



## An outbreak of peste des petits ruminants (PPR) in camels in the Sudan

Abdelmelik I. Khalafalla<sup>a,\*</sup>, Intisar K. Saeed<sup>b</sup>, Yahia H. Ali<sup>b</sup>, Magdi B. Abdurrahman<sup>b</sup>, Olivier Kwiatek<sup>c</sup>, Geneviève Libeau<sup>c</sup>, Ali Abu Obeida<sup>a</sup>, Zakia Abbas<sup>b</sup>

<sup>a</sup> Department of Microbiology, Faculty of Veterinary Medicine, University of Khartoum, Shambat, 13314 Khartoum North, Sudan

<sup>b</sup> Central Veterinary Research Laboratories, Ministry of Sciences and Technologies, P.O. Box 8076, Soba, Sudan

<sup>c</sup> CIRAD, UMR Contrôle des Maladies, F-34398 Montpellier, France

### ARTICLE INFO

#### Article history:

Received 20 March 2010

Received in revised form 2 August 2010

Accepted 4 August 2010

Available online 11 August 2010

#### Keywords:

Outbreak

PPR

Camels

Eastern Sudan

Epidemiology

### ABSTRACT

In mid-August 2004, an outbreak of a previously unknown fatal disease of camels was reported to Kassala State veterinary authorities. Several areas in the state were visited during August–October 2004 to collect epidemiological data and specimens for diagnosis. Clinically the disease was characterized by sudden death of apparently healthy animals and yellowish and later bloody diarrhea and abortion. The disease outbreaks coincided with the seasonal movement of animals towards autumn green pasture. Death was always sudden and proceeded with colic and difficulty in respiration. Mortality rate ranged between 0% and 50% and vary in accordance with the area with a mean of 7.4%. More than 80% of deaths were in pregnant and recently-delivered she-camels. All age, sex and breed groups were affected but more than 50% of deaths were reported in adult animals in comparison to calves and young camels. The main post-mortem findings include lung congestion and consolidation, paleness and fragility of liver, enlarged lymph nodes and congestion and hemorrhage of small intestine and stomach. Agar gel diffusion test (AGDT), RT-PCR and virus isolation in cell culture gave positive results for peste des petits ruminants virus (PPRV), a virus belonging to the *Morbillivirus*, Genus, member of the family *Paramyxoviridae*. The effect of this new devastating disease on camel production in the affected area was discussed as well as proposals for future research.

© 2010 Elsevier B.V. All rights reserved.

### 1. Introduction

Peste des petits ruminants (PPR) by inflicting high losses to livestock is considered as a disease of major economic impact as reported by the World Animal Health Organisation (OIE) particularly in the inter-tropical regions of Africa, in the Arabian Peninsula, the Middle East and Asia (Abu Elzein et al., 1990; El Hag Ali and Taylor, 1984; Govindarajan et al., 1997; Lefèvre and Diallo, 1990; Nanda et al., 1996; Shaila et al., 1989; Taylor, 1984; Wang et al., 2009). The virus belongs to the genus *Morbillivirus*, in the family *Paramyxoviridae*. Highly pathogenic viruses such as rinderpest virus, canine distemper virus, measles virus and marine mammal viruses are closely related members of the genus (Gibbs et al., 1979).

PPR is primarily a disease of sheep and goats. However, there have been several reports of PPR occurring in other wild species,

particularly in captive wild ungulates (Abu Elzein et al., 2004; Kinne et al., 2010).

In acute PPR, pyrexia, oral erosions and pneumonia are the main symptoms of disease. Morbidity and mortality are high (90–100%) when occurring in naive population of small ruminants, the mainly affected species for which PPRV is well documented. However there are limited data on other species such as cattle and buffaloes and particularly camel.

Camels are susceptible to many infectious diseases. Nevertheless, only few viruses were known to cause diseases in this comparatively hardy animal species. Viral diseases commonly seen in the field include camel pox, camel contagious ecthyma, rabies, camel papillomatosis and rotavirus diarrhea. Camels are currently being bred in several provinces of Sudan and represent 4.4 million heads (Anonymous, 2008). Continuous outbreaks of PPR occur since more than 30 years in this country affecting sheep and goats (El Hag Ali, 1973; El Hag Ali and Taylor, 1984; Saeed et al., 2010). It is worth mentioning that when the disease was first noted by El Hag Ali (El Amin and Hassan, 1998), it was thought to be rinderpest even though it was affecting mainly sheep and goats, but a small number of cattle were also clinically affected. Serological surveys have demonstrated that camels are susceptible to the infection (Haroun

\* Corresponding author at: The Arab Center for Studies on Arid Zones and Dry Lands (ACSAD), Damascus, Syrian Arab Republic.

Tel.: +963 574 3039; fax: +963 574 3063.

E-mail address: [Khalafalla@acsad.org](mailto:Khalafalla@acsad.org) (A.I. Khalafalla).

et al., 2002) and in some instance may express a serious illness (respiratory distress) and mortality.

In this communication we report on a newly emerged peracute fatal disease in camels caused by a PPR virus of the genus *Morbili virus*.

## 2. Materials and methods

### 2.1. Field investigations

In the eastern Sudan, the household economy is based on an agro-pastoralist system of production where both livestock and crop production (sorghum) are practiced. Free grazing of rangelands is the most common feeding system for livestock in the area. Goats, sheep, cattle and camels are mingling together allowing inter-species transmission of infectious diseases.

In mid-August 2004, outbreaks of a previously unknown fatal disease of camels were reported to *Kassala State* veterinary authorities. Several areas in the two provinces were visited during August–October 2004 to collect epidemiological data. A total number of 242 camel herds were investigated and their owners interviewed on date of disease outbreak, number of affected animals, mortalities and recoveries, major clinical signs, age and sex distribution of dead animals as well as treatment applied to sick animals.

### 2.2. Post-mortem examination, specimen collection and histopathology

Because of the peracute nature of the disease, only a number of six cadavers were available to do a post-mortem examination. Specimens of lung and prescapular lymph nodes were collected and transported in ice to the laboratory. Pieces of lung and spleens were collected in 10% formol saline, embedded in paraffin wax, sectioned at 5 mm, and stained by routine methods with haematoxylin and eosin (HE).

### 2.3. Laboratory investigations

#### 2.3.1. Culture for bacteria

Routine bacteriological procedures for aerobic and non-aerobic bacteria were performed according to standard techniques.

#### 2.3.2. Immunocapture ELISA (ICE) and agar gel precipitation for antigen detection (AGDT)

Specimens for lung and lymph nodes were homogenized as 20% suspension in phosphate buffered saline with antibiotics and stored at  $-20^{\circ}\text{C}$  until used. Part of the homogenate was tested by agar gel precipitation test (AGDT) PPRV antigens according to standard techniques (OIE, 2009) and also by ICE tests (Libeau et al., 1994).

#### 2.3.3. Reverse transcription polymerase chain reaction (RT-PCR) for amplification of PPRV nucleic acid

The second part of the homogenate was used to extract viral genomic RNA using RNeasy Mini kit (Qiagen, Courtaboeuf, France). The RNA obtained was reverse transcribed using Moloney murine leukemia virus reverse transcriptase using the First-strand cDNA synthesis kit (GEHealthcare, Orsay, France). The PPRV nucleic acid was detected in the clinical tissue material by RT-PCR using primers following the method described by Kwiatek et al. (2007) and Couacy-Hymann et al. (2002) encompassing a highly variable sequence of PPRV N gene. Amplification of 351 bp product was done on a thermal cycler under the following conditions: reverse transcription 30 min at  $50^{\circ}\text{C}$ , initial PCR inactivation during 15 min at  $95^{\circ}\text{C}$  then 40 cycles of amplification corresponding to 30 s at

**Table 1**  
Outbreaks of PPRV infection in two areas of Kassala State, Sudan.

Area	No. of camel herds investigated	Total no. of camels	No. of dead	Mortality rate (%)
Kassala	230	6100	420	6.9
New-Halfa and northern Butana	16	870	96	11
Total	242	6970	516	7.4

**Table 2**  
Age and sex distribution of mortalities due to PPRV infection in camels in Sudan.

Age group	No. of dead	Percentage
Adults (>5 years)	272	53
Young (1–4 years)	135	26
Calves (<1 year)	109	21
Total	516	100

$94^{\circ}\text{C}/30\text{ s}$  at  $60^{\circ}\text{C}/1\text{ min}$  at  $72^{\circ}\text{C}$  and final extension during 10 min at  $72^{\circ}\text{C}$ .

### 2.3.4. Virus isolation attempts

Virus isolation was made on MDBK cells with the original homogenate lung samples. The homogenate was also inoculated onto vero and bovine kidney cell culture as well as into embryonated chicken eggs via the allantoic cavity.

## 3. Results

### 3.1. Field observations

Table 1 shows details on some epidemiological data on the disease in two provinces in *Kassala State*. The disease was believed to originate in early August 2004 in *Al Garada* some 100 km south east of *Kassala* and rapidly spread north and north-westward with the seasonal camel movement (Fig. 1). In early October the disease spreads south-ward and reached central *Butana* region in eastern Sudan (Fig. 2).

Clinically the disease was characterized by sudden death of apparently healthy animals and yellowish and later bloody diarrhea and abortion. Various mild signs of subcutaneous edema and submandibular swelling, chest pain and infrequent coughing, decreased milk production and loss of weight and increased water consumption appeared in some cases and persisted for 10–14 days. Mortality rate ranged between 0% and 50% and vary in accordance with the area.

All age, sex and breed groups were affected. Age distribution of dead animals is shown in Table 2. More than 50% of deaths were reported in adult animals in comparison to calves and young camels.

As shown in Fig. 3 most dead animals (48%) were females recently delivered (within a month period) followed by pregnant females 33%.

According to *Kassala Veterinary authorities* PPR in sheep was present in the area where the camel disease is believed to originate (*Al Garada*) as from December 2003.

In affected cases of the disease there was no satisfactory response to injectable antibiotics and the use of *Hemorrhagic Septicemia* (HS) vaccine produced at CVRL, *Khartoum* failed to stop the spread of the disease towards west *Kassala* and River *Atbara* provinces. After diagnosis, a vaccination campaign using PPR vaccine produced at CVRL was launched in south River *Atbar* and central *Butana* of *Gedarif State* (Fig. 1) covering around 60% of the population, in the area, and from October 2004 no more outbreaks were reported.

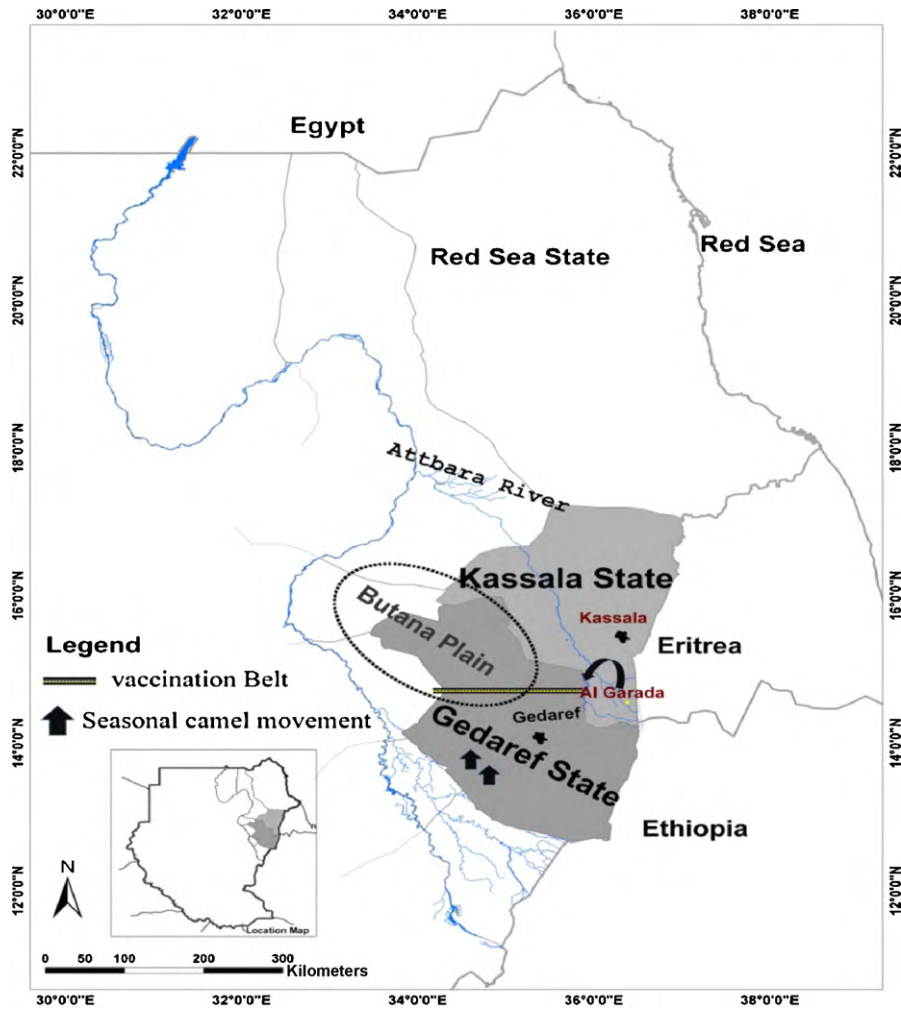


Fig. 1. Map of the Sudan showing area of study.

No human cases related to the disease in camels despite extensive contacts with sick animals.

3.2. Post-mortem findings

The main findings included lung congestion and consolidation mostly in apical lobes, paleness and fragility of liver. Lymph nodes were enlarged and inflamed and small intestine and stom-

ach showed inflammation and hemorrhage. In one case the lips were swollen and hemorrhagic ulcers were seen on the tongue.

3.3. Histopathology

Histopathologically, the bronchioles showed degeneration and denudation of the epithelium and peribronchial infiltration of mononuclear cell (Fig. 4). The alveolar septa was congested and infiltrated by mononuclear cells and in some areas the lung revealed oedema and emphysema. In lymph nodes, the prominent histopathological change was atrophic lymphoid follicles.



Fig. 2. Carcasses of camels died of PPRV infection scattered in northern Butana, Sudan.

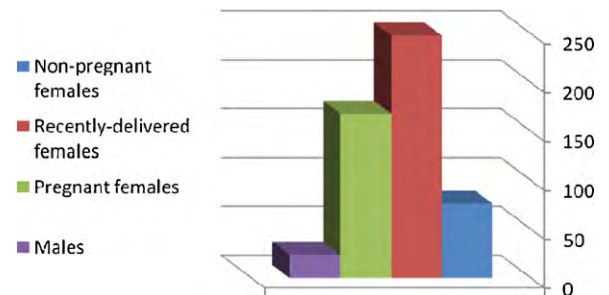
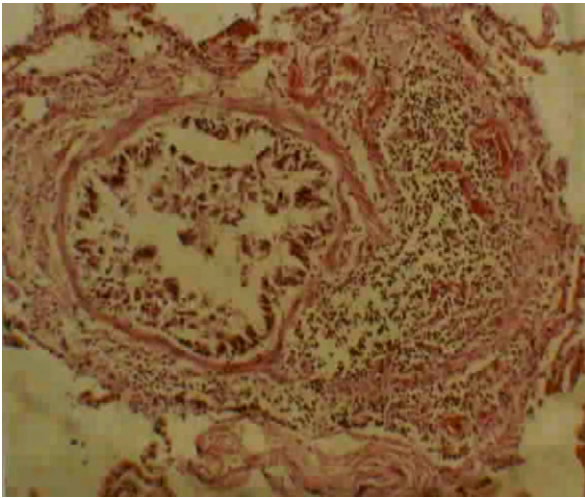
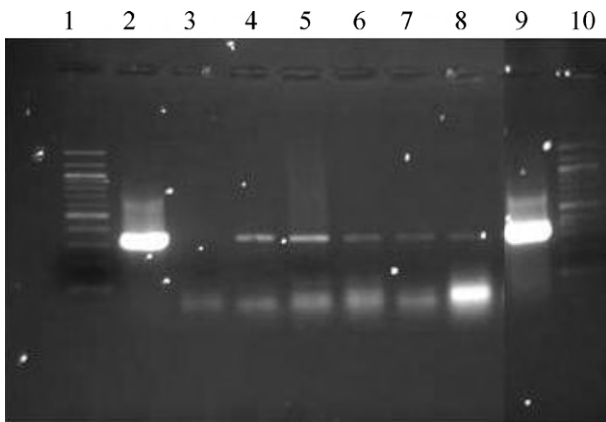


Fig. 3. Distribution of 516 camels died PPRV infection in Sudan by sex and physiological condition.



**Fig. 4.** Section of lung. Note, denudation of bronchiolar lining epithelium and infiltration of mononuclear cells. H&E 10 $\times$ .



**Fig. 5.** Amplified sequences of PPR viral genome in camel samples and from tissue culture isolates recovered from these samples. Ten microliters of the amplified products were analyzed by electrophoresis in 1.5% agarose gel. Lane 1: DNA molecular weight marker (#100 bp ladder, Eurobio); lane 2: positive control (PPR 75-1 vaccine strain); lane 3: no template control; lane 4: camel sample no. 1; lane 5: camel samples no. 3; lane 6: viral isolate from camel samples no. 6; lane 7: viral isolate from camel samples no. 8; lane 8: viral isolate from camel samples no. 9; lane 9: PPR positive goat sample; lane 10: DNA molecular weight marker (#100 bp ladder, Eurobio).

#### 3.4. Laboratory investigations

No pathogenic bacteria were isolated from blood or visceral organs. On AGPT and immunocapture ELISA all specimens (six lungs and six lymph nodes) reacted positively against PPRV antigens.

RT-PCR using Pan-*morbilli* virus primers and PPR-specific primers gave positive results in lung and lymph nodes of five out of six necropsied camels either directly from the camel sample or from tissue culture isolates recovered from these samples (Fig. 5).

A pathogenic agent grew in MDBK cells. Blind passages were made by trypsinization of the MDBK cells until appearance of cytopathic effects. Passage 3 of the agent was confirmed as a PPRV by AGPT, ELISA and RT-PCR. Isolation succeeded with two samples. The virus also grew in vero cell culture and the cytopathic effects (CPE) resembled that produced by PPRV. In chicken embryonated eggs the virus caused death and severe hemorrhages.

#### 4. Discussion

This study describes an outbreak of a previously unknown fatal disease of camels reported in 2004 in Kassala State, East part of Sudan. Some epidemiological as well as laboratory investigations were undertaken in order to understand the etiology of this camel disease. The disease outbreaks coincided with the seasonal movement of animals towards autumn green pasture of Butana plains with an apparent speed of disease supporting a contagious viral infection. Data collected during the field investigation showed that the outbreaks declared in the East part of the country extended northward and to the Central states in almost 3 months and that mortality rates were up to 50%. In general, the clinical feature of PPR in camels, as described in this communication, is not different from those reported in sheep and goats (Abubakar et al., 2009; OIE, 2009). It seems that the severity of PPR in camels is much higher in adult animals since, more than 50% of deaths were reported in adult animals (mostly recently-delivered and pregnant females) in comparison to calves and young camels. There is not, at present, explanation for this observation but according to Abubakar et al. (2009) higher prevalence of PPR in sheep and goats in Pakistan was found in adult animals over 2 years old in comparison to younger groups. Pathological findings, diagnostic investigation on samples collected from diseased camels as well as virus isolation consolidated the aetiology of the disease to be PPRV. The histopathological changes observed in lungs and lymph nodes were suggestive of *morbilli* viruses (Toplu, 2004). However, there is a lack of comprehensive histological material to describe the pathogenesis of this disease in camels. Furthermore, detection of PPRV proteins from tissue samples was made based on AGDT, ICE tests. Finally, conventional RT-PCR, revealed amplified sequences of PPR viral genome as well identified from tissue culture isolates that were recovered from these samples.

The main features of the disease were bloody diarrhea of affected camels, sudden death of apparently healthy animals and abortion of she-camels thus confirming that PPR is also affecting camels. The camel was not reported as a possible host to PPR until Ismail et al. (1992) in Egypt, detected the infection through serology in Sudanese camels. Then serological surveys have indicated the susceptibility of the camels to PPRV (Abraham et al., 2005; Abubakar et al., 2008; Albayrak and Gür, 2010; El Amin and Hassan, 1998; Haroun et al., 2002) although clinical signs have not been observed. According to camel owners no such disease of similar sudden fatality and contagiousness was known in camels in the study area (eastern Sudan). Some years before, in June–July 1992, a contagious acute disease characterized by pneumonia (named camel contagious pneumonia), lacrimation, and difficulty in respiration caused a mortality of about 5% affected camels in southern Butana, southern Gedarif and extended to Kassala provinces [Khalafalla, unpublished]. The cause of that disease was not determined but later a similar disease was reported in Ethiopia in 1995 and laboratory investigations confirmed the involvement of a PPRV (Roger et al., 2000). These authors revealed that the new acute febrile epizootic disease in camel in Ethiopia during 1995–1996 was characterized by highly contagious respiratory syndrome with elevated morbidity and low mortality rates. This was the first documented outbreak of PPR on camels (Roger et al., 2000, 2001).

Our field observations made during the first camel contagious pneumonia episode in Sudan and those made for this study point out variations in the severity of the infection expressed in the occurrence of two forms, acute (1992) and peracute (2004). The reason why PPRV in the last decade increased in virulence and was epidemiologically linked to large outbreaks remains unclear. In a recent publication Saeed et al. (2010) confirmed that PPR is widely spread in Sudan and its incidence increased in most areas with an overall PPR seroprevalence in sheep and goats of 62.8% much higher

than previously reported in Sudan (50%) (Haroun et al., 2002). This phenomenon must be linked to the emergence of a virulent viral strain in this part of Africa, maybe having a selective advantage over the previous strains that were originally present in Sudan resulting in waves of outbreaks in small ruminants and camels as well (Kwiattek et al., 2007). The occurrence over a period of a few years of strains with increased virulence in association with large outbreaks is a phenomenon already observed for measles virus (Santibanez et al., 2002). Molecular typing characterized the strains of PPR discovered in camels in 2004 as lineage IV. Phylogenetic analysis of a wider sampling of PPRV strains isolated between 2000 and 2009 in Sudan evidently showed the emerging of PPRV strains of lineage IV in this country, a lineage present in Asia and in the Middle East. This study also reported on genetic relationship between the PPRV strains circulating in sheep goats and camels and of a possible genetic bias of PPRV according to the host (Kwiattek et al., submitted). Therefore, improved studies of viruses circulating in small ruminants and camels should provide basic data to formulate a policy to control the devastating diseases they cause.

### Acknowledgements

We thank our colleagues Mustafa, Mohamed Salih, Ayman and El Sir Abdelhai of Kassala, New Half and Gedaref Animal Resources Administration for their unlimited help in the field work.

### References

- Abraham, G., Sintayehu, A., Libeau, G., Albina, E., Roger, F., Laekemariam, Y., Abayneh, D., Awoke, K.M., 2005. Antibody seroprevalences against peste des petits ruminants (PPR) virus in camels, cattle, goats and sheep in Ethiopia. *Prev. Vet. Med.* 70, 51–57.
- Abubakar, A.B., El-Yuduga, A.D., Baba, S.S., 2008. Seroprevalence of *Morbillivirus* antibody and abattoir survey of one humped slaughtered camels (*Camelus dromedarius*) in Maiduguri municipal abattoir, Maiduguri, Nigeria. *Asian J. Scient. Res.* 1 (1), 85–89.
- Abubakar, M., Jamal, S.M., Arshed, M.J., Hussain, M., Ali, Q., 2009. Peste des petits ruminants virus (PPRV) infection; its association with species, seasonal variations and geography. *Trop. Anim. Health Prod.* 41 (7), 1197–1202.
- Abu Elzein, E.M., Hassanien, M.M., Al-Afaleq, A.I., Abd Elhadi, M.A., Housawi, F.M., 1990. Isolation of peste des petits ruminants from goats in Saudi Arabia. *Vet. Rec.* 127 (12), 309–310.
- Abu Elzein, E.M., Housawi, F.M., Bashareek, Y., Gameel, A.A., Al-Afaleq, A.I., Anderson, E., 2004. Severe PPR infection in gazelles kept under semi-free range conditions. *J. Vet. Med. B: Infect. Dis. Vet. Public Health* 51 (2), 68–71.
- Albayrak, H., Gür, S., 2010. A serologic investigation for Peste des petits ruminants infection in sheep, cattle and camels (*Camelus dromedarius*) in Aydın province, West Anatolia. *Trop. Anim. Health Prod.* 42 (2), 151–153.
- Anonymous, 2008. Annual Report of Dept. of Statistic and Information, Ministry of Animal Resource and Fisheries, Republic of Sudan, Khartoum.
- Couacy-Hymann, E., Roger, F., Hurard, C., Guillou, J.P., Libeau, G., Diallo, A., 2002. Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay. *J. Virol. Methods* 100, 17–25.
- El Amin, M.A.G., Hassan, A.M., 1998. The seromonitoring of rinderpest throughout Africa, phase III results for 1998. IAEA, VIENNA, Food and Agriculture Organization/International Atomic Energy Agency.
- El Hag Ali, B., 1973. A natural outbreak of rinderpest involving sheep, goats and cattle in Sudan. *Bull. Epizootic dis. Afr.* 12, 421–428.
- El Hag Ali, B., Taylor, W.P., 1984. Isolation of peste des petits ruminants virus from Sudan. *Res. Vet. Sci.* 36, 1–4.
- Gibbs, E.P.J., Taylor, W.P., Lawman, H.P., Bryant, J., 1979. Classification of peste des petits ruminants virus as a fourth member of Genus *Morbillivirus*. *Int. Virol.* 11, 268–278.
- Govindarajan, R., Koteeswaran, A., Venugopalan, A.T., Shyam, G., Shaouna, S., Shaila, M.S., Ramachandran, S., 1997. Isolation of pestes des petits ruminants virus from an outbreak in Indian buffalo (*Bubalus bubalis*). *Vet. Rec.* 141 (22), 573–574.
- Haroun, M., Hajer, I., Mukhtar, M., Ali, B.E., 2002. Detection of antibodies against peste des petits ruminants virus in sera of cattle, camels, sheep and goats in Sudan. *Vet. Res. Commun.* 26 (7), 537–541.
- Ismail, T.M., Hassan, H.B., Nawal, M.A., Rakha, G.M., Abd El-Halim, M.M., Fatebia, M.M., 1992. Studies on prevalence of rinderpest and peste des petits ruminants antibodies in camel sera in Egypt. *Vet. Med. J. Giza* 10 (2), 49–53.
- Kwiattek, O., Minet, C., Grillet, C., Hurard, C., Carlsson, E., Karimov, B., Albina, E., Diallo, A., Libeau, G., 2007. Peste des petits ruminants (PPR) outbreak in Tajikistan. *J. Comp. Pathol.* 136 (2–3), 111–119.
- Kinne, J., Kreutzer, R., Kreutzer, M., Wernery, U., Wohlsein, P., 2010. Peste des petits ruminants in Arabian wildlife. *Epidemiol. Infect.* 138 (8), 1211–1214.
- Lefèvre, P.C., Diallo, A., 1990. Peste des petits ruminants. *Rev. Sci. Technol.* 9 (4), 935–981.
- Libeau, G., Diallo, A., Colas, F., Guerre, L., 1994. Rapid differential diagnosis of rinderpest and peste des petits ruminants infections using an immunocapture ELISA. *Vet. Rec.* 134, 300–304.
- Nanda, Y.P., Chatterjee, A., Purohit, A.K., Diallo, A., Innui, K., Sharma, R.N., Libeau, G., Thevasagayam, J.A., Brüning, A., Kitching, R.P., Anderson, J., Barrett, T., Taylor, W.P., 1996. The isolation of peste des petits ruminants virus from northern India. *Vet. Microbiol.* 51 (3–4), 207–216.
- OIE, 2009. Manual, Chapter 2.7.11 in the Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. Peste Des Petits Ruminants. <http://www.oie.int/eng/normes/MMANUAL/2008/pdf/2.07.11.PPR.pdf>.
- Roger, F., Yigezu, M., Hurard, C., Libeau, G., Mebratu, G.Y., Diallo, A., Faye, B., 2000. Investigations on a new pathological condition of camels in Ethiopia. *JCPR* 7 (2), 163–165.
- Roger, F.M., GuebreYesus, G., Libeau, A., Diallo, L.M., YigezuYilma, T., 2001. Detection of antibodies of rinderpest and peste des petits ruminants viruses (Paramyxoviridae, *Morbillivirus*) during a new epizootic disease in Ethiopian camels (*Camelus dromedarius*). *Revue. Méd. Vét.* 152 (3), 265–268.
- Saeed, I.K., Ali, Y.H., Khalafalla, A.I., Rahman-Mahasin, E.A., 2010. Current situation of peste des petits ruminants (PPR) in the Sudan. *Trop. Anim. Health Prod.* 42 (1), 89–93.
- Santibanez, S., Tischer, A., Heider, A., Siedler, A., Hengel, H., 2002. Rapid replacement of endemic measles virus genotypes. *J. Gen. Virol.* 83 (Pt 11), 2699–2708.
- Shaila, M.S., Purushothaman, V., Bhavasar, D., Venugopal, K., Venkatesan, R.A., 1989. Peste des petits ruminants of sheep in India. *Vet. Rec.* 125 (24), 602.
- Taylor, W.P., 1984. The distribution and epidemiology of peste des petits ruminants. *Prev. Vet. Med.* 2, 157–166.
- Toplu, N., 2004. Characteristic and non-characteristic pathological findings in peste des petits ruminants (PPR) of sheep in the Ege district of Turkey. *J. Comp. Path.* 131, 135–141.
- Wang, Z., Bao, J., Wu, X., Liu, Y., Li, L., Liu, C., Suo, L., Xie, Z., Zhao, W., Zhang, W., Yang, N., Li, J., Wang, S., Wang, J., 2009. Peste des petits ruminants virus in Tibet. *China Emerg. Infect. Dis.* 15 (2), 299–301.