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PESTE DES PETITS RUMINANTS (PPR) IN CAMELIDS WITH OWN INVESTIGATION

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ABSTRACT

Peste des petits ruminants (PPR) is a contagious virus disease of domestic and wild small ruminants. PPR has steadily expanded over the last 2 to 3 decades and occurs regularly in large regions of Africa, the Middle East and Asia. Transmission is mainly oronasally and the disease is clinically characterised by fever, gastroenteritis, bronchopneumonia and erosive lesions of mucous membranes. There are 4 known phylogenetic lineages of the PPRV. A great variation in the pathogenicity of the virus exists. OWCs can contract the disease and outbreaks have been described in Ethiopia, Sudan and Saudi Arabia. Experimental infection with an Ethiopian goat strain in a small number of dromedaries in Dubai did not produce any clinical signs. There is a need for further infection trials with different PPR strains to elucidate the role of camels in the epidemiology of PPR.

Key words: Camelids, infection trial, peste des petits ruminants (PPR), rinderpest (RP)

Peste des petits ruminants (PPR) is a severe contagious viral disease of domestic and wild small ruminants. It is considered the most economically important disease of these species in enzootic regions. Given that sheep and goats are more economically important than cattle in many regions of the world that rely on pastoralism, PPR has a major impact on the food supply of these regions. Since the disease was first described in 1942, the disease has steadily expanded and includes large regions of Africa, the Middle East, and Asia (Fig 1). The disease readily crosses boundaries, and is now considered the most constraining disease of small ruminants in sub-Saharan Africa and the Indian sub continent.

The eradication of Rinderpest from the globe has elevated viral diseases to a different level. New research on PPR has been performed also on the camelid family, and it was found that dromedaries can contract the disease (Khalafalla *et al*, 2010). NWCs so far have been excluded from the disease, but owners and veterinarians should be aware of PPR as more and more NWCs are nowadays living in countries where PPR occurs in small ruminants. PPR is an OIE notifiable disease.

Aetiology

There is a strong clinical resemblance between RP and PPR, and therefore it was concluded some time ago that the causal agent of PPR was a variant of the RP virus, that was well adapted to small ruminants, and that was less pathogenic to cattle. However, a clear differentiation between RP virus and PPR virus was made in 1970s after different serological and cross-protection studies. The PPRV belongs to the genus *Morbillivirus* within the *Paramyxoviridae* along with RPV, Canine Distemper Virus (CDV) and Measle virus (MV). In this group the phocine distemper virus (PDV) and the cetacean *morbillivirus* of dolphins and porpoises are also found. It is now obvious with gene sequence data analysis that PPRV is not a variant of the RPV and not even close to RPV as MV is. There are 4 known phylogenetic lineages of PPRV. Lineage I and II viruses have been found exclusively in West Africa, whereas lineage III occur in East Africa, South India and Arabia. Lineage IV has recently emerged in Asia and the Middle East. The source of this virus is unknown, although it is most closely related to the African Lineage I (Fig 1). Kinne *et al* (2010) described a PPR outbreak in the United Arab Emirates in several different wild ruminants kept under semi-free-range conditions with diarrhoea and more than 100 fatalities. The PPRV belonged to lineage IV. The authors assume that wild ruminants may play an important epidemiological role as virus source for domestic small ruminants. There are also well-documented examples which highlight the emergence of new strains through cross-species transmission.

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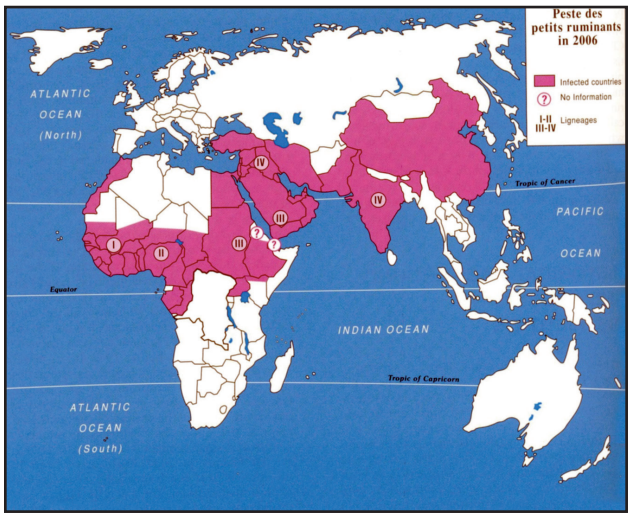

Fig 1. Geographic distribution of peste des petits ruminants with indication of the different virus lineages (Diallo, 2010).

Epidemiology

Transmission of PPR is mainly by oronasal contact with secretions from infected animals, with nearly all outbreaks traced to movement of livestock. The disease is clinically characterised by fever, gastroenteritis, erosive lesions of mucous membranes and respiratory distress caused by bronchopneumonia. Bronchopneumonia is not diagnosed in RP. Mortality can vary considerably and can be as high as 60-70% in small ruminants. However, often animals show only subclinical disease. In some outbreaks in small ruminants only goats and no sheep were involved. In many wild small ruminants the PPR disease can be very mild. Experimental studies conducted in different countries clearly showed that there is a great variation of clinical signs and lesions in experimental animals, infected with the same virus. Hamdy and Dadari (1976) for example infected experimentally white-tailed deer with PPRV. The response varied from fatal consequences to subclinical infection. Al-Naeem *et al* (2009) conducted experimental studies in indigenous Saudi sheep and goats using virulent PPRV isolated from gazelles. Not only in experimental infection trials, this variation occurred, but also in natural outbreaks caused by geography and seasons (Abubaker *et al*, 2009). Although the clinicopathological response was classical in experimental goats and sheep, it was not as severe as it was observed in the naturally infected gazelles. The cause for the great variation in pathogenicity is unknown. In the light of these findings there is a need for experimental infections to elucidate the role of buffaloes, cattle and camels in the epidemiology of PPR.

A new epizootic disease has affected thousands of camels in Ethiopia in 1995 and 1996 characterised by a febrile, highly contagious respiratory syndrome. The morbidity rate reached over 90% with the mortality ranging between 5 and 70%. The major clinical signs were sero-mucopurulent nasal discharge, lacrimation, coughing, dyspnoea and abdominal breathing. Swelling of the submandibular area and diarrhoea was reported in some cases. *Streptococcus equi* spp. *equi* and *M. haemolytica* were also isolated from some of the sick camels. However, their role in the epidemiology of the outbreak was not investigated. The authors suspected that a *Morbillivirus* similar to the RPV or PPRV had been involved in the epizootic disease that affected more or less the entire Ethiopian camel population of 2 million heads within one year. PPR antigens and nucleic acids were detected in some pathological samples collected during that outbreak, but no virus was isolated (Roger *et al*, 1998; Roger *et al*, 2000; Roger *et al*, 2001). The real time (RT)-PCR identified the presence of 2 *Morbillivirus* strains which were named Ethiopia 96 CAMEL 1 and Ethiopia 96 CAMEL 2, and which were closely related to the PPRV. The identified strains belonged to the lineage II and III, respectively. Also sera were analysed for antibodies to RPV and PPRV from dromedaries from affected and non-affected areas using competitive ELISAs. The results showed a seroprevalence of 7.8% for PPRV and 21.3% for RPV from affected areas, but none of the sera from non-affected areas was positive. The authors stated that this epizootic could have resulted from an interspecies transfer of PPRV or RPV strains from cattle, goats and sheep to camels or through an emerging new *Morbillivirus* related to PPRVor RPV.

It is known that cross-reaction exists when antibody cELISAs for PPR and RP are used. Although RP has been declared to be eradicated from the globe, circulating antibodies will continue to be found during the next years especially in vaccinated animals. However, ELISAs for the detection of antigens to PPRV and RPV seem to be designed that there is no cross-reaction. However, cross-reactions between PPR and RP was also reported by Ismail *et al* (1992) when testing Egyptian dromedary sera using the virus neutralisation test VNT with RP Kabete O strain and a PPR virus. Wernery *et al* (2007) used cELISAs from Biological Diagnostics Supplies Ltd. (BDSL), UK for a seroepidemiological survey on 1119 sera from dairy camels. Although outbreaks

of PPR have been seen in small domestic ruminants and gazelles on the Arabian Peninsula regularly, no antibodies to RP and PPR were detected, indicating that PPR most probably does not exist in dromedaries in Arabia.

In a small unpublished experiment, the supernatant (5ml) of a PPRV-positive goat lung (diagnosed with capture antigen ELISA from BDSL) was subcutaneously injected into a 12 year-old castrated camel bull. The virus which was later genotyped as lineage IV did not produce any disease, and the camel bull did not seroconvert. Further serological surveys were carried out to evaluate PPR infection in dromedaries. The serological surveys indicated the susceptibility of the camels to PPRV although clinical signs had not been observed during that time (Abraham *et al*, 2005; Abubaker *et al*, 2008; Albayrak and Gür, 2010; El Amin and Hassan, 1998; Haroum *et al*, 2002; Ismail *et al*, 1992 and Saeed *et al*, 2010). Ismail *et al* (1992) detected with the VNT in 4.2% and 11.9% Egyptian dromedaries antibodies against the PPRV and RPV, respectively.

Recently, important reports have emerged from Saudi Arabia (Abd El-Hakim, 2006) and Sudan (Khalafalla *et al*, 2005 and Khalafalla *et al*, 2010) where PPR outbreaks occurred in dromedaries. Their investigations have clearly shown that dromedaries can contract the disease and play an important role by disseminating the disease to contact goats. However, a different clinical picture was observed in both countries. In Saudi Arabia from 50 dromedaries only 4 animals suffered from fever, nasal discharge and mild cough, whereas in Sudan the disease was characterised by sudden death of healthy dromedaries, bloody diarrhoea and abortions. In both outbreaks, the virus was isolated on Vero cells, MDBK cells, bovine kidney and from the allantoic cavity of embryonated eggs. Molecular typing of the strains revealed lineage IV in Sudan and lineage II and IV in Saudi Arabia.

Clinical signs and pathology

PPR is mainly known to be an acute disease, but often different forms have also been observed which are:

- peracute
- acute
- subacute
- inapparent

In dromedaries, clinico-pathological features can vary widely, and since only 3 outbreaks of PPR in dromedaries have been described so far in Ethiopia, Sudan and Saudi Arabia, it is difficult to make a complete description of the disease and its pathological lesions. However, it seems that this disease affects more adult than young dromedaries. The following clinical signs and pathological/ histopathological lesions have been reported:

- sudden death of healthy animals
- fever
- respiratory distress, heavy abdominal breathing and nasal discharge, cough
- yellowish to bloody diarrhoea
- abortion
- peribronchial infiltration of mononuclear cells, degeneration of the epithelium of the bronchioles
- alveolar septae are congested and infiltrated by mononuclear cells
- lung oedema and emphysema
- lymph nodes show atrophic lymphoid follicles
- degeneration and eroded alimentary tract epithelium

New research on the 7 *Morbilliviruses*; dolphin morbillivirus (DMV), porpoise morbillivirus (PMV), measles virus (MV), rinderpest virus (RPV), peste des petits ruminants virus (PPRV), phocine distemper virus (PDV) and canine distemper virus (CDV) have shown that some of them induce demyelinating disease in infected animals (Sips *et al*, 2007). A possible involvement of a *Morbillivirus* in the pathogenesis of multiple sclerosis in humans is currently discussed between scientists.

Diagnosis

PPR can be confused with many other diseases in dromedaries as the clinical picture can vary. Confirmation of the PPR diagnosis can only be achieved by proper laboratory investigations. In live animals clotted and unclotted blood in EDTA as well as swabs from nasal and ocular discharge and faecal swabs should be dispatched to the laboratory. Sections of lung, lymph nodes, spleen and gut mucosa must be collected ideally from 3 to 4 animals, and dispatched to the laboratory in refrigerated conditions.

AGID, counter immune-electrophoresis (CIEP), immunocapture ELISA and PCR have been widely used to test samples containing RPV or PPRV antigens. The capture ELISA is a very sensitive and easy to perform test which is based on the use of a

monoclonal antibody. No cross reactivity seems to exist between RP and PPR antigen ELISA. The gold standard test for PPR diagnosis is virus isolation. However, very often the virus does not grow despite many passages and the use of different cell lines including primary lamb testis monolayers (Kinne *et al*, 2010). Therefore, some scientists use different isolation techniques like lymphoblast cultures in a homologous system in roller bottles incubated at least for 14 days.

Immunohistochemistry is an alternative and rapid method for diagnosing a *Morbillivirus* infection. *Morbillivirus* antigen might be found in the cytoplasm and nucleus of intestinal epithelial cells, bronchial and bronchiolar epithelial cells, syncytial cells, bile duct epithelia, hepatocytes, and cells of intestinal lymphoid follicles (Kinne *et al*, 2010).

Finally, PPRV infections can also be identified through serology using either the VNT or antibody cELISA. The advantage of the cELISA is that the conjugate is directed against the protein of the virus and can therefore be used for any animal species. Competitive ELISAs have been recommended by the OIE as alternative test for international trade. It should be kept in mind that cELISA cannot differentiate between vaccinated and non-vaccinated animals, and often during an outbreak, sera which were positive in the PPR cELISA were also positive in RP cELISA indicating a cross reactivity between the 2 tests.

Control and treatment

In PPR outbreaks animal movements have to be strictly controlled in the affected area, and camels should not intermingle with small ruminants, which does not occur in the UAE. This may be reason why so far no outbreaks or no serological reactors of PPR have been observed. PPR is controlled by vaccination. Before the PPR vaccine was developed in 1989, the attenuated RP vaccine was used to protect animals against PPR. The attenuated PPR vaccine provides lifelong immunity in vaccinated animals, and as the RP vaccine has now been discontinued worldwide, the attenuated PPR vaccine is the principle vaccine used to protect small ruminants against PPR. The efficacy has recently been dramatically improved through an ultra rapid dehydration process with trehalose and the insertion of PPR genes into the capripox virus genome. The dual thermostable recombinant capripoxvirus vaccine protects small ruminants against 2 economically important diseases (Berhe *et al*, 2003). The vaccine has not yet been used in dromedaries.

Own investigation

Three dromedaries were inoculated with a goat PPR virus strain received from CIRAD, France. This PPR strain was isolated in Ethiopia in 1994. The virus was grown on Vero cells, and 2 ml of the virus from a 5th cell culture passage with a titre of 1.0×10^4 $TCID_{50}$/0.1 ml were inoculated subcutaneously into one adult 10-year-old 8 – month pregnant dromedary, and intravenously into 2 female 14-year-old dromedaries at CVRL. Three weeks earlier, 5 dromedaries kept on the same premises as the infected dromedaries were vaccinated twice with a Jordanian PPR vaccine derived from a sheep strain (Pestevac, Jovac, Jordan). This PPR vaccine is an attenuated vaccine, and 1 ml was administered subcutaneously to protect dromedaries from PPR in case the infected dromedaries would shed the virus. Blood samples were regularly taken as well as rectal temperatures on a daily basis for 3 weeks.

Only one of the vaccinated camels produced antibodies to the PPR vaccine virus using a competetive antibody ELISA (Biological Diagnostics Supplies, BDSL, UA), and 2 of the infected dromedaries. The 3 infected dromedaries remained healthy throughout the trial. No clinical sign, no increase of body temperature and no alteration in haematology were observed. The pregnant camel did not abort and all EDTA blood samples were negative in the PCR.

It seems that a goat PPR virus does not produce a disease in dromedaries, and that an attenuated sheep PPR vaccine does not induce seroconversion. New experiments are necessary to elucidate if the PPR virus isolated from dromedaries in Sudan and Saudi Arabia may cause disease in camels. However, it should be kept in mind that a great variation in pathogenicity exists even with one PPR virus strain in the same animal species.

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