

## REVIEW

# Role of Wild Small Ruminants in the Epidemiology of Peste Des Petits Ruminants

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**Keywords:**

PPRV; wild ruminants; epidemiology; phylogenetic analysis; clinical assessment; diagnosis; control

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**Summary**

Peste des petits ruminants virus (PPRV) causes one of the most contagious and highly infectious respiratory diseases in sheep and goats known as peste des petits ruminants (PPR). Reports of outbreaks of PPR in captive and wild small ruminants have extended the known spectrum of susceptible species to include antelopes. Phylogenetic analysis of nucleoprotein and fusion genes indicates that all PPRVs isolated from wild ungulate outbreaks belong to lineage IV. While it is clear that a number of wildlife species are susceptible to infection, the role of wildlife in the epidemiology of PPR remains uncertain. The available information about the occurrence of disease in free-ranging wildlife is mainly derived from surveys based on serological evidence. Data on the genetic nature of circulating PPRV strains are scarce. Given the scope of PPR in wild ungulates that are widespread in many countries, current disease surveillance efforts are inadequate and warrant additional investment. This is crucial because domestic and wild ruminants mingle together at several points, allowing inter-species transmission of PPRV. There is no reason to believe that PPRV circulates in wild animals and acts as a potential source of virus for domestic species. Irrespective of the possibility of wild small ruminants as the reservoir of PPRV, concerns about the role of susceptible species of antelopes need to be addressed, due to the fact that the disease can pose a serious threat to the survival of endangered species of wild ruminants on the one hand and could act as a constraint to the global eradication of PPR on the other hand. In this review, knowledge gained through research or surveillance on the sustainability of PPRV in wild ruminants is discussed.

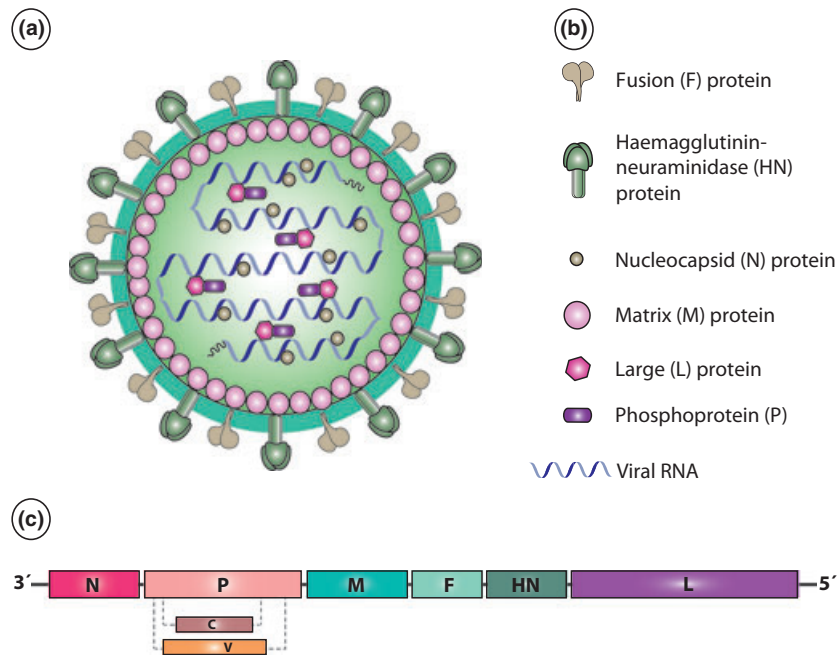
**Introduction**

Peste des petits ruminants (PPR), a disease of both domestic and wild ruminants, is a highly contagious disease, which spreads rapidly regardless of country borders. In this review, several concepts that help to understand the emergence of PPR in wild small ruminants, and the possible role of wild ruminants in the disease epidemiology, are discussed. Moreover, the requirements for surveillance and management of the disease needed for monitoring and elimination of PPR from wild ruminants are described.

**Causative Agent**

Peste des petits ruminants virus (PPRV) causes a highly contagious and economically important disease in both domes-

tic and wild small ruminants, and camels. PPRV is placed in the genus *Morbillivirus*, subfamily *Paramyxovirinae*, family *Paramyxoviridae* and order *Mononegavirales* along with other members of the genus *Morbillivirus*: rinderpest virus (RPV), canine distemper virus (CDV), measles virus (MV) and cetacean morbilliviruses (CeMV) (Gibbs et al., 1979; Barrett, 1999). The host ranges for PPRV and RPV are probably identical and comprise members of the order *Artiodactyla* (Scott, 1964), whereas CD is reported to infect several families: *Canidae*, *Mustelidae*, *Procyonidae*, *Felidae* and *Viverridae* of the order *Carnivora* (Budd, 1981). Like other paramyxoviruses, PPRV is an enveloped virion (mean diameter of 400–500 nm), which contains a genome of single-stranded RNA with negative polarity (Fig. 1a and b). It has a genome length of 15 948 nucleotides (nt), which is the second longest genome among morbilliviruses (Bailey et al.,



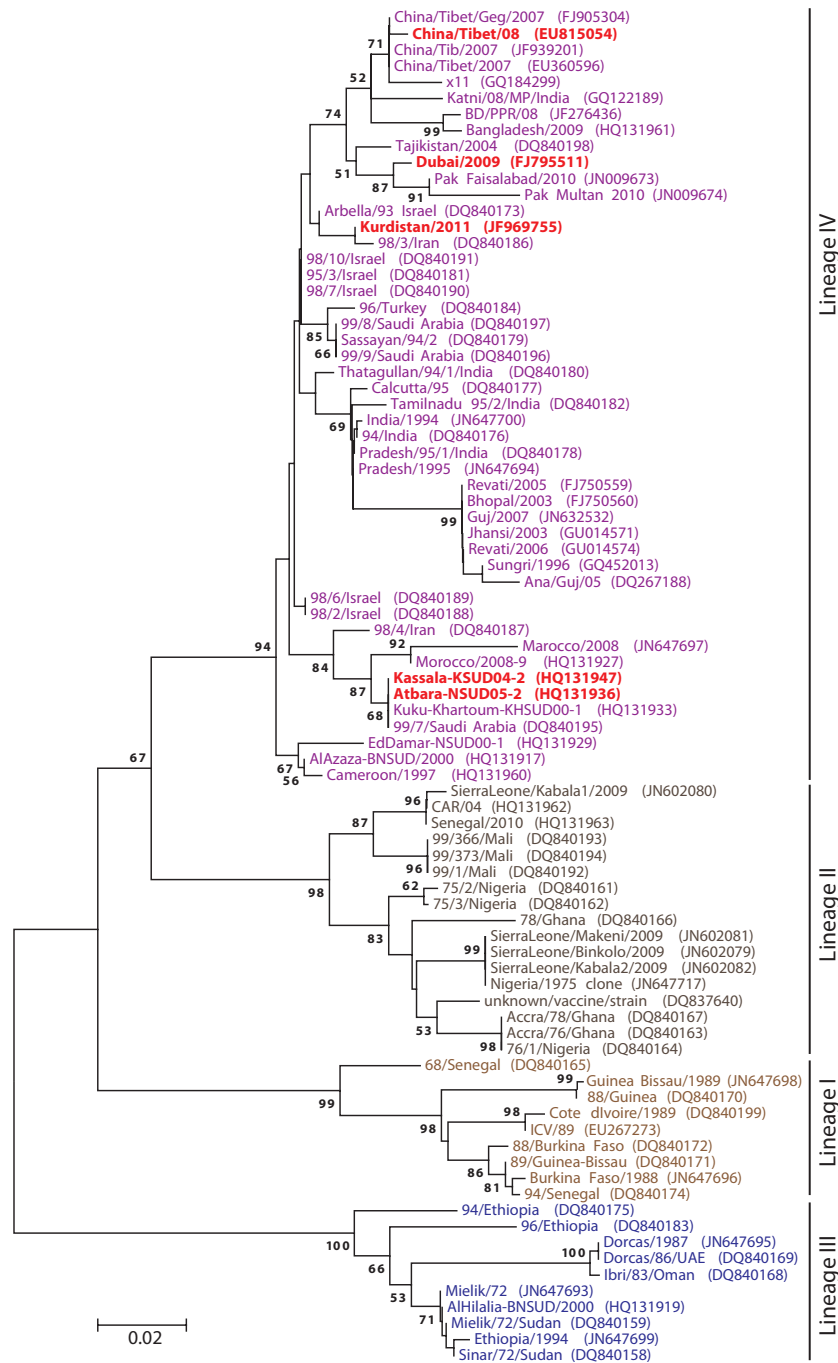
**Fig. 1.** (a) Schematic structure of a typical morbillivirus (PPRV). (b) The structural components of PPRV. (c) The genome organization of all known genes of PPRV.

2005), after a newly identified feline morbilliviruses (Woo et al., 2012). The genome of PPRV contains six transcriptional units, which collectively encode for at least six structural and two non-structural proteins (Fig. 1c). The structural proteins include the nucleocapsid protein (N), the phosphoprotein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin-neuraminidase protein (HN) and the large RNA-dependent RNA polymerase protein (L), whereas the two non-structural proteins, which are encoded in the P gene through an alternative open reading frame and RNA editing, are known as C and V, respectively (Fig. 1c) (Munir et al., 2012a). Although it is plausible to interpret the functions of these proteins based on structural and genetic similarity to the proteins of other morbilliviruses, the mechanism of PPRV assembly and the interactions between viral proteins and host factors warrant future investigations.

### Historical Origin of PPRV

Peste des petits ruminants was first described in 1942 in the Republic of Côte-d'Ivoire in West Africa (Gargadennec and Lalanne, 1942). There are historical records available that provide evidence of the existence of PPRV much earlier (Baron et al., 2011). The lack of earlier diagnosis was probably due to the clinical similarity to ruminant plague (RP) and the use of diagnostic tests, which could not differentiate between them. It is therefore probable that the RP that

was described in small ruminants was actually a PPRV infection (Baron et al., 2011). Additionally, owing to cross-neutralization between PPRV and RPV, it is likely that small ruminants infected with RPV would have developed protective antibodies suppressing the clinical outcome of PPRV infection. Thus, the lack of clinical signs in PPRV infection and inability of the serological tests to differentiate PPRV and RPV may have left PPRV undiagnosed in small ruminants. However, RPV was undoubtedly the cause of some outbreaks of disease in sheep in India in the 20th Century as it was in UK in the 19th century. Nevertheless, the disease gained attention when a severe rinderpest-like disease was observed in sheep and goats that was not transmissible to the cattle reared in the same herd or in the close vicinity. This condition was first reported as bluetongue, followed by ulcerative stomatitis and finally regarded as PPR (small ruminant plague) because of the clinical, pathological and immunological similarities with RP (large ruminant plague) (Gargadennec and Lalanne, 1942). At the time of first PPRV recognition, it was considered a variant of rinderpest virus. However, Gibbs et al. (1979) demonstrated, based on the biological and physicochemical characteristics, that PPRV is distinct enough to be considered a new member in the genus *Morbilliviruses* (Gibbs et al., 1979). As the disease spread, it has become evident that wild small ruminants are equally susceptible and may show the same clinical outcome as the domestic and natural hosts, sheep and goats.

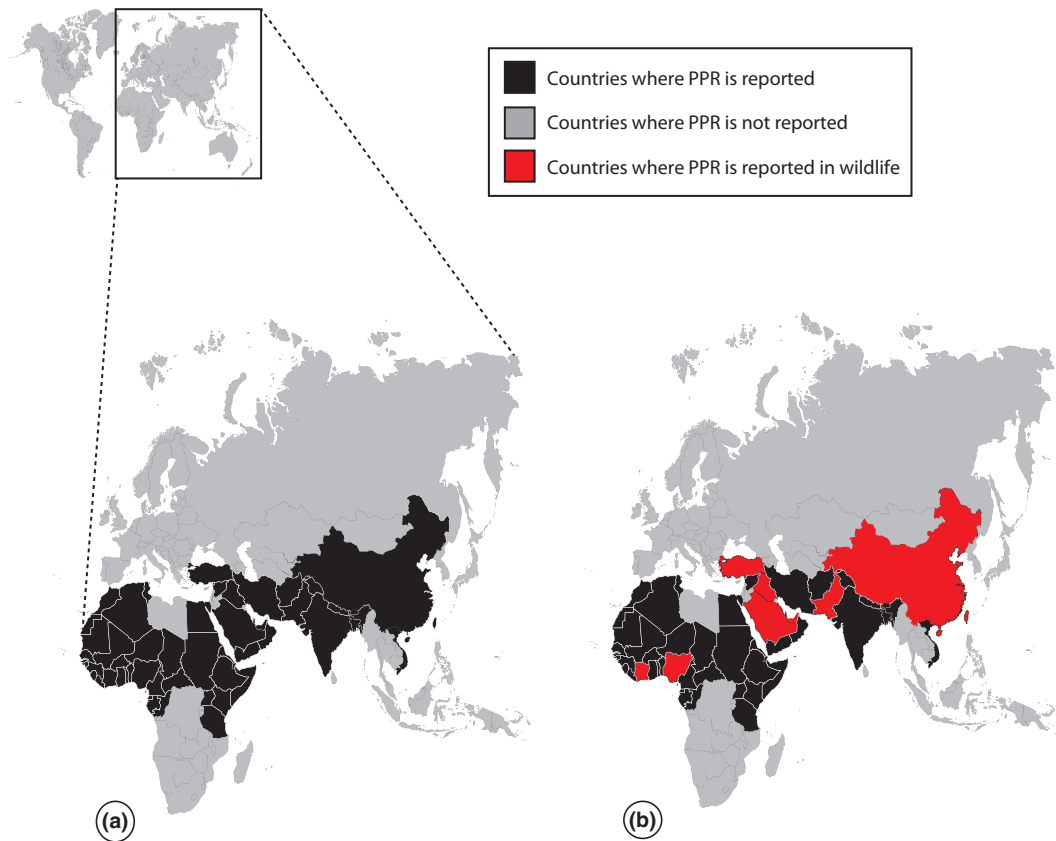


**Fig. 2.** Phylogenetic analysis of the N genes of PPRV representing all four lineages. The PPRV strains characterized from wild small ruminants are coloured red and made bold. All the PPR viruses characterized from wild ruminants clustered in lineage IV.

### Epidemiology and Distribution

Since the first confirmation of PPR, in the Republic of Côte-d’Ivoire in West Africa during the 1940s (Gargadennec and Lalanne, 1942), the disease has been confirmed in most countries in West Africa, such as Nigeria, Senegal,

Togo and Benin, while by 1982 the disease had been diagnosed in Sudan, an eastern African country (Banyard et al., 2010; Munir et al., 2012a). Phylogenetically, based on sequences of the N and F genes, PPRV can be classified into four lineages (Fig. 2). PPRV belonging to lineages I and II are exclusively isolated in West Africa. Lineage III is



**Fig. 3.** Current distribution of PPRV around the globe. (a) The distribution of PPRV is based on reports of disease; either based on serology or genome characterization, to OIE up to 2011, (b) The countries where PPRV is reported in wild small ruminants. Most of these reports were based only on serological demonstration of the antibodies.

restricted to Arabia and East Africa, although virus belonging to lineage III has also possibly been isolated once from southern India. Lineage IV is considered to be a recent lineage comprising newly emerging viruses. Through an unknown source, this lineage succeeded to invade the Middle East and Africa. In Asia, PPRV has recently been reported for the first time in China, Nepal, Vietnam and Tajikistan, while in Africa PPRV has now expanded south of the Equator to Gabon (1996), Kenya (2006), the Congo (2006), Uganda (2007) and Tanzania (2008), and also to the north of the Sahara into Morocco (2007), which indicates its continuing threat to Europe (Fig. 3a). Access of the virus to Europe is now threatened through Turkey and North Africa. Algeria having 19 million sheep and 3 million goats is highly vulnerable to an introduction of PPRV. In fact, Algeria, Tunisia and Libya are now known to have experienced infection (Banyard et al., 2010; De Nardi et al., 2011; Munir et al., 2012a).

Although PPR was formerly restricted to Africa, Asia and the Middle East, its distribution has expanded in the last 10 years (Fig. 3a). Such observations have highlighted the importance of understanding the factors that determine the

distribution and spread of the disease, an understanding that will become increasingly relevant to the success of attempts to eradicate this disease on both a local and global level. The fatal outbreak of PPRV among different species of wild small ruminants kept under semi-free-range conditions in the United Arab Emirates (UAE) in 2005/06 was followed by a clinically identical outbreak of PPRV in another private small ruminant farm in 2008/09 with 100% mortality. Interestingly, the infection appeared in the winter of both years, and the genetic characterization of the N gene of the isolates demonstrated that both belonged to lineage IV of PPRV (Kinne et al., 2010). Notably, they were neither identical to a previously characterized PPRV (lineage III) from UAE in 1986 (Furley et al., 1987), nor identical to PPRV characterized from two neighbouring countries, Saudi Arabia and Oman (Taylor et al., 1990). The genetic relatedness of lineage IV of wild small ruminants with recently characterized Chinese isolates provided clues that importation of domestic or wild small ruminants may play a role in transmission of the disease from Asia to the UAE. Alternatively, based on current reports, it may be that lineage IV is now overwhelming the other lineages in

many countries, such as Sudan and Tanzania (Kwiatk et al., 2011) (Fig. 3b).

The role of wildlife in the spread of PPRV is not completely understood, and it may be too early to draw any firm conclusions. However, the role of domestic small ruminants in the spread of the disease to wild ruminants is clear. One such example was the PPRV in free-living bharals (*Pseudois nayaur*) in Tibet, China (Bao et al., 2011). The two cases of PPRV in bharal have been reported from two different places in the country, each having a history of PPRV in sheep and goats in nearby villages. Moreover, the PPRV isolate in wild ruminants showed high nucleotide identity (99.7–100%) to that of a Chinese PPRV strain isolated from sheep and goats (Wang et al., 2009; Bao et al., 2011). It is likely that in a situation where domestic and wild animals share pastures, the spread of PPRV is facilitated between the two populations. Similarly, the reports of PPRV in Saudi Arabian wild ungulates suggest that the source of infection was sheep and goats (Furley et al., 1987; Frolich et al., 2005) (see below for detail). It is also possible that sheep can be infected asymptotically, due to a degree of natural resistance, and are able to distribute virus over wide areas (Elzein et al., 2004). Recently, high mortality has been observed in Sindh Ibex (*Capra aegagrus blythi*) in a national park in Pakistan. Epidemiological investigations indicated that a clinically similar disease was observed in a nearby village in goats, several months previous to the outbreak in the Ibex (Abubakar et al., 2011). From an epidemiological point of view, species that succumb peracutely to PPR will be less significant in spreading the disease.

Based on the susceptible host range of wild small ruminants, as described so far, it is tempting to speculate that all antelope species are potentially susceptible to PPRV infection (Table 1). Some species of wildlife that are currently known to be susceptible to PPRV infection, such as gazelle (*Gazelle spp.*), bushbuck (*Tragelaphus scriptus*), impala (*Aepyceros melampus*) and duiker (*Cephalophus spp.*), are relatively prevalent in both African and Middle Eastern countries. Other wild small ruminants are more widely distributed in PPRV endemic countries and inhabit most of the pastoral areas alongside sheep and goats. This suggests that there is a great potential for wild ruminants to transmit the disease between domestic and wild species, and this could raise concerns for PPRV eradication.

### Pathogenesis and Clinic Pathology

Based on severity, the disease caused by PPRV is classified into four different courses of manifestation in domestic small ruminants: peracute, acute, subacute and subclinical (Hamdy et al., 1976; Bundza et al., 1988; Lefevre and Diallo, 1990; Roeder et al., 1994). Among these, the acute nature of the disease is most obvious and therefore appears to be most

prevalent among both domestic and wild ruminants. The outcome of any disease type depends upon several factors such as age, season, immune status of the host, concurrent infection, stress and previously existing parasitism. The morbidity frequently reaches as high as 100 per cent. Typically, PPR causes 100 per cent mortality in lambs and kids, 40 per cent in young sheep and goats, but >10% in adult animals (Baron et al., 2011). In captive gazelles, probably the most susceptible wild ruminant species for PPRV, the morbidity rate was 51 per cent and the case fatality rate was observed to be 100 per cent (Elzein et al., 2004).

Peste des petits ruminants is a small ruminant's disease, and sheep and goats are the most common natural hosts for the virus. Goats suffer more severe clinical disease than do sheep (Shaila et al., 1989; Lefevre and Diallo, 1990; Munir et al., 2009). Besides sheep and goats, cattle, buffaloes, camels and pigs can be infected, but they only rarely show clinical signs, except camels. Recent outbreaks of PPRV in camels in the Sudan raised concerns about the role of camels in the epidemiology of the disease; however, this still remains debatable (Khalafalla et al., 2010; Kwiatek et al., 2011).

The initial PPR studies were conducted mostly in domestic ruminants; however, several recent studies have characterized the disease outcome and clinical picture in some of the susceptible wild ruminant species (Hamdy and Dardiri, 1976; Furley et al., 1987; Elzein et al., 2004; Kinne et al., 2010). Based on these studies, it has been observed that the clinical outcome of PPR infection starts with the onset of fever, which ranges from 39–41°C, and the disease appears as an acute ailment in most susceptible wild ruminants. In infected wild small ruminants, similar to other morbilliviruses infections, PPRV replicates in the lymph nodes (primarily in the oro-pharynx, mandibular) and tonsils. During an incubation period of 3–4 days, it causes systemic infection via the blood and lymph. The viraemia spreads the virus to the spleen, bone marrow, mucosa of the gastrointestinal tract (GIT) and the respiratory system, especially the lungs. Infection of the lungs causes primary viral pneumonia, which leads to fast breathing together with lacrimation, congested mucous membrane and nasal discharges in wild goats (Fig. 4a) (Elzein et al., 2004; Bao et al., 2011; Hoffmann et al., 2012). The nasal discharge, which turns mucopurulent from serous over the time course of the infection, may result in crusts over the nostrils that occlude them, results in severe sneezing. The ocular discharges may lead to matting of the eyelids. Ulcerative keratitis and conjunctivitis have been observed in wild goats (Fig. 4b) (Hoffmann et al., 2012), which may cause unilateral corneal opacity (Elzein et al., 2004). One to 2 days post-pyrexia, the oral and ocular mucous membranes become red due to congestion. In wild ruminants, lameness may be present (Bao et al., 2011) or absent (Elzein et al., 2004).

**Table 1.** Wildlife species and the characteristics of the PPRV outbreaks

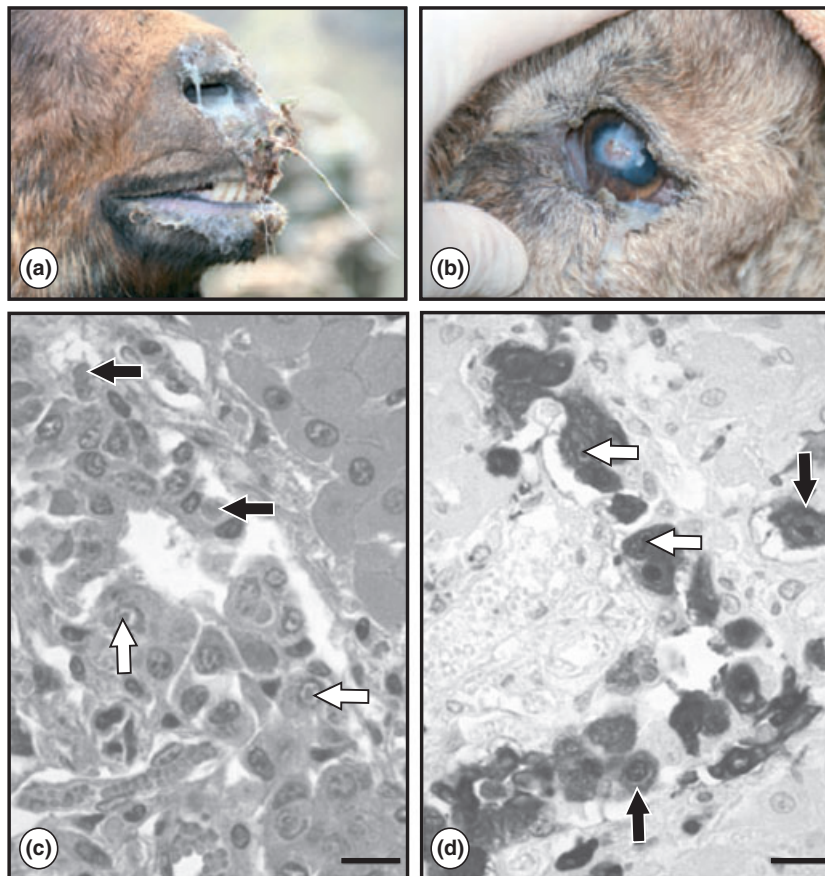
Host taxonomy		Common name			Virus demonstration		Short description of the report		Reference
Family	Subfamily	Genus	Species	Common name	Virus demonstration	Short description of the report	Reference		
Bovidae	Caprinae	<i>Pseudois</i>	<i>P. nayaur</i>	Bharal	Clinical assessment Serology (c-ELISA), RT-PCR, Phylogenetic analysis	PPRV infection was confirmed in two bharals in Rutog County of Tibet, China	(Bao et al., 2011)		
Bovidae	Caprinae	<i>Capra</i>	<i>C. aegagrus</i>	Wild goat	Clinical assessment, RT-PCR ELISA	Clinically suspect wild goats were sampled from Iraq (Kurdistan), and analysis was carried out at FLI, Germany	(Hoffmann et al., 2012)		
Bovidae	Antilopinae	<i>Gazella</i>	<i>G. dorcas</i>	Dorcas gazelle	Clinical assessment Serology	Demonstration of the disease in a zoological collection, United Arab Emirates (UAE)	(Furley et al., 1987)		
Bovidae	Antilopinae	<i>Gazella</i>	<i>G. dorcas</i>	Dorcas gazelle'	Serology (AGID, CNST), Virus isolation, Antigen detection (FAT)	Natural PPR infection in gazelles that were kept under semi-free-range conditions, Saudi Arabia	(Eizein et al., 2004)		
Bovidae	Antilopinae	<i>Gazella</i>	<i>G. thomsoni</i>	Thomson's gazelle	Serology (c-ELISA), Virus neutralization test	Natural infection in goitered gazelle in Turkey that were kept in domestic farms due to fear of their extinction	(Gur and Albayrak, 2010)		
Bovidae	Antilopinae	<i>Gazella</i>	<i>G. subgutturosa subgutturosa</i>	goitered gazelle, or black-tailed gazelle	Virus neutralization test				
Bovidae	Hippotraginae	<i>Oryx</i>	<i>O. gazella</i>	Gemsbok	Clinical assessment, Serology	Demonstration of the disease in a zoological collection, UAE	(Furley et al., 1987)		
Bovidae	Caprinae	<i>Ovis</i>	<i>O. gmelini laristanica</i>	Laristan sheep	Clinical assessment, Serology	Demonstration of the disease in a zoological collection, UAE	(Furley et al., 1987)		
Bovidae	Caprinae	<i>Capra</i>	<i>C. nubiana</i>	Nubian ibex	Clinical assessment, Serology	Demonstration of the disease in a zoological collection, UAE	(Furley et al., 1987)		
Bovidae	Bovinae	<i>Bubalus</i>	<i>B. bubalis</i>	Indian buffalo	Pathology, Virus isolation	PPRV isolation from rinderpest-like diseased animals, India	(Govindarajan et al., 1997)		
Bovidae	Cephalophinae	<i>Sylvicapra</i>	<i>S. grimmia</i>	African Grey duiker	Serology	Sera were collected from Irewole Local Government Area of Osun State in the rain forest vegetation of Nigeria	(Ogunsanmi et al., 2003)		
Bovidae	Alcelaphinae	<i>Alcelaphus</i>	<i>A. buselephus</i>	Bubal hartebeest	Serology (c-ELISA), RT-PCR, Hybridization	Sera and nasal swabs were collected from wildlife of nine different species, Côte-d'Ivoire	Couacy-Hymann et al. (2005)		
Bovidae	Bovinae	<i>Syncerus</i>	<i>S. caffer</i>	African buffalo	Serology (c-ELISA), RT-PCR, Hybridization	Sera and nasal swabs were collected from wildlife of nine different species, Côte-d'Ivoire	Couacy-Hymann et al. (2005)		
Bovidae	Reduncinae	<i>Kobus</i>	<i>K. ellipsiprymnus</i>	Waterbuck	Serology (c-ELISA), RT-PCR, Hybridization	Sera and nasal swabs were collected from wildlife of nine different species, Côte-d'Ivoire	(Couacy-Hymann et al., 2005)		



Table 1. (continued)

Host taxonomy			Common name	Virus demonstration	Short description of the report	Reference
Family	Subfamily	Species				
Bovidae	Reduncinae	<i>K. kob</i>	Kob	Serology (c-ELISA), RT-PCR, Hybridization	Sera and nasal swabs were collected from wildlife of nine different species, Côte-d'Ivoire	(Couacy-Hymann et al., 2005)
Bovidae	Antilopinae	<i>G. gazelle cora</i>	Arabian mountain gazelle	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Antilopinae	<i>A. maysupialis</i>	Springbuck	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Antilopinae	<i>G. gazella</i>	Arabian gazelle	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Caprinae	<i>A. levis</i>	Barbary sheep	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Bovinae	<i>T. scriptus</i>	Bushbuck	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Aepycerotinae	<i>A. melampus</i>	Impala	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Antilopinae	<i>G. subgutturosa marica</i>	Rheem gazelle	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Caprinae	<i>C. falconeri</i>	Afghan Markhor goat	Morphology, Immunohistochemistry, Serology, PCR, Phylogenetic analysis	Clinical disease observed in private collection of different wild small ruminants kept under semi-free-range conditions in the UAE	(Kinne et al., 2010)
Bovidae	Caprinae	<i>C. aegagrus blythi</i>	Sindh ibex	Clinical assessment, Antigen detection, RT-PCR	Disease appeared with high mortality in Sindh Ibex, Pakistan	(Abubakar et al., 2011)
Cervidae	Capreolinae	<i>O. virginianus</i>	White-tailed deer	Clinical assessment, Virus isolation	Experimental infection revealed a disease similar to goat	(Hamdy and Dardiri, 1976)

c-ELISA competitive ELISA; RT-PCR Reverse transcriptase-polymerase chain reaction; AGID Agar gel immune-diffusion test; CNST C cross-neutralization serum test; FAT Fluorescent antibody technique; FLI Friedrich-Loeffler-Institute; UAE United Arab Emirates.



**Fig. 4.** Clinical outcome, histological and immunohistological demonstration of PPRV infection. (a) Mucopurulent nasal discharge and (b) ulcerative keratitis along with conjunctivitis in a wild goat from Kurdistan region of Iraq. (c) Cytoplasmic inclusion bodies (black arrows) and intranuclear inclusion bodies in several hepatocytes in Afghan Markhor goat in UAE. (d) PPRV antigen (black arrows) and inclusion bodies (white arrows) as demonstrated by immunohistological staining in the epithelium of the bile duct collected from PPRV-infected Afghan Markhor. These figures were reproduced from Hoffmann et al. (2012) and Kinne et al. (2010) with permission from the respective publishers.

The gross lesions in wild small ruminants are, in general, identical to those of domestic small ruminants and are characterized by the pinpoint greyish areas of necrosis around the mouth areas, gums, dental pad, palate, lips, inner sides of the cheeks and the dorsal surface of the tongue (Elzein et al., 2004). Dissemination of viral infection to the digestive tract of the infected animals starts 2–3 days post-pyrexia. The oesophagus is usually smeared with thick mucoïd deposits. The rumen is usually congested and empty. The abomasum exhibits tiny haemorrhagic erosions with marked congestion and oedema of the pyloric region. In the small intestine, mucosal congestion, haemorrhages and erosion are evident. Infection in the large intestine causes watery diarrhoea, whereas necrosis of the intestine lining makes the faeces foul smelling. The jejunum becomes congested, and peripheral hyperaemia can be seen in the Peyer's patches (Elzein et al., 2004; Kinne et al., 2010). The ileum is the least affected part of the GIT, and congestion is more prevalent in the ileocaecal valve. The congestion in

the terminus of the large intestine, colon and rectum causes linear discoloration known as zebra striping, considered a pathognomonic sign in both domestic and wild small ruminants. The large intestine is more severely affected than the small intestine. Persistent diarrhoea leads to dehydration and protein loss causing weight loss, prostration and death. Death occurs in approximately 3 days in wild small ruminants (Elzein et al., 2004). It is likely that small nodular lesions may appear outside the lips and around the muzzle in later stages of the infection. Besides the GIT and respiratory tracts, congestion is obvious in other visceral organs such as the liver, kidney, pancreas, spleen and brain in wild ruminants (Elzein et al., 2004; Kinne et al., 2010).

Histopathologically, large necrotic or haemorrhagic lesions are evident in the alimentary tract, spleen and lymph nodes in both domestic and wild small ruminants (Elzein et al., 2004; Kinne et al., 2010). Lungs undergo severe broncho-interstitial pneumonia with hyperplasia of type II pneumocytes. Being sites for PPRV replication, the



spleen, tonsils, and oro-pharynx and mandibular lymph nodes show marked necrosis of lymphocytes. The tracheal and bronchial and bronchi epithelial cells, pneumocytes, alveolar macrophages and pathological syncytial cells show viral antigen upon immunohistochemical staining. In the liver, multifocal hepatocellular coagulation necrosis with infiltration of macrophages has been observed in wild small ruminants (Fig. 4c) (Kinne et al., 2010). Eosinophilic cytoplasmic and nuclear inclusion bodies can also be found in gastrointestinal epithelial cells, macrophages/reticular cells of lymphoid tissues, bronchial and bronchiolar epithelial cells, syncytial cells and biliary epithelial cells (Fig. 4d).

### Transmission and Host Range

Transmission of PPRV from infected animals is via the discharges from the eyes, nose and mouth, as well as the loose faeces, all of which contain high titres of the virus and usually occurs by an aerosol over a very short distance. Besides close contact, which remains the most frequent means of disease transmission, contaminated water, feed troughs and bedding may act as additional routes of transmission. Fortunately, the virus does not survive for long outside the host, and therefore, most transmission occurs during the febrile stage of the disease (Braide, 1981).

### Susceptibility of Wild Ruminant Species for PPRV

Initially, the disease has only been reported in (and thought to be confined to) the natural hosts, domesticated sheep and goats. The susceptibility of wild small ruminants was not confirmed until 1976, when Hamdy and Dardiri (1976) experimentally infected American white-tailed deer (*Odocoileus virginianus*) with Senegalese and Nigerian strains of PPRV (Hamdy and Dardiri, 1976). As expected, based on reports of RPV in wild ruminants, the experimentally infected deer showed clinical signs, which varied from fatal to subclinical. The clinical picture appeared to be the same as that in sheep and goats, in which respiratory tract and GIT were the most affected body systems. The clinical picture, accompanied by the isolation of the virus from the blood and lymph nodes of infected animals, confirmed the susceptibility of wild small ruminants for PPRV infection and added another dimension into the host range dynamics of the PPRV. Later reports of the PPR disease in a wide range of wild small ruminants species have clarified the full susceptibility to natural infection. Based on these reports, it has been observed that PPRV mostly causes fatal and acute disease with high mortality, but the disease outcome varies. A complete list of susceptible wild ruminants along with a short description of the severity of disease is provided in Table 1.

Most of the reports of wild ruminants that died because of natural infection with PPRV are from Al Ain in the

Persian Gulf, where Furley et al. (1987) described the disease, for the first time, in Dorcas gazelle (*Gazella dorcas*) and gemsbok (*Oryx gazella*) and subclinical infection in Nilgai (*Tragelophinae*) which were held in a zoological collection. As a result of this report, the existence of sylvatic reservoirs for PPRV has been postulated (Table 1). Collectively, these outbreaks of disease, often described in wildlife collections living in semi-free-range conditions, have shown that many species are susceptible. Regardless of sampling bias, Gazelle species were found to be the most susceptible animals.

Although the host range of peste des petits ruminants in wild animals is still not completely known, it is possible that PPR could threaten the conservation of some wildlife species that are already at risk of extinction. Moreover, the majority of the susceptible wild small ruminant species are reduced and the areas in which they live are poorly protected and increasingly subject to incursion by land-hungry farmers and their small ruminants. The wild populations are therefore increasingly at risk and vulnerable.

The host, pathogen and environment interact in a complex manner to determine the outcome of the disease in an exposed population. One such outcome can be host resistance to infection that renders the host as a 'dead-end', unable to maintain the infection without an external source, or as a 'spillover' capable of maintaining the infection for a certain period of time but requiring input from another host. An alternative outcome can be a 'maintenance host', which is able to maintain infection within the population without further transmission to another species. Among these, the maintenance hosts are considered epidemiologically significant because they are able to transmit the virus to other susceptible wildlife or to sheep and goats.

The role of wild small ruminants in the maintenance of the PPRV is not fully elucidated yet. In order to understand the reintroduction or maintenance of several pathogens, including PPRV, a serological study was conducted on 294 sera collected from both captive and free-ranging Arabian Oryx (*Oryx leucoryx*) in the UAE and Saudi Arabia (Frolich et al., 2005) where Oryx are considered a highly vulnerable species close to extinction. Samples from 1999–2001 showed that this species remained serologically negative for PPRV, although they were positive for several other enzootic diseases. In contrast, a natural infection has been observed in goitered gazelles in Turkey, with low mortality compared to other susceptible wild small ruminant species (Gur and Albayrak, 2010). The virus was neither isolated nor characterized. In a study conducted by Couacy-Hymann et al. (2005), analysis of serum samples from nine different species indicated a low seroprevalence of antibodies to PPRV in three farms in Côte-d'Ivoire. Although PPR was confirmed by both serological and antigen detection methods, the isolates or the involved strains remain to be

characterized genetically. Based on this study, it was proposed that PPRV was unable to sustain itself in wildlife (Couacy-Hymann et al., 2005). Such interpretation is now well accepted for RPV, a virus similar to PPRV where the outbreaks of RP in wildlife were due to spillover from cattle (Kock et al., 2006).

### Density of the Wild Ruminant Populations

Data available on the spread of PPR within a susceptible wildlife population suggest that PPRV can temporarily establish itself in relatively small populations. However, larger populations are required to sustain the virus for longer periods of time as infection induces a long-lasting and sterile immunity in recovered animals. Even less is known of the determinants of virus persistence in populations than is the case for rinderpest. However, by analogy with rinderpest, it is possible that diminished virulence might actually help to sustain the virus in an extensive population (Mariner et al., 2005). Owing to their larger populations, wild ruminants in African countries may play a significant role in the spread of PPRV compared to Middle East and southern Asia. However, the disease has only been identified serologically in Nigerian wild ruminants (Ogunsanmi et al., 2003).

### Distribution of Wild Ruminant Populations

The PPRV-susceptible wildlife species are widely spread over all PPR endemic regions, especially in most African countries. However, it is of note that PPR is not reported in most of the countries where the wild susceptible populations are largely concentrated. This is primarily due to either lack of an efficient surveillance system to screen the entire population for virus genome detection or inability of wild ruminants to maintain the virus for a longer time. Additionally, expansion of the human population is increasingly encroaching on wildlife and it is plausible that wild ruminant populations might not be able to support sustained transmission of PPRV as is the case with rinderpest. However, given the intensive migratory patterns of wild ruminants, the potential exists for them to amplify and spread the virus over long distances (Anderson, 1995).

### Pattern and Range of Movement

Peste des petits ruminants virus transmission among susceptible wildlife population greatly depends upon the rate of contact between herds of different or the same species. The movement of susceptible small ruminants may contribute to the spread of infection within the Middle East (Fig. 5) (Mallon and Kingswood, 2001). The movement of animals can be in the form of natural migration or

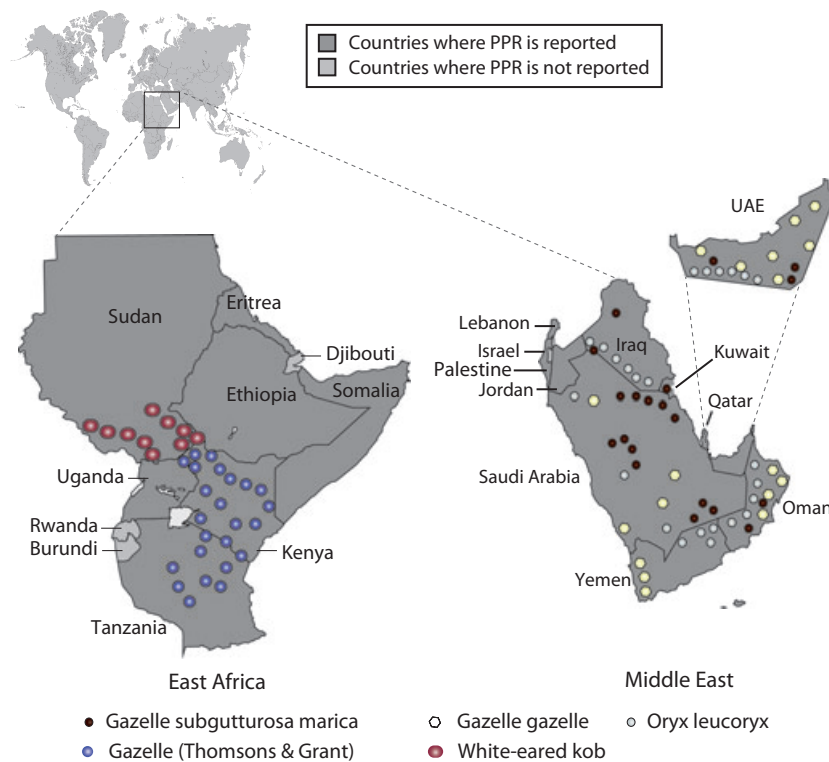
transportation of the animals to zoological gardens. Seasonal migrations can spread infection over significant distances, oblivious of borders. However, transportation of wild ruminants can result in spread of the virus across countries and even continents. Recently, PPRV has been demonstrated both serologically and genetically in free-living bharals (*Pseudois nayaur*) in Tibet, China (Bao et al., 2011). The bharal is an abundant species not only in China, but is also frequently found in the Tibetan Plateau of China and the high Himalayas of Nepal, Pakistan, Bhutan and India. Domestic and wild animals share pastures, which facilitates the spread of PPR between the two populations. Therefore, it is likely that the PPR virus was transferred from domestic small ruminants to wild bharals while at pasture (Bao et al., 2011). However, the contact of bharal with other wild and domestic ruminants species could spread the PPRV across the bharal's geographic distribution. The same may also be possible in many African countries, although the disease has only been described serologically in a limited number of wildlife species. It is likely that PPR virus has only recently been introduced into those countries with the largest populations of the most susceptible gazelles, such as Kenya, Uganda and Tanzania, and has still not been spread to Southern Africa.

### Behavioural Influence on PPR Spread

As described above, the spread of PPRV infection requires close contact between infected and susceptible hosts. Therefore, the species of wild ruminants that tend to live in herds have a greater chance of acquiring PPRV infection. The females and the immature offspring of Thomson's gazelle, one of the wild ruminant species most susceptible to PPRV, are social animals and live in herds of over 200 individuals. Additionally, Thomson's gazelle congregate with Grant's gazelle and with larger ungulates such as wildebeest, zebra and cattle. This could facilitate the transmission of any pathogen, including PPRV between different and similar species.

### PPRV in Unusual Hosts: Cattle, Buffaloes, Pigs and Camels

Besides domestic and wild small ruminants, it has been reported that PPRV can successfully infect several other species of mammals, including cattle, buffaloes, pigs and camels. PPRV has been isolated once from Indian buffaloes in Tamil Nadu, India, where 50 of 385 buffaloes were clinically infected and showed conjunctival congestion, profuse salivation and depression, but none showed a febrile response (Govindarajan et al., 1997). However, no further investigations were made about the source of infection or



**Fig. 5.** Distribution of some of the known species of wild small ruminants. The distribution patterns of selected species were obtained from Mallon and Kingswood (2001).

evidence of virus secretion. PPR has also been reported both serologically and genetically in African buffaloes (Couacy-Hymann et al., 2005). Cattle may be infected without showing any clinical signs on experimental inoculation. However, in poor conditions, it might be possible that cattle develop lesions following PPRV infection, clinical signs which could be ascribed to rinderpest. Disease and death were described in calves experimentally infected with PPRV-infected tissue (Mornet et al., 1956). During a 10-year surveillance programme on RP, antibodies against PPR infection were determined in parallel in African buffaloes and several species of wild small ruminants (Koch, 2011). The area of detected PPR antibodies in Ugandan buffalo increased significantly during 2002–2004. No further information was forthcoming due to the lack of resources; however, it has been shown that buffaloes can seroconvert but their ability to spread virus has not been proven so far. Both cattle and buffaloes are considered dead-end hosts and might not play any significant role in the epidemiology of the disease, although this may depend on the strain of PPRV. Pigs undergo subclinical infection by experimental inoculation or contact with infected goats but are unable to transmit the virus, and therefore are not regarded as important in the epidemiology of PPR (Nawathe and Taylor, 1979).

Initially, camels were not included in the host range of PPRV; however, in 1992 and afterwards, several reports described antibodies against PPRV in camels (Ismail et al., 1992; Haroun et al., 2002; Abraham et al., 2005; Albayrak and Gur, 2010). Based on these reports, it is possible that camels might play a role in the epidemiology of the disease. It was not until 1996 that the first outbreak of PPRV was documented in Ethiopian camels, causing a highly contagious disease characterized by pneumonia, lacrimation and respiratory distress, with comparatively low mortality (Roger et al., 2000). This outbreak was latter confirmed to be caused by a PPRV (Roger et al., 2001) belonging to lineage III (A. Diallo, unpublished data, described in (Kwiatk et al., 2011)). Recent studies have demonstrated the first virological, epidemiological investigations and successfully isolated the virus from Sudanese camels (Khalafalla et al., 2010; Kwiatek et al., 2011). Based on the results presented in these studies, it was indicated that lineage IV of PPRV is currently replacing the existing lineage III in camels in the Sudan. Because vaccination against rinderpest has been stopped, it is believed that RPV-immune camels have now been replaced with a new generation that are fully susceptible for PPRV (Kwiatk et al., 2011). Although it is believed that camels may disseminate PPRV between countries, a great deal of research into this aspect of the disease is urgently required.

## Molecular Diagnosis and Control of PPRV

Diagnosis of PPR is usually made by clinical observation, and in typical cases, animals show characteristic signs. However, due to the presence of aggravating factors or concurrent infections, the disease may be confused with several clinically similar diseases. In this case, serological or virological confirmation is required. The diagnosis can be based on virus isolation (Taylor and Abegunde, 1979), detection of viral antigens (Obi, 1984; Abraham and Berhan, 2001), detection of viral genome or sequencing of viral segments (Couacy-Hymann et al., 2002; Bao et al., 2008; Munir et al., 2012b,c). The detection of specific antibodies against PPRV is the most common and economical way of identifying evidence of infection in a geographic area, because antibodies against the virus remain in infected animals for a long time. The available serological techniques for the diagnosis of PPRV have been reviewed (Munir, 2011; Munir et al., 2012d).

The control and eradication of PPR is possible because of the availability of very efficient and economical vaccines. An attenuated tissue culture vaccine based on Nigeria75/1 (Nig75/1), one of the very first isolates of PPRV, is protective for at least three years, and immunized animals are unable to transmit the disease to nearby healthy flocks. The vaccine appears to be safe for pregnant animals and, under field conditions, induces protective immunity in at least 98% of vaccinated animals (Diallo et al., 1995). This vaccine is currently being extensively used in the endemic areas of Africa, the Middle East and South Asia, whereas three other vaccines are currently in use in India. Less well-characterized vaccines are also being produced in Egypt and Bangladesh.

## Future Perspectives

There is an advantage in PPRV vaccination that is lacking in many other epizootic diseases. The animals that recover from infections or following proper vaccination can develop sterile protective immunity, which persists for at least three years and may last for life. Vaccination of captive wild ruminants and domestic small ruminants will reduce opportunities for the transmission of PPRV. Furthermore, the PPRV-susceptible wildlife species such as gazelle, deer, wild sheep or feral goats should be prevented from having close contact with domestic sheep and goats. In either situation (with or without vaccination), reducing other bacterial and parasitic complications in wild or captive ruminants will reduce the PPRV mortality in endemic regions.

Having in mind the currently available PPRV vaccines, it seems not to be practical to vaccinate all the wild small ruminants in captivity or protectorates. Future research

could develop oral PPRV vaccines with the thermostability required to facilitate their use. Ideally, like existing vaccines, novel vaccines should induce life-long immunity.

The primary objective of future PPRV research in wild ruminants should be to completely characterize the strains of PPRV isolated from wildlife. This characterization is fundamental to understand the genetic nature of these strains of PPRV and will also help to fill the epidemiological gaps that currently exist with respect to wild small ruminants.

## Conclusions

Based on available observations, it is plausible to conclude that PPRV infection is not self-sustaining in wild small ruminants, and most of the epidemics of PPR probably originate from nearby infected domestic sheep and goats. However, such a hypothesis needs to be tested. The disease can pose a serious threat to the survival of endangered species of wild ruminants. This danger has already been identified for endangered species such as Arabian oryx (*Oryx leucoryx*) in the Middle East and the goitered gazelle (*Gazella subgutturosa subgutturosa*) in Turkey. Only global eradication of PPR will effectively safeguard the valuable heritage of wild small ruminant populations. Concern regarding the role of wild small ruminants in the epidemiology of PPRV is increasing due to the current increase in reports of the disease in wildlife population, but the lack of data on the epidemiology of PPRV in different wildlife species might be a constraint on the development of effective strategies for global eradication of PPR.

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