

## Peste des petits ruminants, growing incidence worldwide

Like rinderpest, peste des petits ruminants (PPR) is caused by a virus of the *Morbillivirus* genus but, unlike rinderpest, which has now been eradicated, PPR remains one of the most serious viral infections of sheep and goats and its incidence is growing.

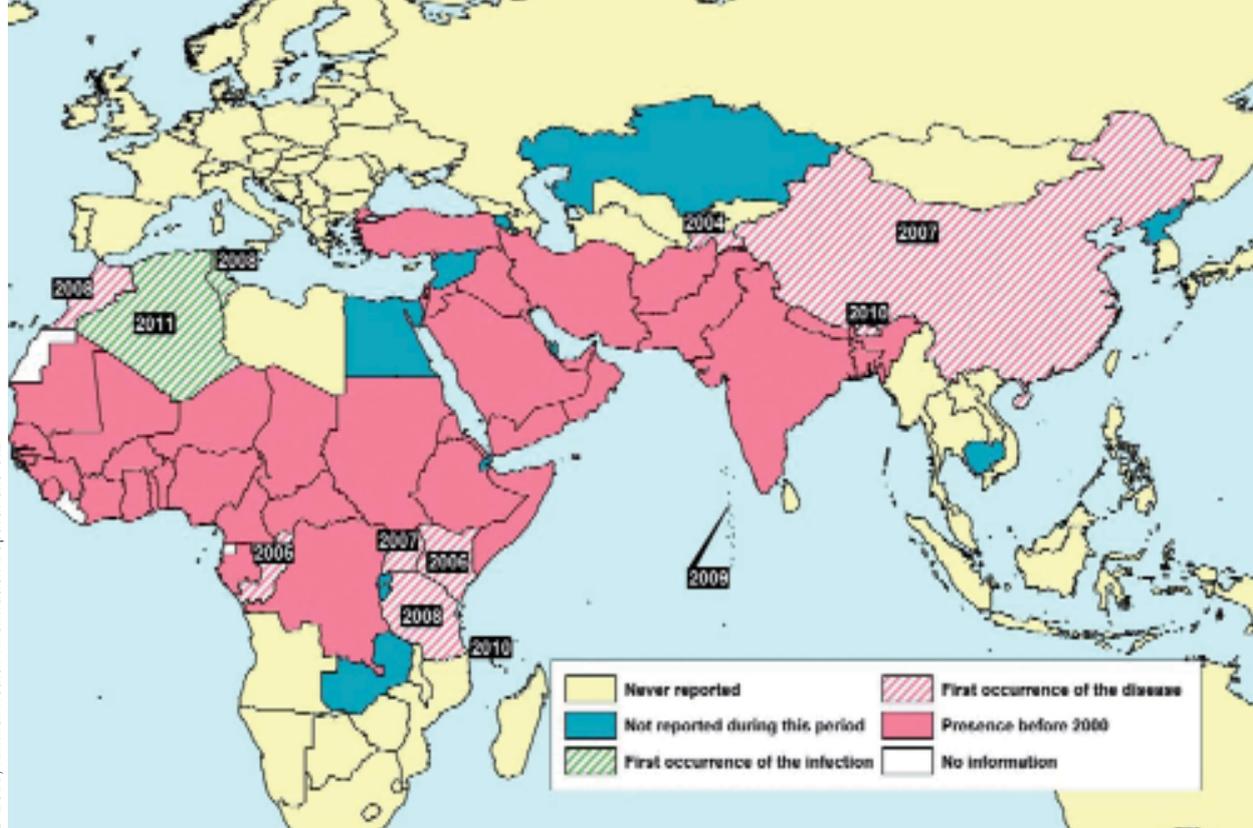
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PPR is spreading throughout Africa south of the Sahara and north of the equator, as well as Asia and the Middle East. It is a major factor of food insecurity for populations reliant on the production of small ruminants. Indeed, sheep and goats play an important role in the rural economy because they can be raised in a range of different production systems, even the most difficult ones. In spite of the huge socioeconomic impact of PPR, the lack of interest paid to the disease since its discovery is largely responsible for its spread. However, the recent changes observed in the geographical distribution of PPR, bringing it close to Europe, as well as in terms of host susceptibility, call for more attention to be paid to the disease.

In its acute form, PPR can cause herd mortality of between 80% and 100%. The disease is characterised by a rapid rise in body temperature, lacrimation, nasal discharge and an erosion of mucous membranes (Fig. 1: insert the two photos). In lethal infections, death occurs from bronchopneumonia, diarrhoea and severe dehydration. Although the virus is highly contagious, it can only be transmitted when a healthy animal comes into direct contact with the secretions or excretions of a sick animal. The clinical signs are often confused and exacerbated by secondary bacterial infections that make PPR difficult to diagnose and treat.



**Fig. 1**  
Purulent nasal discharge in a goat suffering from peste des petits ruminants  
(Photos from Abdallah Traoré, Central Veterinary Laboratory, Bamako, Mali)



Peste des petits ruminants evolution between 2000 and early 2011

### Recent data

Described for the first time in Côte d'Ivoire (Gargadennec and Lalanne, 1942), PPR was long considered to be confined to West Africa but later it was described throughout Africa south of the Sahara and north of the equator, as well as the Middle East and Asia. Recent field and laboratory data show that PPR is spreading, with recent incursions reported into China and Bhutan, and that it is moving fast towards southern and eastern Africa where it affects a wide belt of countries south of the equator, from Gabon to Somalia. In northern Africa, the PPR epizootic that occurred in Morocco in 2008 has extended the disease's geographical distribution to the Mediterranean. This was the first episode of PPR to be reported in a Maghreb country.

The global spread of PPR is probably related to the progressive control and later, eradication, of rinderpest. The cessation of rinderpest vaccination campaigns and loss of antibody cross-protection between the two diseases means that small ruminants are now fully exposed to PPR. Its spread has certainly also been encouraged by the growing population of small ruminants, with the virus colonising new areas as a result of animal movements during seasonal transhumance, people fleeing from socio-politically and climatically insecure areas and the intensification of trade associated with human population growth. These cross-border movements have a very significant impact on the spread of many infectious agents and pose increasing problems for the surveillance and control of animal, zoonotic and human diseases (Domenech *et al.*, 2006).

There is now a resurgence of PPR in some areas, with a parallel incursion of new genotypes. Four phylogenetic lineages have been

## References

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identified, three of which are established in Africa (lineages I to III) and lineage IV, in Asia (Shaila *et al.*, 1996; Kwiatek *et al.*, 2007).

The recent emergence of PPR is overturning knowledge about the geographical distribution of lineages and has led Reference Laboratories to update the distribution of current genotypes based on virus data from epidemiological surveillance surveys. Molecular typing has revealed that Asian lineage IV has become established in an area of Sudan where PPR has re-emerged, edging out the indigenous African lineage. Similarly, the introduction of PPR into Morocco in 2008, which was hitherto free from the disease, also involved lineage IV strains (Banyard *et al.*, 2010).

Changes in the allopatric speciation of lineages suggest that, when competing with indigenous strains, some strains have great power to spread because they are better adapted to the natural host and/or by switching to a new host. It is important to emphasise the increasing frequency of camel involvement in PPR. The camel is suspected of being a risk factor in the long-distance transmission of transboundary viral diseases causing major infections in other species. It has now been shown that camels are susceptible to the PPR virus (Roger *et al.*, 2001) and that the clinical expression of the disease is emerging in this species (Khalafalla *et al.*, 2010). Recent observations in Sudan suggest that camels could be victim to PPR, as well as acting as long-distance vectors (Kwiatek *et al.*, 2011). However, the scale of this phenomenon needs to be evaluated, especially from an epidemiological standpoint, by comparing long-distance movements of camels with the phylogeographic distribution of PPR virus strains.

## Conclusion

The eradication of rinderpest suggests that a global control strategy could now be adopted for PPR. The disease's incidence is growing worldwide and continues to undermine the activities of the poorest producers. The possible transmission of PPR between different species – small ruminants and camels – compounds the already significant health constraints on livestock. However, the role of the different species involved in PPR epidemiology should be elucidated and the consequences of control strategies, including partial or full vaccination of susceptible species, should be tested. One possibility might be to assess the need to vaccinate camels during PPR vaccination campaigns. The existence of an effective vaccine against PPR, as well as sensitive and specific diagnostic tools, mean that strategies for controlling, and in the longer term eradicating, the disease could be envisaged.