucosa of the whole gut, from mouth to rectum. The rumen was empty and had congested papillae. The abomasa exhibited tiny hemorrhagic erosions with marked congestion and edema of the pyloric region. Mucosal congestion, hemorrhage and small erosions were observed in the duodenum. The jejunum was congested and Peyer's patches appeared shallow with hyperemic rims. The ileum showed little changes but congestion was evident around the ileocecal valve. The colon and rectum had congested mucosa with linear hemorrhages. The liver, kidney, pancreas and brain were congested. The mesenteric lymph nodes were edematous, and the spleen was small and flat without thickness. The trachea and bronchi were filled with froth, and the lungs were congested.

### *Virus isolation*

The inoculated Vero and BHK-21 cell culture monolayers showed cell rounding by five days postinoculation. The cytopathic effect progressed to form cell aggregates in both cell types. Syncytium formation was seen at low level in both cell systems. Two weeks after inoculation, destruction of the cell monolayers was evident. Passage one (P/1) in each cell system was stored at -86°C until used. Two further passages were made on Vero cells. The virus was designated SG/PPR/Zn/Sau/02.

### *Virus identification*

#### *AGID*

A line of precipitation was produced when each of the tested tissue samples and the rinderpest reference virus antigen were reacted against the rabbit anti-rinderpest hyperimmune serum. These lines merged to make a line of complete identity.

#### *FAT*

Intracytoplasmic fluorescence was detected in sections from the infected animals. Slides which were treated with non-immune serum did not show fluorescence.

#### *VNT*

The PPR reference serum neutralized the suspected virus at a dilution of 1.8 log<sub>10</sub>, while the rabbit anti-RP serum gave a

neutralizing titer of  $0.6 \log_{10}$ . The rabbit anti-RP serum had a titer of  $2.1 \log_{10}$  against the RP virus, but only  $0.3 \log_{10}$  against the SG/PPR/Zn/Sau/02 virus isolate.

## DISCUSSION

The clinicopathological and virological findings in the present outbreak confirmed that the disease was PPR. Although Saudi Arabia had got rid of RP infections few years ago, PPR still seemed to impose a threat to sheep and goats in the country.

It is rather difficult to determine precisely the source of the present outbreak. However, sheep and goats were imported from countries known to be infected endemically with PPR. In addition, PPR has been reported in neighboring countries of the Arabian peninsula and in the Middle East (15, 22, 24), and cross-border animal movements do occur; the disease can thus find its way into the country.

There is a wide spectrum of known natural animal host species for PPR in Saudi Arabia. Sheep and goats constitute a big sector. There are also various species of wild gazelles and deer in zoos, in

private farms and in protectorates administered by the National Commission for Wildlife Conservation and Development. Some of these species have been reported to contract severe PPR infections, such as the gemsbok (*Oryx gazella*), the dorca gazelle (*Gazella dorcas*), and the Ibex (*Capra ibex nubiana*) (10). Other local species of gazelles such as the sand gazelle (*Gazella subgutturosa marica*) could be vulnerable to PPR infection.

These wild species of ungulates are not involved in the routine vaccination programs against PPR in Saudi Arabia. They can create a permanent focus of infection for domestic sheep and goats in the country. Thus, it is advisable to include them in the national PPR vaccination programs. Since little published data is available regarding the epidemiology of PPR in Saudi Arabia (2, 5, 9, 13), it will be necessary to conduct in depth studies to clarify the situation in the country.

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## REFERENCES

1. ABU ELZEIN E.M.E., CROWTHE J.R., 1978. Use of enzyme-linked immunosorbent assay technique in foot-and-mouth disease virus research. *J. Hyg. Camb.*, **80**: 319-399.
2. ABU ELZEIN E.M.E., HASSANIEN M.M., ALAFALEQ A.I., ABDELHADI M.A., HOUSAWI F.M.T., 1990. Isolation of PPR virus from goats in Saudi Arabia. *Vet. Rec.*, **127**: 309-310.
3. ANDERSON J., MCKAY J.A., 1994. The detection of antibodies against PPR virus in cattle, sheep and goats and the possible implication to rinderpest control programmes. *Epidemiol. Infect.*, **112**: 225-234.
4. APPEL M.J.G., GIBBS E.P.J., MARTIN S.J., TERMEULEN V., REMA B.K., STEVENSON J.R., TAYLOR W.P., 1981. *Morbillivirus* diseases of animals and man. In: Kurstak E., Kurstak C. Eds, Comparative diagnosis of viral diseases IV. New York, USA, Academic Press, p. 235-297.
5. ASMAR J.A., RADWAN A.I., 1980. A PPR-like disease of sheep in Central Saudi Arabia: evidence of its immunologic relation to rinderpest; prospects for a control method. In: Proc. 4th Conf. Biological aspects of Saudi Arabia, University of Riyadh, Riyadh, 10-13 March 1980, p. 325-338.
6. BOURDIN P., LAURENT-VAUTIER A., 1967. Note sur la structure du virus de la peste des petits ruminants. *Revue Elev. Méd. vét. Pays. trop.*, **20**: 383-386.
7. DUROJAIYE O.A., 1984. Detection of the antigen of the *peste des petits ruminants* virus in tissues by indirect immunofluorescence technique. *Niger. vet. J.*, **13**: 77-80.
8. EL HAG ALI B., TAYLOR W.P., 1984. Isolation of PPR virus from the Sudan. *Res. vet. Sci.*, **36**: 1-4.
9. EL NAEEM A., ABU ELZEIN E.M.E., AL AFALEQ A.I., 2000. Epizootiological aspects of PPR and rinderpest in sheep and goats in Saudi Arabia. *Revue sci. tech. Off. int. Epizoot.*, **19**: 855-858.
10. FURLEY C.W., TAYLOR W.P., 1987. An outbreak of *peste des petits ruminants* in a zoological collection. *Vet. Rec.*, **121**: 443-447.
11. GARGADENNEC L., LALLANE A., 1942. La peste des petits ruminants. *Bull. Serv. Zool. (AOF)*, **5**: 15-21.
12. GIBBS E.P.J., TAYLOR W.P., LAWMAN M.J.P., BRYANT J., 1979. Classification of *peste des petits ruminants* virus as the fourth member of the genus *Morbillivirus*. *Intervirology*, **2**: 268-274.
13. HAFEZ S.M., SUKAYRAN A.A., CRUZ D., BAKAIRI S.I., RADWAN A.I., 1987. Serological evidence of *peste des petits ruminants* and deer and gazelles in Saudi Arabia. In: Proc. Symp. Potential wildlife conservation, Riyadh, Saudi Arabia, 15-18 Feb. 1987.
14. LEFVRE P.C., DIALLO A., 1990. Peste des petits ruminants. *Revue sci. tech. Off. int. Epizoot.*, **9**: 951-961.
15. LEFVRE P.C., SCHENKEL A., HUSSEIN S., STAAK G., 1991. Serological evidence of *peste des petits ruminants* in Jordan. *Vet. Rec.*, **128**: 110.
16. PERL S., ALEXANDER A., YAKOBSON B., NYSKA A., HARMELIN A., SHEKHAT N., SHIMSHONY A., DAVIDSON N., ABRAMSON M., RAPOPORT E., 1994. *Peste des petits ruminants* (PPR) of sheep in Israel. *Isr. J. vet. Med.*, **49**: 59-62.
17. Peste des petits ruminants, 2001. In: Manual of standards diagnostic tests and vaccines. Paris, France, OIE. [http://www.oie.int/eng/normes/mmanual/A\\_00026.htm](http://www.oie.int/eng/normes/mmanual/A_00026.htm)
18. REED L.I., MUENCH H., 1938. Estimation of end-point 50. *Am. J. Hyg.*, **27**: 493.
19. ROSSITER P.B., TAYLOR W.P., CROWTHER J.R., 1988. Antigenic variation between three strains of rinderpest virus detected by kinetic neutralization and competition ELISA using early rabbit antisera. *Vet. Microbiol.*, **16**: 195-200.
20. SHAILA M.S., PURUSHOTHAMAN V., BHASAVAR D., VENUGOPAL K., VENKATESAN R.V., 1989. *Peste des petits ruminants* in India. *Vet. Rec.*, **125**: 602.
21. TAYLOR W.P., 1979. Serological studies with the virus of *peste des petits ruminants* in Nigeria. *Res. vet. Sci.*, **26**: 236-242.
22. TAYLOR W.P., 1984. The distribution and epidemiology of PPR. *Prev. vet. Med.*, **2**: 157-166.
23. TAYLOR W.P., ABEGUNDE A., 1979: The isolation of *peste des petits ruminants* virus from Nigerian sheep and goats. *Res. vet. Sci.*, **26**: 94-96.
24. TAYLOR W.P., ALBUSHAYDI S., BARRETT T., 1990. The epidemiology of *peste des petits ruminants* in the Sultanate of Oman. *Vet. Microbiol.*, **22**: 341-352.

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## Résumé

**Housawi F.M.T., Abu Elzein E.M.E., Mohamed G.E., Gameel A.A., Al-Afaleq A.I., Hegazi A., Al-Bishr B.** Emergence de la peste des petits ruminants chez des moutons et des chèvres à l'est de l'Arabie saoudite

Au début d'avril 2002, un foyer important de maladie a été rapporté chez des moutons et des chèvres de la province d'Al-Hasa, commune de Hofuf (25° de lat. N, 47° de long. E), dans la région de l'est de l'Arabie saoudite. Le troupeau concerné était composé de 70 moutons et chèvres adultes. L'apparition de cette maladie a été soudaine. Les manifestations cliniques ont été le larmolement, du jetage, de la salivation, des diarrhées abondantes, suivis d'un décubitus et de la mort. La durée de la maladie chez les animaux affectés a été de trois à quatre jours. Sur les 70 animaux du troupeau, 30 ont été affectés et le taux de mortalité chez ces derniers a été de 100 p. 100. Un virus a été isolé en culture de cellules Vero et identifié par neutralisation avec un sérum de référence hyper-immun contre la peste des petits ruminants (PPR) et la peste bovine. Le virus a bien été identifié comme étant celui de la PPR. L'épidémiologie de cette maladie en Arabie saoudite et dans la péninsule arabe est discutée.

**Mots-clés :** Ovin – Caprin – Virus peste petits ruminants – Réaction antigène anticorps – Epidémiologie – Arabie saoudite.

## Resumen

**Housawi F.M.T., Abu Elzein E.M.E., Mohamed G.E., Gameel A.A., Al-Afaleq A.I., Hegazi A., Al-Bishr B.** Brote de peste de los pequeños rumiantes en ovejas y cabras de la zona oriental de Arabia Saudí

A principios de abril de 2002, se informó de la aparición de un foco importante de la enfermedad en ovejas y cabras de la provincia de Al-Hasa, municipio de Hofuf (25° de lat. N, 47° de long. E), en la región oriental de Arabia Saudí. El rebaño estaba formado por 70 ovejas y cabras adultas. La enfermedad se declaró repentinamente. Las manifestaciones clínicas fueron: una temperatura elevada (41°C), lagrimeo, secreciones, salivación, diarreas abundantes, seguidas de decúbito y muerte. La duración de la enfermedad en los animales afectados fue de tres a cuatro días. De los 70 animales del rebaño, 30 se vieron afectados y la tasa de mortalidad de éstos fue del 100%. Se aisló un virus en cultivo de células Vero y se identificó mediante neutralización con un suero de referencia hiperinmune contra la peste de los pequeños rumiantes (PPR) y la peste bovina. El virus identificado era, efectivamente, el de la PPR. Se analiza la epidemiología de esta enfermedad en Arabia Saudí y en toda la península arábiga.

**Palabras clave:** Ovino – Caprino – Virus peste rumiantes pequeños – Reacción antígeno anticuerpo – Epidemiología – Arabia saudita.