

## SCIENTIFIC OPINION

### Scientific Opinion on peste des petits ruminants<sup>1</sup>

#### EFSA Panel on Animal Health and Welfare (AHAW)<sup>2,3</sup>

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#### ABSTRACT

Peste des petits ruminants (PPR) is a severe viral disease of small ruminants caused by a *Morbillivirus* closely related to rinderpest virus. It is widespread in Africa and Asia and is currently also found in Turkey and Northern Africa. PPR is transmitted via direct contact, and the disease would mainly be transferred to infection-free areas by transport of infected animals. In the EU, it could only happen through illegal transport of animals. The risk of that depends on the prevalence in the country of origin and the number of animals illegally moved. The extent of the spread would depend mainly on the time during which it is undetected, the farm density, the frequency and distance of travel of animals. PPR has a high within-herd transmission rate, therefore contacts between flocks, e.g. through common grazing areas, should be avoided when PPR is present. If PPR enters EU areas with dense sheep population but low goat density, it may spread rapidly undetected, since goats are considered more susceptible than sheep. Effective measures in limiting the spread of PPR in the EU include prompt culling of infected herds, rapid detection, movement restriction, and disinfection. Live attenuated vaccines against PPR are available, safe and effective, and have been successfully used to control PPR epidemics, but no method exists for differentiating between infected and vaccinated animals; therefore, the development of one is recommended. Awareness-raising campaigns for farmers and veterinary staff to promote recognition of the disease should be considered. The cooperation of the EU with neighbouring countries should be encouraged to prevent the spread of PPR and other transboundary diseases.

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#### KEY WORDS

Peste des petits ruminants, spread, prevention, control, surveillance, vaccines

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## SUMMARY

Following a request from the European Commission, the EFSA Panel on Animal Health and Welfare (AHAW Panel) was asked to deliver a scientific opinion on peste des petits ruminants (PPR), in order to provide an update on the characterisation of the disease; to assess the risk of introduction into the European Union (EU) and the speed of spread, the risk of becoming endemic and its impact; and to determine if further measures are justified. This request is linked to PPR being currently reported in Turkey and several other Northern African countries. If the virus were to enter the EU, it could cause severe direct losses related to important mortality rates in naive populations.

In particular, EFSA was asked to (i) characterise the disease and provide an update on the global occurrence of PPR and changes in the distribution during the last 10 years; (ii) map the region of concern and other countries of the Mediterranean Basin and Black Sea, displaying identified, or likely, major live animal trade routes; (iii) evaluate all possible pathways of introduction of PPR into the EU, ranking them on the basis of their level of risk, with a view to enhancing preparedness and prevention; (iv) assess the risk of introduction and speed of propagation of PPR into the EU and neighbouring countries; (v) assess the risk of PPR becoming endemic in animal populations in the EU; (vi) assess the impact PPR would have if it were to enter the EU, considering different scenarios as regards the effectiveness of surveillance and control measures; and (vii) review the feasibility, availability and effectiveness of the main disease prevention and control measures (diagnostic tools, biosecurity measures, restrictions on movement, culling).

Regarding disease characterisation, the AHAW Panel reported that PPR is a severe viral disease of small ruminants caused by a *Morbillivirus* closely related to rinderpest virus. It is widespread in Africa, the Middle East and Southern Asia. It is one of the priority animal diseases whose control is considered important for poverty alleviation in those regions. PPR causes severe disease in its acute form, with fever, respiratory symptoms, congestion and necrosis of mucous membranes, diarrhoea, abortion and immunosuppression. The mortality can range between 10 and 90 %, depending on host status, and if animals recover there is no persistent infection or carrier state. PPR virus (PPRV) resistance is extrapolated from its similarity with rinderpest virus. The virus is considered sensitive to heat, ultraviolet light and pH lower than 5.5 and higher than 10, and it does not survive in the environment, unless in shaded conditions, where it can survive for up to 72 hours. In fresh and chilled meat, it may survive for a few days. PPR transmission is usually via contact with infected animals, or with their fresh secretions or faeces. The virus is found in all kinds of secretions from approximately 3 to 22 days post infection. Goats are considered more susceptible than sheep, and in the latter PPR may circulate undetected for some time. Cattle and pigs can be infected with the virus and then develop specific antibodies, but show no clinical signs. Camels and several wild ruminants can be infected and show clinical disease, although their role in the epidemiology of PPR, in particular in further eliminating the virus, needs to be clarified.

Considering the identified or probable animal movements in the regions of concern, the movements of small ruminants related to trade (both legal and illegal) are the most likely cause of the spread of PPR across borders, as movements often occur between East Africa and the Arabian Peninsula, where the sudden and large increase in livestock movements owing to religious festivity can negatively affect the containment of the disease. The movement of live animals from third countries in the Mediterranean Basin and Black Sea areas into the EU is currently forbidden, according to EU animal health legislation on the import of live animals from countries where PPR is endemic. However, illegal movements of animals cannot be quantified. The illegal movement of animal products, including meat products, carried by tourists and visitors from countries that are at high risk of PPR and communicable animal diseases is large and underestimated.

Regarding the main possible pathways for PPR introduction into free areas, the introduction of infected animals is largely the most efficient pathway to introduce PPR into a country. In the EU, this could occur by the illegal transport of infected animals. In addition, the introduction of PPRV into the EU through infected animal products may occur, in particular when illegally or intentionally carried to

spread the virus (e.g. bioterrorism), although the risk of this is low and the further spread of PPR via this route is unlikely. Of less importance is the introduction of PPRV via fomites into the EU, which is considered to be unlikely. This could occur when vehicles carrying livestock return to the EU after the delivery of animals in infected areas or farms and where no biosecurity measures are applied.

In order to estimate the risk of introduction of PPR into the EU via the illegal movement of animals, a model was used to assess the probability of an individual being infectious in a given shipment size. For example, for a level of seroprevalence in the country of origin equal to 37 %, the number of animals that would need to be moved to have a probability of introduction greater than 0.95 or lower than 0.05 to introduce PPR into Europe would be 421 and 8, respectively. On the other hand, if the seroprevalence is 8 %, the number of animals that would need to be moved to have a probability of introduction greater than 0.95 or lower than 0.05 to introduce PPR into Europe would be 1 952 and 34, respectively.

In order to derive an estimate of the potential ranges of speed of propagation, outbreak data of PPR in Tunisia as reported to the World Organisation for Animal Health (OIE) were used to plot temporal and spatial linkages between outbreaks. According to this, the median speed of propagation was estimated to be 3.9 km/day, with a 95 % confidence interval of 0.3 to 65.5 km/day. Nevertheless, this result should be interpreted with caution without direct extrapolation to the potential epidemiological behaviour of PPR if it entered the EU. Because the control measures applied in the EU would aim at culling infected flocks and restricting movements, the spread of PPR in European situations would depend on farm density, travelling frequency and distance of small ruminants, and the duration of silent spread (high-risk period). In such conditions, long-distance transmissions would be more important for initiating new epidemics and thus for the spatial spread of the infection. The basic reproductive ratio calculated with data from a case study in Senegal and compared with other studies in Tanzania and Pakistan showed that PPR has a high within-flock transmission rate, and an outbreak would lead to infection of most animals in a herd. The between-herd transmission would be very variable in Europe, and common grazing grounds would be a major risk source and should be avoided when PPR is detected in the neighbourhood.

Regarding the risk of PPR becoming endemic in animal populations in the EU, although PPR is endemic in several countries neighbouring or close to the EU, owing to a lack of data regarding PPR transmission in the EU, the international data cannot be extrapolated directly to the European situation to make a quantitative assessment of the risk of endemicity. Given the control measures foreseen by the current EU policy, PPR would most likely not become endemic in the EU.

When assessing the impact and consequences of PPR entering the EU, from worldwide experience in endemic areas it can be assumed that, in the EU, goats would be more susceptible than sheep. Therefore, there might be a risk that, if PPR enters EU areas with dense sheep populations but low goat densities, such as Great Britain or Ireland, it would start circulating without being promptly detected, and would lead to widespread infection.

The main prevention and control measures for PPR have been assessed. In general, the AHAW Panel concluded that, as clinical signs of PPR are not disease specific and clinical diagnosis is not reliable, PPR should be confirmed by laboratory testing. The most common and reliable laboratory techniques for PPRV detection are polymerase chain reaction (PCR) and immunocapture enzyme-linked immunosorbent assay (ELISA). The latter is of choice where molecular techniques are not available or biological samples are poorly preserved, although this method is not as sensitive as PCR. Among serological tests, the one that is most used is competitive ELISA.

As far as vaccines for PPR are concerned, only live, attenuated vaccines are available, with high safety and efficacy, protecting against all known isolates of PPRV, but not supporting the differentiating infected from vaccinated animals (DIVA) principle. Possible DIVA vaccines based on recombinant techniques have been shown to be efficacious but are still at the experimental stage. Inactivated

vaccines are not available and, owing to the immunological response to PPRV, would not be fully effective.

The lessons learnt from the PPR epidemics in Morocco are that PPR can be controlled in areas, such as Northern Africa, through mass vaccination campaigns implemented at the national level, provided that adequate means are available and correctly implemented. However, in endemic areas, assiduous vigilance is needed because there is a risk of PPR reoccurrence, especially with risk factors of continuous introduction such as the illegal cross-border movements of livestock. In general, early detection of (re)occurrence is a necessary condition for rapid response and the effective management of possible outbreaks of PPR.

Owing to several knowledge gaps about PPR and PPRV, the AHAW Panel recommends further investigation on (i) virus survival and infectiousness in different matrices (e.g. meat, milk) and under different environmental conditions (e.g. temperature, pH, humidity); (ii) the virulence of different virus isolates, the capacity of virus excretion and infectiousness in the same host animals, or of a single isolate in different host species, including European goat, sheep besides cattle, camels and pig breeds, to PPR; (iii) the collection and analysis of data on the transmission and spread within and between herds in different situations, including in a situation comparable to the EU; (iv) the knowledge on populations, movements and contact patterns of small ruminants; (v) the impact of socio-economic factors on the efficacy and efficiency of vaccination campaigns (e.g. vaccine delivery systems) in countries where PPR is endemic; and (vi) the development of safe, efficient and non-replicating DIVA vaccines against PPRV, as well as an associated diagnostic test.

In term of preparedness, the AHAW Panel recommends designing and implementing a regional PPR control strategy, especially in endemic countries or where PPR has occurred and been controlled (e.g. Morocco), relying on coordinated mass vaccination, and post-vaccination monitoring and efficient active surveillance measures.

Better knowledge of legal and illegal livestock and animal product movements should be sought, especially in areas at risk of or affected by PPR. Furthermore, adequate veterinary care and improved surveillance should be in place, in particular for transhumant flocks along the migratory routes in risk areas and especially for long-distance migrations. Awareness-raising campaigns and training for farmers and veterinary staff in recognising the disease under field conditions should be considered, especially for regions at higher risk of introduction of PPR (i.e. those bordering affected regions). Finally, if non-biological drivers of transmission of transboundary animal diseases will change (e.g. breakdown of veterinary infrastructures, human migration, political unrest), the risk of PPR introduction should be accordingly reassessed. Under this perspective, the cooperation of the EU with endemic countries should be encouraged for the prevention of introduction of PPR and other transboundary animal diseases and to enhance preparedness.

In terms of control, if PPR entered the EU, rapid detection, movement restriction, prompt culling of infected herds and disinfection measures should be considered effective measures in limiting the spread and impact of the outbreaks.

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## BACKGROUND AS PROVIDED BY THE EUROPEAN COMMISSION

Peste des petits ruminants (PPR) is an acute contagious disease caused by a *Morbillivirus* in the family *Paramyxoviridae*. It affects mainly sheep and goats and occasionally wild small ruminants. It has been reported on a few occasions in camels, cattle and buffaloes. PPR represents one of the most economically important animal diseases in areas that rely on small ruminants.

PPR is exotic to the EU. It occurs in Africa except Southern Africa, in the Arabian Peninsula, throughout most of the Near East and Middle East, and in Central and South-East Asia. The disease is currently being reported in Turkey and several other Northern African countries. If the virus were to enter the EU it could have severe direct losses related to important mortality rates in naïve populations. The consequential losses related to trade restrictions could be even more important.

PPR is a disease included within the category of sheep and goat diseases on the OIE list of diseases in Article 1.2.3 of the Terrestrial Animal Health Code (the Code) of the World Organisation for Animal Health (OIE). This consequently entails notification obligations to the OIE for the EU Member States and its trading partners. Specific international trade standards for PPR are provided for in Chapter 14.8 of the Code as well as in Chapter 2.7.11 of the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.

There are several legislative acts in the EU that pertain to PPR, of which the most relevant ones are:

Council Directive 82/894/EEC of 21 December 1982 on the notification of animal diseases within the Community sets an obligation for Member States to notify the Commission of the confirmation of any outbreak of PPR.

Council Directive 90/425/EEC of 26 June 1990 concerning veterinary and zootechnical checks applicable in intra-Community trade in certain live animals and products with a view to the completion of the internal market recognises PPR as a disease subject to mandatory emergency action, including territorial restrictions.

Council Directive 92/65/EEC of 13 July 1992 laying down animal health requirements governing trade in and imports into the Community of animals, semen, ova and embryos identifies PPR as a notifiable disease and requires that trade in *Bovidae*, including small ruminants, and their products be subject to specific health requirements.

Council Directive 92/119/EEC of 17 December 1992 introducing general Community measures for the control of certain animal diseases foresees control and eradication measures for certain diseases exotic to the EU, including PPR.

Council Directive 2002/99/EC of 16 December 2002 laying down the animal health rules governing the production, processing, distribution and introduction of products of animal origin for human consumption addresses all stages of the production, processing and distribution within the Union and the introduction from third countries of products of animal origin intended for human consumption. Specific inactivation protocols are provided for PPR virus.

The risk manager is in need of updated scientific advice in order to assess the risk of introduction of PPR and to determine if further measures are justified. This is linked to the absence of the disease from the EU while it appears to spread in some neighbouring countries.

Another important aspect is related to the characterisation of the disease in order to assist the risk manager in any future categorisation exercise carried out in the framework of the prioritisation of actions.

Therefore, the Commission is in need of scientific advice on the assessment of the significance of the risk posed by PPR considering the current control measures.

## TERMS OF REFERENCE AS PROVIDED BY THE EUROPEAN COMMISSION

In view of the above, and in accordance with Article 29 of Regulation (EC) No 178/2002, the Commission asks EFSA for a scientific opinion on the following aspects of PPR:

1. Characterise the disease and provide an update on the global occurrence of PPR and changes in the distribution during the last 10 years.
2. Provide a mapping of the regions of concern and other countries of the Mediterranean Basin and Black sea, displaying identified, or likely, major live animal trade routes.
3. Evaluate all possible pathways of introduction of PPR into the EU, ranking them on the basis of their level of risk, with a view to enhance preparedness and prevention.
4. Assess the risk of introduction into the EU and of the speed of propagation of PPR if it had affected certain Member States.
5. Assess the risk of PPR becoming endemic in the animal population in the EU.
6. Assess the impact of PPR if it were to enter the EU considering different scenarios as regards the effectiveness of surveillance and control measures.
7. Briefly review the feasibility, availability and effectiveness of the main disease prevention and control measures (diagnostic tools, biosecurity measures, restrictions on the movement, culling).



## ASSESSMENT

### INTRODUCTION

Peste des petits ruminants (PPR) is an acute infectious disease caused by PPR virus (PPRV), a *Morbillivirus* in the family *Paramyxoviridae*. PPR affects mainly sheep and goats and occasionally wild small ruminants, and represents an economically important disease of sheep, which is of great concern for animal health and welfare. PPR is currently exotic to the European Union (EU), but incursions of PPR have occurred in neighbouring areas of the EU. Recent outbreaks of PPR in Northern Africa, the Middle East and Turkey have stressed the need to update the disease profile, specifically focusing on surveillance and control measures, as well as the need to assess the risk of spread to the EU and for it to become endemic in affected Member States (MSs).

### APPROACH TAKEN TO ANSWER TERMS OF REFERENCE

This scientific opinion was prepared by an ad hoc working group, and reviewed and adopted by the Animal Health and Animal Welfare (AHAW) Panel of EFSA, to answer the terms of reference (ToRs) as provided by the European Commission (EC). The following approach was followed to answer risk questions:

- In order to update the disease profile and the global occurrence of PPR (**ToR 1**), a literature review was conducted to retrieve recent scientific information on aetiology, pathogenesis, clinical signs and lesions, and epidemiology. This information is contained in section 1. The global occurrence of PPR and changes in its distribution during the last 15 years were analysed through data reported to the World Organisation for Animal Health (OIE). For the EU neighbouring regions, maps were produced showing the occurrence of the infection in different years (2005, 2010 and 2013; see section 1.2) and the number of years of presence. Case studies were described on Northern Africa, Turkey and China (section 2).
- The identification and display of probable major live animal trade routes (**ToR 2**) was based on trade and animal movement data from TRACES, COMTRADE and Eurostat, providing information of trade routes from third countries of the Mediterranean Basin and Black Sea and describing drivers for uncontrolled animal movements and socio-political drivers (section 3).
- To identify possible pathways of introduction of PPR into the EU (**ToR 3**), information from the literature and about recent outbreaks occurring in the north of Africa and in Turkey was reviewed with the aim of understanding the complex events that could lead to transboundary transmission into the EU (section 4). The identified pathways were ranked by importance using this information provided by experts (section 4.2).
- The risk of introduction (section 5) and speed of propagation (section 6) of PPR into the EU and neighbouring countries (**ToR 4**) was assessed by using data from outbreaks in Tunisia and Senegal to derive an estimate of the potential ranges of speed of propagation. Data from these countries were chosen because no vaccination was applied in them, thus PPR transmission was in unvaccinated herds as it would be in the EU.
- The risk of PPR becoming endemic in animal populations in the EU and neighbouring countries (**ToR 5**) was assessed qualitatively by a review of data on viral physicochemical and biological properties, stability in the environment and the ability of the virus to persist in animals and herds.
- The impact and the consequences of PPR (**ToR 6**) were assessed for both endemic and disease-free areas. Information regarding direct and indirect losses was collected from areas recently affected by the disease (see section 8).
- The feasibility, availability and effectiveness of the main disease prevention and control measures (**ToR 7**) were reviewed using available information from the scientific literature and expert opinion (see section 9).

## 1. Disease characterisation

Peste des petits ruminants (PPR) is a systemic infectious viral disease of small ruminants caused by a *Morbillivirus* (*Paramyxoviridae*). The disease is endemic in many countries of Africa, the Middle East and Asia, and it is of great concern for animal health and welfare. PPR is an acute disease characterised by fever, anorexia, ocular and nasal discharge, erosions and ulcers in digestive mucosa, diarrhoea and marked leucopenia with immunosuppression. Pregnant animals may abort. Affected animals may die from direct effects of the infection or from increased susceptibility to secondary infection by other pathogens. Case-fatality rates vary enormously (anywhere from 10 to 90 %) depending on the species infected, age, and prevalence of secondary infectious agents.

### 1.1. Agent characteristics

#### 1.1.1. Phylogenetics

PPR virus (PPRV) is a member of the genus *Morbillivirus*, closely related to rinderpest virus (RPV), a highly infectious cattle pathogen now eradicated worldwide. Other members of the genus include measles virus (MV) in humans; canine distemper virus (CDV), affecting dogs and many other carnivore species with a broad range of hosts; phocine distemper virus (PDV), which infects pinnipeds; and cetacean morbillivirus (CeMV), affecting several cetacean species. The morbilliviruses form one genus within the sub-family *Paramyxovirinae* of the family *Paramyxoviridae*; as with all the paramyxoviruses, the morbilliviruses have a single-segmented RNA genome of negative sense. PPRV has not been extensively studied in terms of its structure and physical characteristics, and most of our picture of PPRV comes from studies on RPV or MV.

The genome length of PPRV is 15 948 nucleotides (Bailey et al., 2005). There are six genes, or transcription units, with promoter sequences (that is to say, binding sites for the viral RNA-dependent RNA polymerase (RdRP)) at only the 3' ends of the genome and antigenome. The viral genes encode the nucleocapsid (N) protein, the phosphoprotein (P), the matrix protein (M), the fusion (F) and haemagglutinin (H) membrane glycoproteins and the large protein (L), which is the viral polymerase. The P gene also encodes the three accessory proteins V, W and C, which are sometimes referred to as non-structural, although it is not conclusively proven that they are not part of the virion. The accessory proteins are not required for the replication and assembly of the virus (Schneider et al., 1997; Baron and Barrett, 2000). The P and L proteins together form the functional viral RNA polymerase. The F and H proteins are found in the viral envelope, projecting to the outside of the virion and the infected cell. The H protein is responsible for binding to the host cell receptor, whereas the F protein allows the viral envelope to fuse with the host cell envelope, transferring the nucleocapsid to the host cell cytoplasm. The M protein plays an as-yet not understood role in virus budding and structure. A detailed description of the molecular biology of the virus can be found in Baron (2011).

All PPRV strains belong to a single serotype, but the different strains have been grouped into four distinct lineages, with lineages I and II occurring in West Africa, lineage III in East Africa, the Middle East and southern India, and lineage IV in Asia (Shaila et al., 1996; Dhar et al., 2002; Kwiatek et al., 2010). These lineages are based on sequence differences on a short (approximately 300 bases) specific section of the viral F gene or N gene. The utility of lineage identification lies in the information it provides on the probable origin of the virus causing a new outbreak. PPRV lineage identification proved that the outbreak in Morocco in 2008—the first time the virus had been seen in North Africa—was a lineage IV virus, and therefore had not come from West Africa, where all the viruses are lineage I and II, but had come from the Middle East or Turkey, where lineage IV PPRV strains circulate. PPRV lineage IV has been found in recent years in sub-Saharan Africa, and appears to be displacing viruses of the so-called “African” lineages (lineages I–III).

#### 1.1.2. Virulence

Field observations in different countries, with different host animals at different times, have suggested that the virulence of virus strains causing specific outbreaks can vary. However, there have been

almost no studies comparing the virulence of different virus isolates in the same host animals, or comparing a single isolate in different host species. Couacy-Hymann et al. (2007) compared five different isolates in the same group of animals, showing mild, severe and very severe disease. Similarly, Baron et al. (2014) compared six different isolates in UK goats, showing either severe or mild disease, although there was a suggestion that the mild isolates were those that had been passaged more in tissue culture. Wernike et al. (2014) compared a single isolate in sheep and goats and confirmed the previous anecdotal suggestions that sheep suffer less severe pathology.

### **1.1.3. Resistance of the virus**

Information about the resistance of PPRV is limited, but it is assumed that the survival characteristics of PPRV are similar to those of RPV. The virions do not survive for any extended period in outdoor environments, being susceptible to heat, ultraviolet light and dehydration. They show stability (at 4 °C) at a pH of 7.2–7.9, with a half-life of 3.7 days, and they can tolerate a pH of between 5 and 10. However, the virus can be expected to last for up to 72 hours in shaded conditions and at the normal range of environmental temperatures. The virus is not rapidly inactivated in fresh meat.

Table 1 summarises information from the literature about survival times of PPRV in different matrices.

**Table 1:** Period of detection of PPR virus (PPRV) in different matrices

Matrix	Period of PPRV detection (days post infection, dpi) <sup>(a)</sup>	Reference	Notes <sup>(a)</sup>	
Live animals and products	Blood	Virus RNA detected 2–21 dpi by RT-PCR <sup>(b)</sup>	(Baron et al., 2014; Truong et al., 2014)	
		Virus RNA detected 2–13 dpi by RT-PCR	Seemed to be up to 15 dpi; may be assay difference	
	Saliva	Virus RNA detected 3–22 dpi by RT-PCR	Liu et al. (2013)	Liu et al. checked samples up to 40 dpi
	Nasal discharge	Virus RNA detected 3–22 dpi by RT-PCR. Live RPV could be isolated from nasal discharge from 2 to 14 dpi	(Liess and Plowright, 1964; Liu et al., 2013)	The viral RNA may still be detected even when the virus is not itself infectious. Antibodies will be present in neutralising amounts from 10–14 dpi onwards, which could explain the difference in assays for viral genome and assays for live virus
	Ocular discharge	Virus RNA detected 3–26 dpi by RT-PCR	(Liu et al., 2013)	
	Urine	No data on PPRV. Live RPV found in urine 3–14 dpi	(Liess and Plowright, 1964)	
	Faeces	Virus antigen found 12 weeks after “recovery”	(Ezeibe et al., 2008)	It could be old infected tissue: no measure of infectious virus
	Semen	No data on PPRV		
Meat	No data on PPRV	(Scott, 1959; De Boer and Barber, 1964; Liess and Plowright, 1964)	Fresh and chilled lamb and sheep meat does not usually go below pH 5.6–5.8 (lower border line value for RPV/PPRV survival), at which RPV, susceptible to pH < 5 and > 10, and probably also PPRV, can still be viable. A test with wild-type PPRV would be helpful, as the virus has a half-life of 2–3 days at 4–8 °C, and is found in tissues of infected animal before clinical signs are observed. Wild-type viruses are more sensitive than cell culture-adapted viruses, with a half-life of 1–4 hours at pH 5 (Liess and Plowright, 1964). Temperature studies in Scott’s paper and that of de Boer and Barber suggested a half-life at chilled temperatures (4–8 °C) of 2–3 days. Scott asserted that meat could be a source of infectious RPV. RPV is considered to remain infective in salted and frozen meat for several months (MacDiarmid and Thompson, 1997), in particular if frozen before pH drop of rigor mortis, although this should be proven for PPRV; moreover, the stability of the	

Matrix	Period of PPRV detection (days post infection, dpi) <sup>(a)</sup>	Reference	Notes <sup>(a)</sup>
			virus to the freeze–thawing process in meat is unknown but is probably limited, as is experienced with freeze–thawing of viruses in cultured cells from –20 °C
Milk	No data on PPRV	(Spinage, 2003)	Half-life for RPV at 60 °C is 3 minutes. Raw milk from RPV-infected animals was thought to be infectious for up to 45 days after recovery, but there are no peer-reviewed studies on the effect of pasteurisation on either RPV or PPRV in milk, which has been noted in a number of risk assessments
Hides	No data on PPRV	(Spinage, 2003)	No recent studies on RPV. Historical records suggest only fresh hides are infectious; records in older studies suggest that salting or heating is sufficient to render the hides uninfected
Wool/hairs	No data on PPRV or RPV		The virus is not associated with skin, so the main problem with wool or hair would be fomites
Feed	No data on PPRV or RPV		No specific data on RPV for feed or fodder—material contaminated with urine or faeces is as infectious as the rest of the shed or pasture
Fodder	No data on PPRV or RPV		See note above
Shaded pens, bedding	No data on PPRV	(Jackson and Cabot, 1987)	See general note on “Environment”. RPV is eliminated from sheds by 72 hours and from pastures by 24 hours, but at Kenya temperatures
Insects	No evidence, even for mechanical transmission of PPRV or RPV. No role as an infected vector		
Environment, pastures	No data on PPRV	(Hyslop, 1979)	This is very difficult to quantify, as the half-life of the virus depends on the pH, temperature, rate of drying (amount of sunlight, wind and relative humidity), rate of putrefaction, etc. The bottom line is that places where infected animals have been kept will be infectious. They will remain that way for some days, unless thoroughly disinfected. RPV is inactivated more rapidly at moderate humidity (50–60 %) than at high or low humidity. The aerosolised virus is rapidly inactivated. At normal humidity (40–60 %), the virus is 99.9 % inactivated by 30 minutes. The virus in urine or faeces may last much longer, depending on pH

Matrix	Period of PPRV detection (days post infection, dpi) <sup>(a)</sup>	Reference	Notes <sup>(a)</sup>
Fomites	Vehicles, clothing equipment	No data on PPRV or RPV	It can be expected to survive for 2–72 hours, depending on heat and humidity and the nature of the original fomite (aerosol decays very fast, faeces much more slowly)

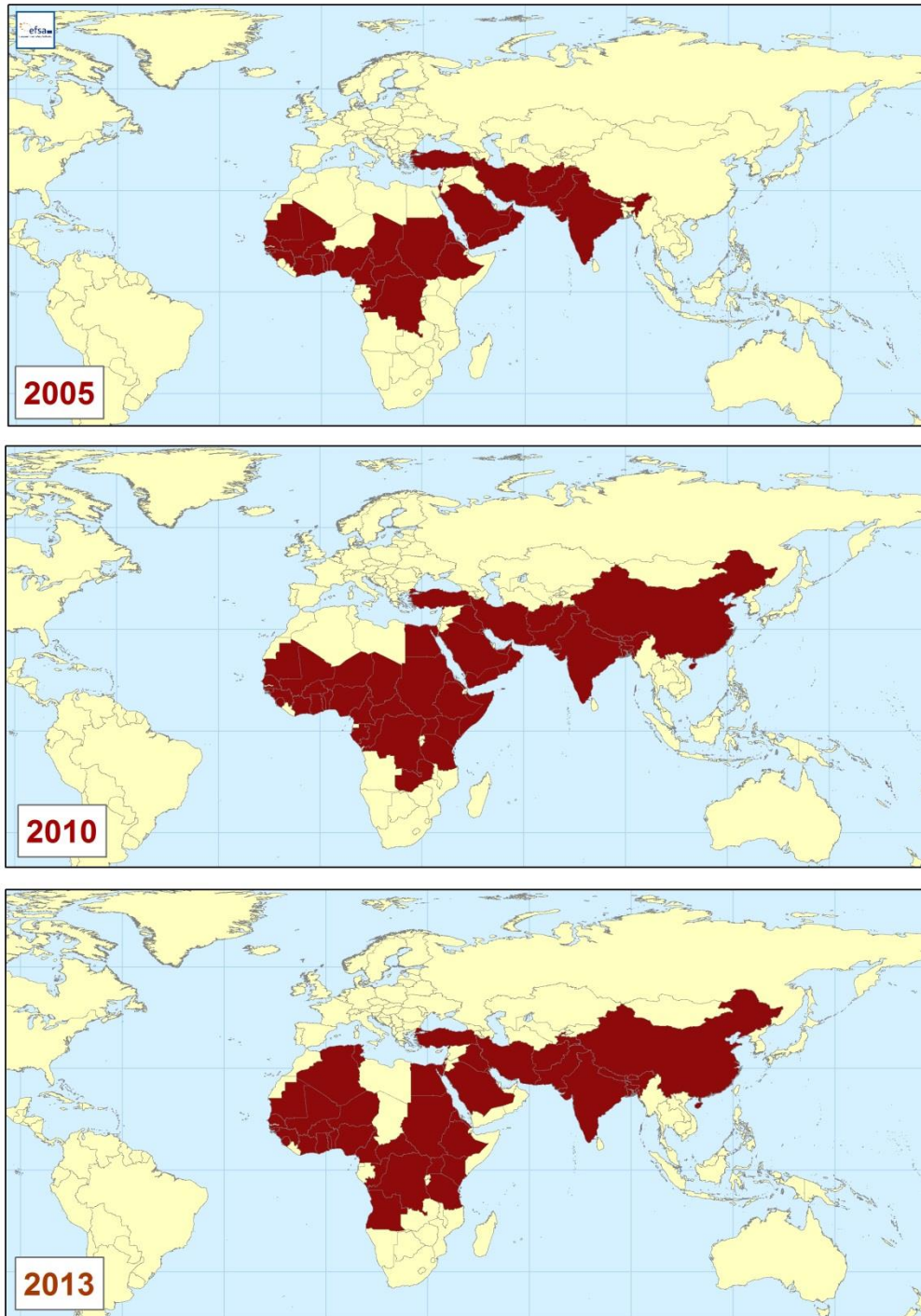
(a): In almost no studies was the infectious virus assayed, but only the viral antigen or RNA was.

(b): RT-PCR, reverse transcription polymerase chain reaction.



## 1.2. Geographical distribution

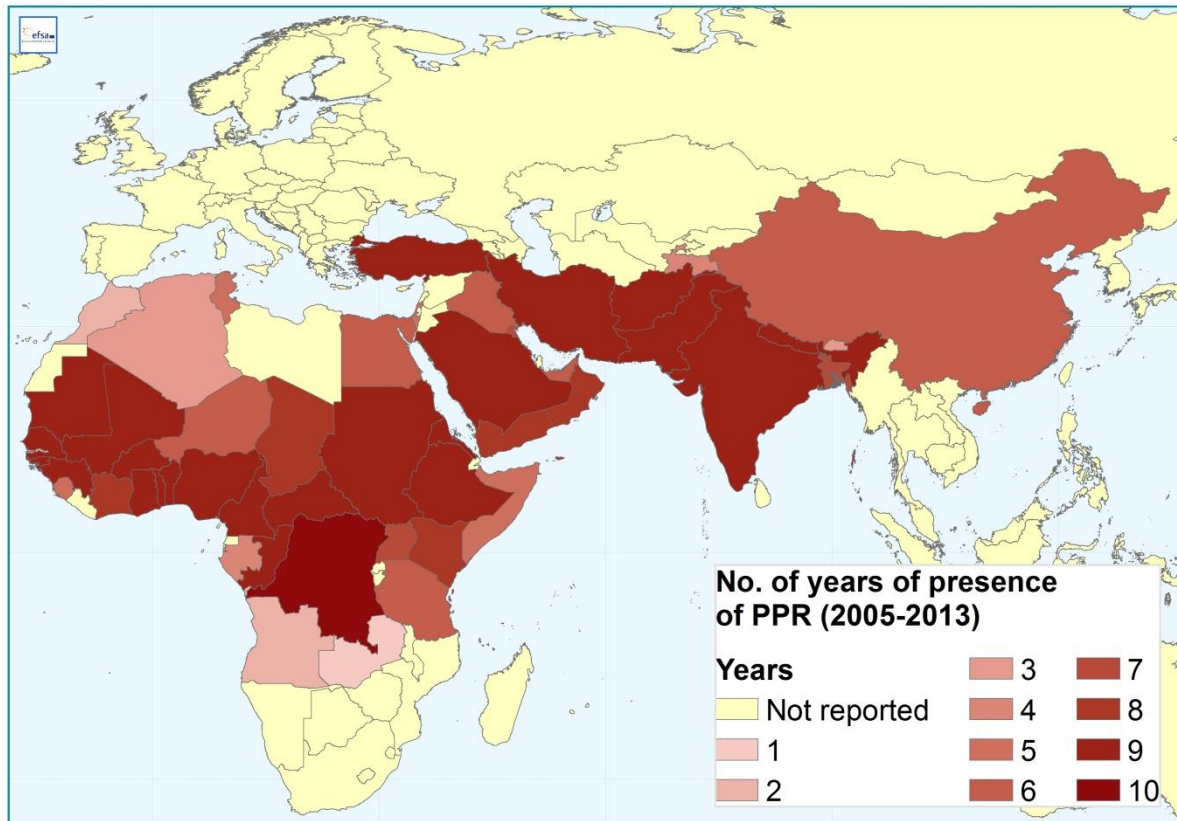
PPR occurs in Africa (except Southern Africa), in the Arabian Peninsula, throughout most of the Near East and Middle East, and in Central and South East Asia. PPR was first described in Côte d'Ivoire, but it occurs in most African countries from North Africa to Tanzania, in nearly all Middle Eastern countries and in Turkey, and is also widespread in countries from Central Asia to South and South East Asia (Banyard et al., 2010). The global occurrence of PPR from 2005 to 2013 and the number of years of presence is reported in the maps below (Figures 1 and 2).



Data source: OIE

**Figure 1:** Global occurrence of PPR as reported to OIE in 2005, 2010 and 2013





Data source: OIE

**Figure 2:** Number of years of presence of PPR in different countries as reported to OIE for the period 2005–2013

### 1.3. Host range

PPR affects small ruminants and it is not a zoonosis. Goats have been reported to be more susceptible than sheep (Nanda et al., 1996), but this has not been confirmed in other outbreaks (Singh et al., 2014). West African goats have been found to be more susceptible than European varieties, and, within the former group, the dwarf varieties were most susceptible to disease (Couacy-Hymann et al., 2007). Cattle and pigs can be infected with the virus, but this is only detected by the presence of specific antibodies, while the animals show no clinical signs (Nawathe and Taylor, 1979; Anderson and McKay, 1994; Lembo et al., 2013). It is not known if these species could spread the virus or have viraemia.

Camels are considered susceptible to PPR but this is still to be clarified by experimental infections. It has been shown that camels can seroconvert to the PPRV (Roger et al., 2001). Recent observations in Sudan suggest that camels could be affected by PPR, as they can show clinical expression of the disease and positive results were detected by serological tests, including reverse transcription polymerase chain reaction (RT-PCR), and PPRV was isolated in cell culture (Khalafalla et al., 2010; Kwiatak et al., 2011). In one study, antibodies against PPR were detected in Ethiopia in 3 % of the 628 tested camels (Abraham et al., 2005). Nevertheless, experimental infection with an Ethiopian goat strain in a small number of dromedaries in Dubai did not produce any clinical signs (Wernery, 2011), and also two trials in Morocco showed very poor immunological responses tested by serum neutralisation and enzyme-linked immunosorbent assay (ELISA) in camels vaccinated with Nigeria 75/1 strain. There is a need for more research in this domain to elucidate the role of camels in the epidemiology of PPR, in particular to find out if this species can excrete the virus.

### 1.3.1. Wildlife

Wildlife susceptibility to PPR is a complicating factor, with infection and clinical disease reported in Dorcas gazelle (in captive groups), Thomson's gazelle, gemsbok and ibex (Wohlsein and Saliki, 2006; Gur and Albayrak, 2010), as well as in wild sheep such as bharals in Tibet and in wild goats in Kurdistan (Bao et al., 2011; Hoffmann et al., 2012). Recently, an outbreak of PPR in truly free-ranging Sindh ibex was confirmed by immunocapture ELISA and PCR in Pakistan in 2010 with 36 deaths, possibly associated with the sharing of water pasture with a presumed infected goat herd (Abubakar et al., 2011).

The role of wild species as a reservoir has not been studied. However, considering the role of wildlife in the epidemiology of rinderpest (Shanthikumar et al., 1985; Anderson et al., 1990; Couacy-Hymann et al., 2005; Kock et al., 2006; Rossiter et al., 2006), further research is needed about potential PPR spread through wild species. This may have serious repercussions in Europe, where several wild ruminant species—which are sympatric with domestic sheep and goats, e.g. in mountain areas, such as ibex, chamois and moufflon—would potentially play a role if PPR were to enter Europe.

### 1.4. Pathogenesis and clinical signs

There is variation in the inherent resistance of different breeds of sheep and goats to PPRV (Couacy-Hymann et al., 2007). There is anecdotal evidence that younger animals show higher mortality rates, but this has not been confirmed by experiments. Sheep and goats infected with PPRV show a similar, if slightly less severe, clinical picture to that seen in cattle infected with RPV. There is a rare peracute form of the disease causing death four to six days after the onset of fever. The more frequent acute form is characterised by a sudden rise in body temperature, peaking at 2–2.5 °C above normal. The mucous membranes of the eyes and nose become congested and there is noticeable discharge from the eyes and nose. There is a marked and rapid loss of circulating white blood cells (leucopenia) at this time, starting from two to three days post infection (dpi). The white blood cell count will remain low (about 20 % of normal) and will return to normal only during the convalescent phase. As the disease progresses, congestion can be seen in the gums, and necrotic lesions appear in the epithelial tissue lining the mouth, first in the gums and the inside of the lower lip, and in severe cases can be seen on the top and sides of the tongue and in other parts of the buccal mucosa. The necrotic areas throughout the mouth and gums readily erode. As the disease progresses further, diarrhoea develops, which is occasionally bloody. Many animals with PPR show abnormally rapid or laboured breathing, and a productive cough. By this stage, the animal is apathetic, with laboured breathing and an unwillingness to move. Convalescence, if it occurs, takes several weeks. Any animals that are pregnant at the time of infection will abort. The white blood cell count slowly returns to normal and the oral lesions heal over a period of two to three weeks. This transient loss of white cells, and the generalised immunosuppression that can go on for even longer (see below), means that the animal is susceptible to activation of latent or chronic infections (e.g. with parasites) or to secondary infection by other pathogens. The virus infection, on the other hand, completely resolves in recovered animals, and there is no persistent infection or carrier state (Lefevre and Diallo, 1990). In post-mortem examination, PPRV infection reveals significant lung pathology, with patches of congestion in the lung tissue and signs of pneumonia. Animals show extensive damage to mucous membranes of the digestive tract and to lymphoid organs. Immunohistological examination shows that the virus is primarily lymphotropic, with epithelial tissue involvement in only later stages of infection (Pope et al., 2013). Further details on the pathology of PPR disease can be found in Wohlsein and Saliki (2006).

The morbidity and mortality rate varies enormously (up to 100 %) depending on the species infected, the age of the animals, the prevalence of secondary infectious agents and the PPRV lineage involved (Zahur et al., 2009; Kivaria et al., 2013; OIE, 2013; Chowdhury et al., 2014).

### 1.5. Immune response

The relative importance of humoral (antibody) and cell-mediated (cytotoxic T cell) responses in the recovery from infection with PPRV is not clear. Animals that recover from infection (including

infection with the attenuated vaccine strains of PPRV) have high levels of circulating neutralising antibody as well as antigen-specific proliferating CD4<sup>+</sup> T cells (Dhar et al., 1995; Lund et al., 2000), so either or both responses may be involved in the clearance of the virus. The protective immune response of the host to PPRV infection is, in any event, obscured to some extent by the generalised immunosuppression common to infections with any of the morbilliviruses (reviewed in Schneider-Schaulies and Schneider-Schaulies, 2008). This is not just the marked lymphoid depletion seen in severe disease, as *Morbillivirus*-induced immunosuppression persists after the recovery of peripheral white blood cell count. Peripheral blood leucocytes from animals infected with virulent RPV show reduced response to mitogens and to both RPV and other, unrelated, antigens (Lund et al., 2000), although the vaccine strain of RPV had only a transient immunosuppressive effect (Heaney et al., 2005). Similar results were seen in PPRV-infected goats (Rajak et al., 2005) as well as in rabbits infected with the lapinised strain of RPV (Yamanouchi et al., 1974a; Yamanouchi et al., 1974b).

## **1.6. Routes of transmission**

### **1.6.1. Direct transmission**

Virus transmission is essentially by contact with infected animals, or fresh secretions or faeces from infected animals. Transfer of the virus across any distance is through the movement of livestock, usually through migration or trade. The virus is found in all kinds of secretions, from approximately 3 to 22 dpi (see Table 1), although is probably only infectious up to 12 dpi.

There is no evidence, either direct or indirect, for a role of any arthropod as a vector for PPRV or any other *Morbillivirus*.

### **1.6.2. Indirect transmission**

Virus can be transmitted by fomites, in which the virus can survive up to 72 hours, depending on humidity, temperature, amount of sunlight and many other factors (see Table 1). The virus is probably stable in chilled meat for some days (by analogy with RPV) (Table 1), although there have been no studies with PPRV *per se*. Similarly, the virus is probably stable in milk if kept chilled, with a half-life of two to three days, but there have been no studies on PPRV.

## 2. Case studies of PPR outbreaks

In order to provide possible insights into PPR dynamics and its possible control, some examples of the epidemics of PPR that have occurred in Northern Africa, Turkey and China are reported in the following sections.

### 2.1.1. PPR occurrence in Northern African countries

The recent results from the Food and Agriculture Organization (FAO)-funded project “Toward a harmonized strategy for the control of Peste des Petits Ruminants in North Africa FAO Project (TCP/RAB/3302)” provide insights into the situation on PPR in the Northern African countries Algeria, Egypt, Libya, Morocco, Mauritania and Tunisia updated up to 2012–2013.

This project was set up in 2010, following the emergence of PPR in Morocco (2008). Indeed, Morocco was probably the last Northern African country to be infected by PPRV, which was first detected in Egypt during the 1980s (Ismail and House, 1990). An outbreak of PPR was later reported in the Nile delta in 2006 (Abd et al., 2010); phylogenetic analyses revealed that the causative PPRV belonged to lineage IV and was closely related to PPRV isolated in Morocco in 2008 (Kwiatek et al., 2011). Moreover, serological evidence of PPRV infection was observed in Tunisia in small ruminant samples collected in 2006 (Ayari-Fakhfakh et al., 2011). On the other hand, retrospective surveys on a Moroccan serological bank could not detect PPRV antibodies in small ruminant sera collected before 2008 (Ettair, 2012).

The regional FAO project aimed to adopt a regional approach to fight the disease by strengthening the capacity of epidemiological surveillance and diagnosis in the Maghreb. The socio-economic impact of PPR on farmers and rural communities was also examined.

The results of the epidemiological survey in each country show that the virus is circulating throughout the sub-region except in Morocco, which adopted four years of mass vaccination (the last was in 2011 in eastern Morocco).

Further information was sought through the distribution of a questionnaire to the Northern African countries belonging to the Réseau Méditerranéen de Santé Animale (REMESA)<sup>4</sup> network, in order to increase the understanding about the epidemiology of PPR in Northern Africa and to explore possible pathways of potential introduction of the disease into the EU. The information provided within the timeframe of the present opinion is presented in the following section, and, for the answers from some REMESA countries that will be received at a later stage, the analysis will be performed later and the results given back to the REMESA network.

### 2.1.2. PPR in Mauritania

PPR is enzootic in Mauritania, where livestock farming is generally extensive and where more than 13 million small ruminants are raised. PPR is well known from veterinary services, at least since the early 1980s (Le Jan et al., 1987). PPR incidence has a seasonal pattern, with peaks in autumn and winter. A network for epidemiological surveillance has been in place since 1999 and passive surveillance is conducted at the national level, based on suspected cases. Vaccination is recommended, but the coverage rate is only around 8 %, although projects are in place in order to increase vaccination coverage (El Arbi, 2012).

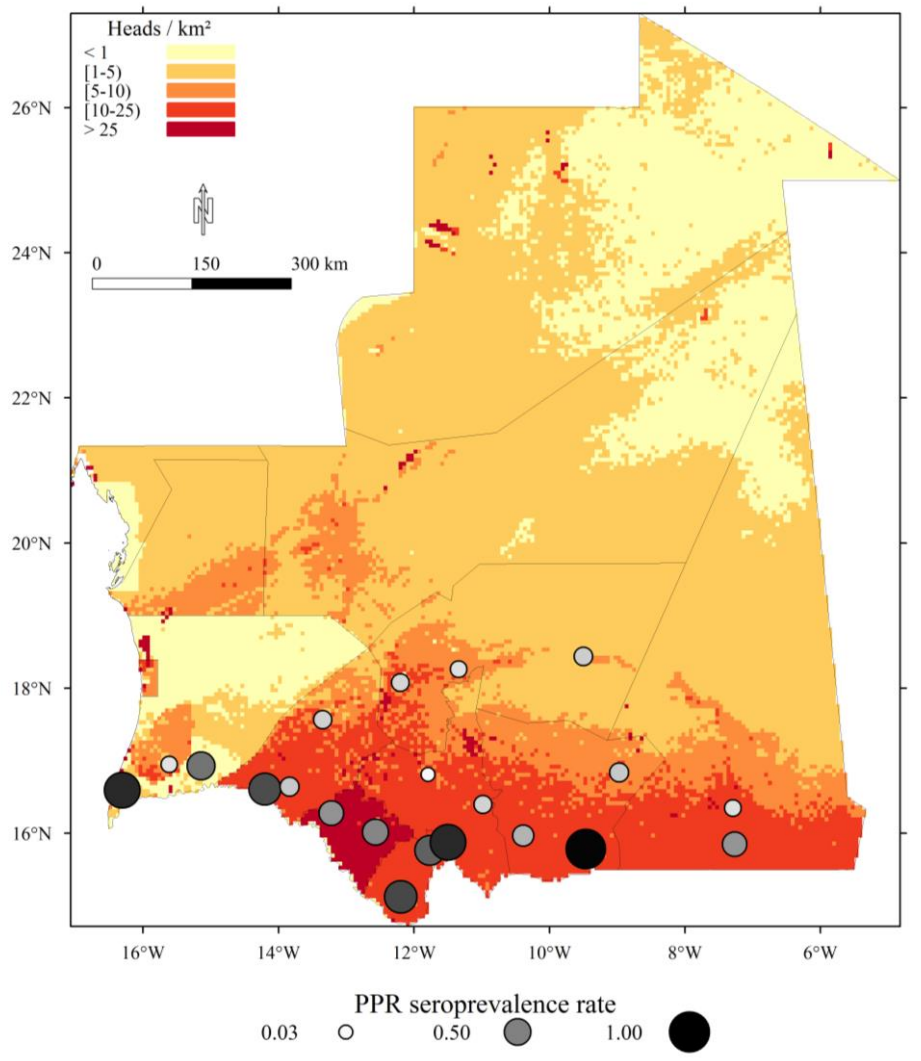
A nationwide seroprevalence survey was implemented in October 2010 (El Arbi et al., 2014). All serum samples were analysed by ELISA. A total of 1 190 sheep and 714 goat serum samples were

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<sup>4</sup> In 2009, the Chief Veterinary Officers of 10 western Mediterranean countries (Algeria, Egypt, France, Italy, Libya, Morocco, Mauritania, Portugal, Spain and Tunisia) created a common framework for work and cooperation, which has the necessary capabilities to assist and coordinate the development and implementation of animal health regional projects and programmes: the Mediterranean Animal Health Network (REMESA, [www.remesanetwork.org](http://www.remesanetwork.org)).

collected and the estimated PPR seroprevalence was 43 % (95 % confidence interval (CI): 38–47 %). PPRV infection was widespread, with an increasing seroprevalence from north to south, which is expected considering the higher density of animals (Figure 3).

In 2012, three suspected outbreaks of PPR were investigated, with morbidity rates of 11 to 17 % and case-fatality rates from 39 to 58 %. A total of 43 animals were sampled for virus detection, and 12 animals from two sites tested positive by RT-PCR. Seroprevalence rates were estimated to be 61 %, 70 %, and 75 % in the three location (n = 87, 31, and 12, respectively). RT-PCR on swab samples revealed that PPRV strain from Mauritania belonged to lineage II, which is closely related to those collected in Senegal and quite distinct from those identified in Morocco and northern Africa (lineage IV) (El Arbi et al., 2014).



Source: Modified from El Arbi et al. (2014)

**Figure 3:** Seroprevalence of PPR according to the serological survey and density of small ruminant population

The situation of PPR outbreaks in Mauritania in 2013 as reported to OIE is displayed in Figure 4.





In 2012, a serological survey was run to assess the level of vaccine protection in small ruminant vaccinated adults, and to detect any virus circulation among unvaccinated young animals. The 846 sera taken from young animals (aged < 8 months) tested negative by ELISA, and 69 % of 455 adults (> 8 months) tested positive by virus neutralisation test.

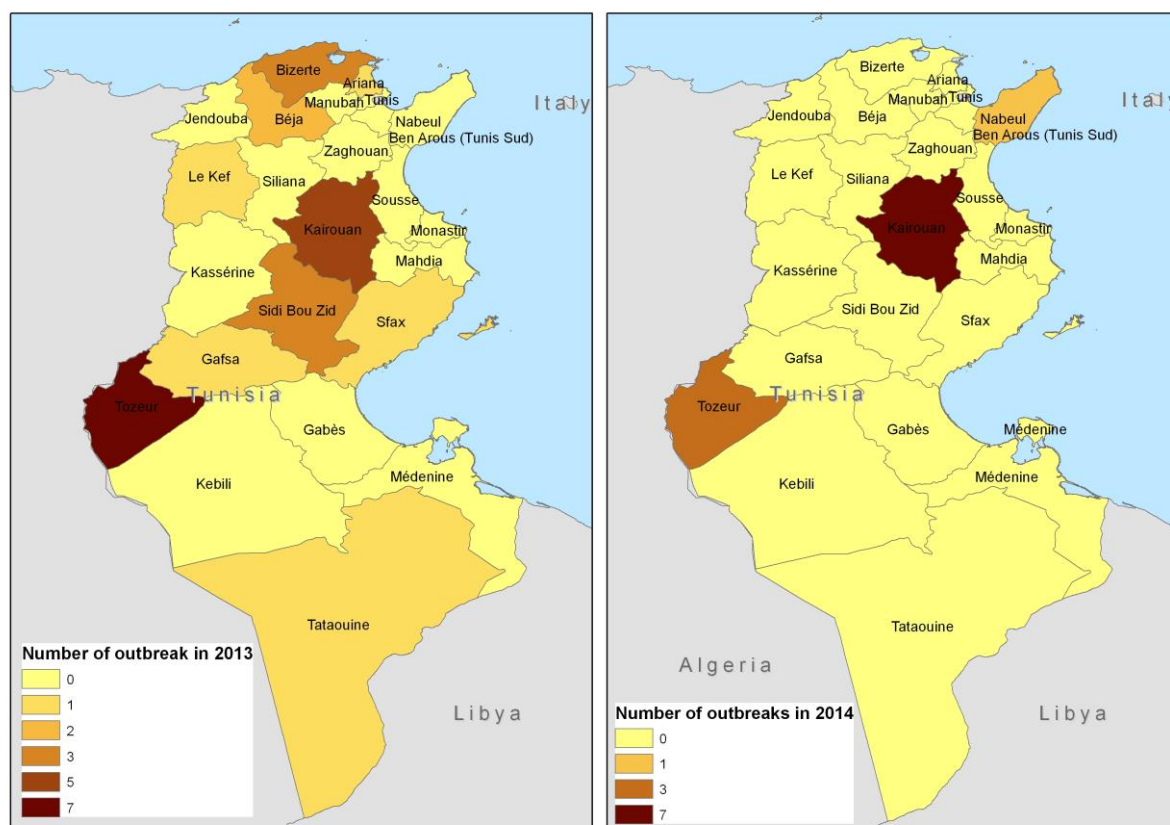
In Morocco, PPR was well controlled at the national level through mass vaccination, thus providing very strong evidence that PPR control can be achieved in Northern Africa, provided that adequate means are available and correctly implemented. Moreover, after the vaccination campaigns, the epidemiological situation was assessed. No viral circulation could be observed among young unvaccinated animals, and a good immune protection rate was achieved in vaccinated adults. However, assiduous vigilance is still needed because there is a risk of PPR reoccurrence given the illegal cross-border livestock movements. Indeed, early detection of such reoccurrence is a necessary condition for a rapid response and effective management of possible outbreaks of PPR. This fragile PPR-free situation in Morocco highlights the importance of designing and actually implementing a regional PPR control strategy, relying on coordinated mass vaccination in infected countries, together with post-vaccination monitoring and efficient active surveillance measures. In particular, a better knowledge of legal and illegal livestock movements is of crucial importance. With regard to this, Morocco has started an individual identification programme for small ruminants.

#### **2.1.4. PPR in Tunisia**

In Tunisia, the first positive serological sample was detected in 2006 (Ayari-Fakhfakh et al., 2011), while the first clinical cases were only reported in 2011. PPRV was isolated from outbreaks that occurred in 2012 and 2013. Phylogenetic studies revealed the isolated strains belonged to lineage IV of PPRV (Soufien et al., 2014).

In 2012, the mean prevalence rate of infected herds was 46 % of 3 619 samples from 367 breeders in 23 governorates, while the average individual-level seroprevalence rate was  $13 \pm 1$  %. The situation of PPR outbreaks in Tunisia in 2013 and 2014 as reported to OIE is displayed in Figure 5.





Data source: OIE

**Figure 5:** Number of outbreaks in Tunisia at the governorate level in 2013 and 2014

According to the answers provided in the questionnaire distributed to the REMESA network, the possible source of the infection of PPR in the country, although not certain, could be the introduction of subclinical infected animals and contaminated vehicles across the borders. The risk of persistence of the infection is probably linked to the non-systematic application of health control programmes, particularly due to insufficient resources for their implementation, and to the continuous introduction of infected animals from abroad. In particular, the southern borders of Tunisia are the ones most at risk of the uncontrolled introduction of animals, owing to the length of the borders and to their unstable geopolitical situation.

Possible measures to reduce the illegal movement of animals could include health checks carried out at border checkpoints and quarantine centres in areas at risk, which at present are not in place. The control measures currently in place include passive surveillance, and no vaccination is performed. Four months before and after Eid al-Adha, a period during which trade and movements of small ruminants are particularly intense, there is a system of permanent sanitary control, put in place mainly because of foot-and-mouth disease outbreaks in 2014, and health checks are carried out.

### 2.1.5. PPR in Algeria

In Algeria, a serological survey in 2011 revealed positivity of 15.20 % in sheep (88/579), and 8.31 % in goats (61/734). In 2012, 20 clinical cases were detected in three outbreaks and attributed to lineage IV PPRV. An emergency vaccination was conducted. A new serological survey in the framework of the FAO project (TCP/RAB/3302) was done on 3 840 small ruminants and 640 camels from 48 provinces. This revealed a prevalence rate of infected herds of 69 % in sheep, 40 % in goats and 0 % in camels. The herd-level prevalence rate was higher in eastern and western provinces (85 %), and lower in southern provinces (29 %). The comparison of sedentary with transhumant herds showed a 61 % prevalence rate in the former and a 57 % prevalence rate in the latter. The individual-level prevalence rate was 18 % in sheep and 24 % in goats.

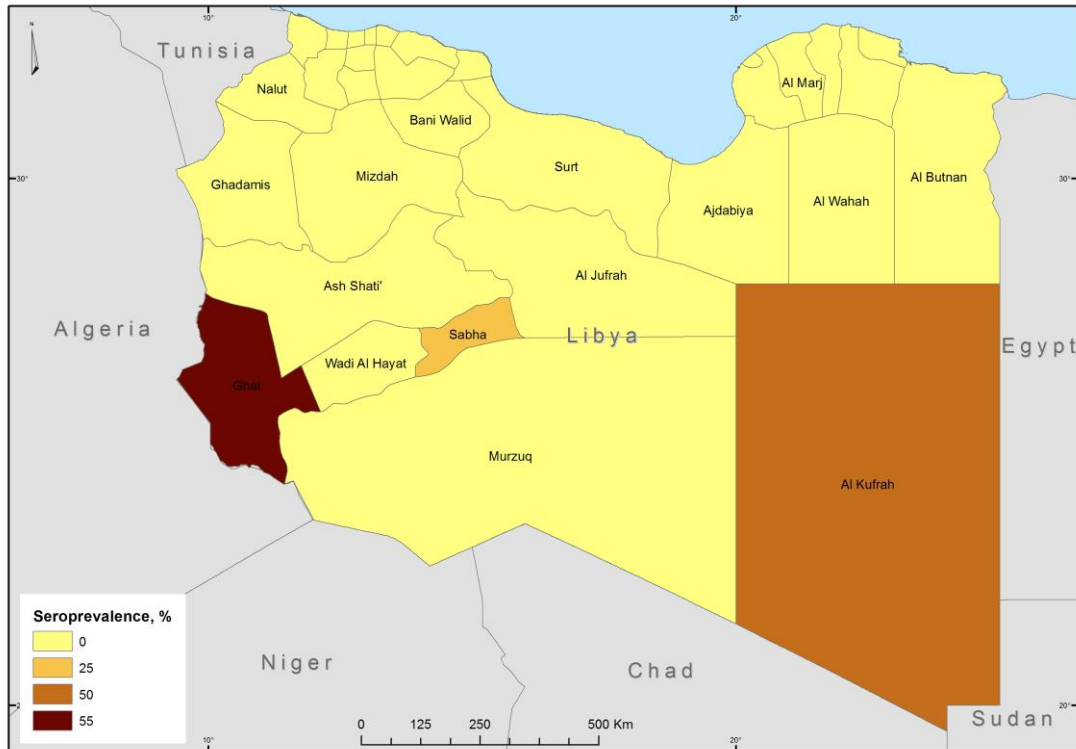
Nevertheless, clinical signs have seldom been observed in Algeria. According to OIE reports, there have been sporadic outbreaks of PPR in the last three years; the most recent ones were four outbreaks reported in January 2013 in the province of Ghardaia, in the centre of Algeria. This situation induced the national authorities to implement vaccination campaigns not at the national level, but only around the outbreaks. A further serological survey is planned in 2015 and, based on the results obtained, a nationwide vaccination campaign will be considered as an option, possibly using a regional approach involving neighbouring countries.

In a previous epidemiological survey conducted in 2010 in Western Saharan territories close to Algerian and Moroccan borders, 133 out of 461 goats (29.0 %), 110 out of 457 sheep (28.8 %) and 1 out of 58 dromedaries (1.7 %) were positive for PPR (Rossi, 2012). Laboratory results confirmed the presence of PPRV in 33.3 % of the samples. Sequence analysis revealed that the virus belonged to PPRV lineage IV and phylogenetic analysis indicated a close relationship (99.3 %) with the PPRV isolated during the Moroccan outbreak in 2008 (De Nardi et al., 2012).

According to the answers provided in the questionnaire distributed to the REMESA network, the possible introduction of PPR into Algeria is likely to be via transboundary uncontrolled movement of infected animals, mostly via land transportation. These movements are favoured by the vastness of the Algerian borders, which are difficult to control, especially in the southern desert areas bordering with Sahel countries where the disease is endemic. Owing to this situation, the movement of animals within the country from southern provinces to northern provinces is forbidden. Within the country, PPR is considered to spread further, mostly because of extensive farming and the high density of small ruminants in the steppe areas, where common pasture and water points are shared, thus enhancing contact rate and favouring the transmission of the disease. Moreover, cold and rainy seasons are considered one of the factors that help in maintaining virus persistence at the farm level. In this context, the identification of small ruminants, which is not fully implemented at present, and effective veterinary checks at the gathering points of animals (markets, fairs, etc.) are additional elements that may reduce illegal movements and consequently the risk of PPR spread. In particular, before and during Eid al-Adha, the Islamic festivity during which trade and movements of small ruminants are particularly intense, supplementary measures are put in place including (i) the identification and authorisation by local authorities of the points of animal sale, where veterinary controls are strengthened, (ii) action of different sectors of the authorities (customs, police, etc.) for the control of animal movement that can only take place if certified and (iii) implementation of awareness campaigns for farmers and the general public.

#### **2.1.6. PPR in Libya**

According to the results produced in the FAO project TCP/RAB/3302, the mean serological prevalence rates registered in 2013 were 34.5 % (25 % in local animals and 45 % in imported heads from e.g. Sudan, Chad, Mali; 25 % in animals < 12 months and 41 % in animals older than two years). Higher serological prevalence rate was observed in southern provinces, probably due to significant uncontrolled animal movements (Figure 6).



Data source: FAO

**Figure 6:** PPR seroprevalence rate in the Libyan provinces in 2013

### 2.1.7. PPR in Egypt

In Egypt, PPRV was isolated in 1987 and 1990. Since then, all the PPRV reports have been based on the detection of virus antibodies. However, an outbreak was observed in goats in the Nile Delta in 2006 (Abd et al., 2010). Nevertheless, Egypt was officially free of PPR according to OIE until the outbreaks at sheep farms in Ismailia and Cairo provinces in 2012. In a study conducted by (Soltan and Abd-Eldaim; Soltan and Abd-Eldaim, 2014), a total of 40 clinical samples were tested in 2010 (goats) and 2012 (sheep) for PPRV by RT-PCR. About 21 samples (52.5 %) were positive. The phylogenetic analysis of the detected viruses revealed the circulation of PPRV lineage IV. In 2012, the serological survey revealed 29 % positives out of 1 525 collected serum samples.

The situation of PPR outbreaks in Egypt in 2013 and 2014 as reported to OIE is displayed in Figure 7.



### 2.1.8. PPR in Turkey

In Turkey, the first detection of PPR was in 1992, in Mardin/Kiziltepe (south-east Anatolia), and it was detected using serological techniques (Guler et al., 2014). The initial occurrence of PPR in Turkey also coincided with the eradication of rinderpest from the country.

Later, in 1995–1996, PPR was suspected by clinical and necropsy findings, and 17 sheep and 6 goat herds were investigated with serological and virological tests, confirming the diagnose of PPR (Alcigir et al., 1996; Tatar and Alkan, 1999). The virus was isolated and identified phylogenetically as PPRV lineage IV. In affected herds, incidence was reported to be 87 % in sheep and 90 % in goats, while case-fatality during outbreaks was found to be 6–30 % in sheep and 32–80 % in goats.

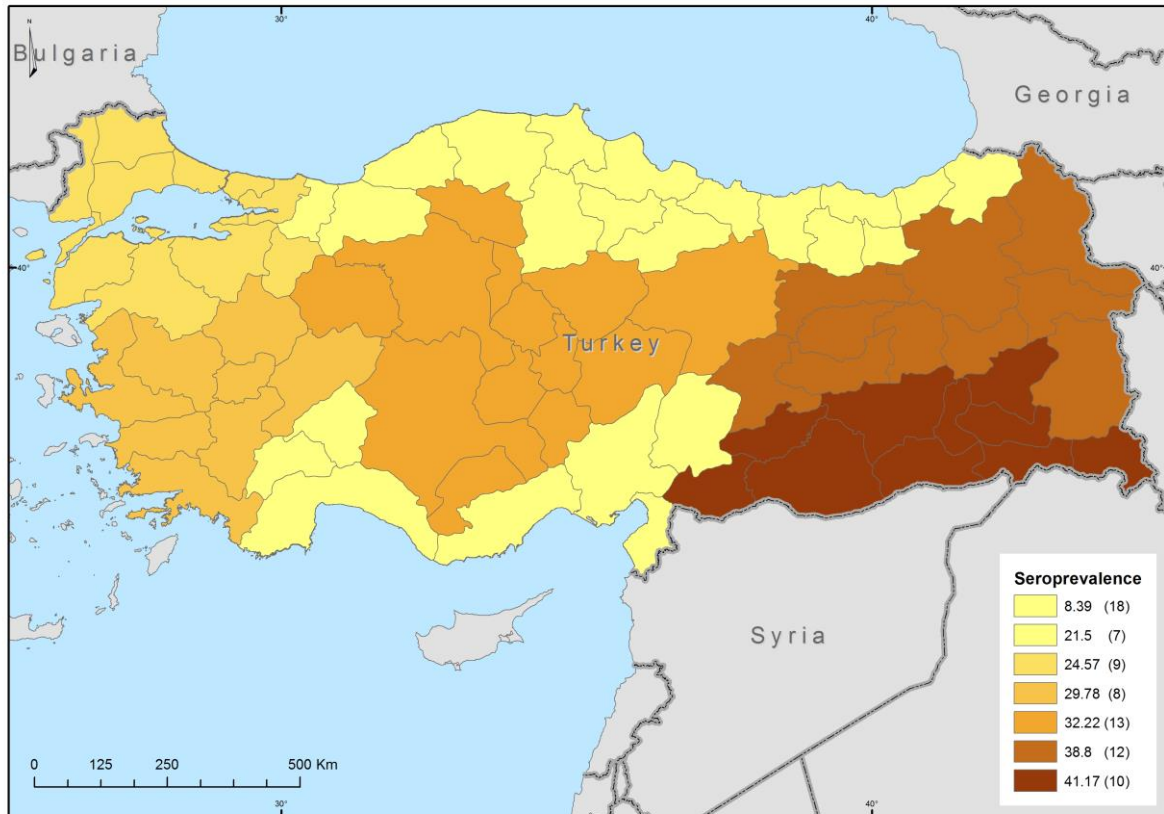
Since then, PPR has rapidly spread in all regions of Turkey to become an endemic disease. Serological virus detection and immunohistochemical studies carried out in different regions of Turkey indicated the presence of PPRV infection in all regions of Turkey (Guler et al., 2014).

In a prevalence study conducted in 1999–2000, a total of 12 799 sheep and goat blood serum samples from 590 herds were collected and tested (Tatar et al., 2002). Prevalence was reported to be 29 % in sheep and 24 % in goats with an average prevalence of 28 %.

As PPR is endemic in Turkey and extensive vaccination has been implemented, typical clinical signs of the disease and/or high mortality rates have been seldom observed in recent years. Mixed infections with pestiviruses, sheep and goat pox virus, bluetongue virus and secondary bacterial agents of pneumonia, probably caused by the immunosuppressive effects of the virus, have frequently been observed (Toplu, 2004; Kul et al., 2008; Ozmen et al., 2009). In this situation laboratory diagnosis in particular is essential for confirmation of the disease.

Seroprevalence results by geographical regions of a regional reference laboratory are given in Figure 8. No significant difference was found in seroprevalence in terms of species, age groups, sex and breeding type of sheep and goats. On the other hand, differences in prevalence between regions were interpreted to be possibly connected with the extent of breeding activities and animal movements.



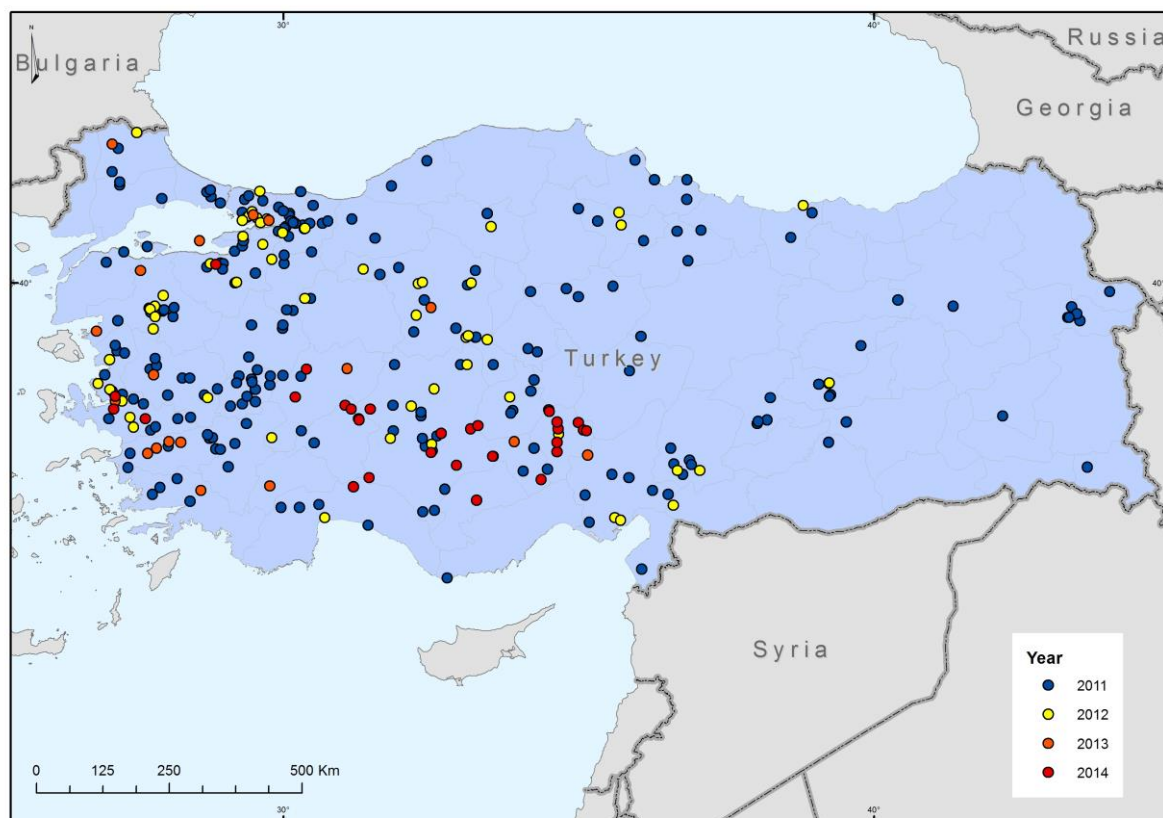


Data source (Tatar et al., 2002): In brackets: the number of provinces in each region

**Figure 8:** PPR seroprevalence in 1999–2000 (%) before vaccination

PPR has been included in the list of notifiable diseases since September 1997. Since the first official report of PPR in the Eastern Anatolia region, outbreak vaccinations were carried out with rinderpest vaccines until 1999 and, after that, following the termination of rinderpest vaccination, imported PPR vaccines were used for the same purpose. In 2002, the Central Veterinary Research Institute initiated the production of PPR vaccines (Nigeria 75/1 strain). Vaccination has been reported to provide at least two-year immunity under suitable conditions and in appropriate age groups of sheep and goats. In the following years, the disease was included in the scope of disease control activities with the implementation of vaccination of contact sheep and goats in PPR outbreaks. In 2004, a seroprevalence study was performed in the Thrace region within the scope of the FAO technical cooperation project (TCP/RER/2903) and the average PPRV seroprevalence was found to be 13.61 %, with prevalence values for the different provinces of 3.5 % in Canakkale, 15.7 % in Edirne, 27.5 % in Istanbul, 15.1 % in Kırklareli and 23.2 % in Tekirdag.

The occurrence of PPR outbreaks reported in 2011–2014 is displayed in Figure 9.



Data source: ADNS

**Figure 9:** Reported outbreaks of PPR in Turkey from 2011 to September 2014

The implementation of the project TR 0802.08 “Ear-Tagging and Vaccination of Sheep and Goats Project” was initiated in 2010 and run for two years upon a ministerial decision and is on-going with financial support from the EU and the Turkish Ministry of Agriculture. The objective of this project was ensuring the ear-tagging and identification of small ruminants, registration of these in the relevant database and their vaccination against PPR, thereby improving herd control measures for the gradual reduction of disease incidence. This project has been completed and the project report has been submitted to the EU. During this three-year period, vaccination campaigns were performed. The vaccination coverage was 65 % in 2010 (22 922 758 animals), 76 % of young animals in 2011 and 68.5 % of young animals in 2012. The vaccinated animals were registered, television spots and leaflets were prepared, training campaigns were run for public awareness and a contingency plan was prepared. The control strategy for 2014 includes the vaccination of all susceptible animals in Thrace and of all animals in the Anatolian areas where PPR outbreaks are reported. In addition, young animals and previously unvaccinated adult animals will be vaccinated in areas where PPR outbreaks have not been observed.

### 2.1.9. PPR in China

PPR has previously been identified in Tibet (2007, 2008 and 2010), which were the first reports of PPRV in China. The outbreaks were tackled with a combination of stamping out, movement control and vaccination. The outbreaks were limited and did not spread to the rest of the country (Figure 10).





Source: OIE

**Figure 10:** Maps illustrating the locations of reported outbreaks of PPRV in China in 2007–2010

PPRV was again reported in China in December 2013. This outbreak began in Xinjiang province, on the border with Kazakhstan and Kyrgyzstan (neither of which has reported PPR at any time). It is possible that the virus came from Tajikistan, which also reported PPR in 2013. The virus isolated in China throughout this outbreak has been shown by sequence analysis to be related to the Tajikistan virus (manuscript submitted for publication), although this does not mean that the virus entered China directly from that country. The virus was then discovered in many places across the country, reaching the north-east border with Russia and the south-east border with Vietnam by April 2014 (Figure 11).



The dates given are the dates of the official reports provided to OIE

**Figure 11:** Map illustrating the locations of reported outbreaks of PPRV in China in December 2013 to April 2014

The initial control procedures (first six months) involved stamping out and movement control. The large number of outbreaks suggests that, even in a relatively well-organised country, this was ineffective. Vaccination commenced around the end of March 2014. After the outbreak observed on 25 May and reported on 17 June 2014, at which time there had been 254 individual premises/areas reporting infection (EMPRES-i), two further outbreaks have been reported in October 2014 with 94 cases and 67 deaths. Nevertheless, the observation date of these last outbreaks was not provided, so this information should be confirmed in order to clarify the timeline of the events.

Since December 2013, in China there have been 33 500 PPR cases and 14 700 deaths, and 56 300 animals have been destroyed.

This large-scale outbreak suggests that either the disease was present in large parts of China for some time before it was recognised (which is possible, but requires mortality rates of 16–40 % to have been overlooked or misinterpreted), or the disease has been spread over most of the country in six months, presumably by the movement of animals for trade. It is notable that the initial set of outbreaks followed the main transport routes from west China to the more populated regions in the east of the country.

The Chinese view, from senior scientists at the China Animal Health and Epidemiology Centre (CAHEC), Qingdao, China, is that the widespread outbreak was the result of extensive movement of animals prior to the Spring Festival (Chinese New Year). Once the animal movements had been traced fully, no further outbreaks were observed (after May 2014).

## **2.2. PPR occurrence in other countries in 2014**

OIE reports of the immediate notifications and follow-up contain some epidemiological information about outbreaks, including their possible source. In Table 2, this information is summarised for the countries that reported PPR outbreaks in 2014.

**Table 2:** PPR reports from affected countries in 2014 including the origin of the outbreaks and control measures applied (Source: OIE)

Country	Year of outbreaks	Source of the outbreak(s) or origin of infection	Epidemiological comments	Control measure applied
Angola	2012 and continuing	<ul style="list-style-type: none"> <li>• Introduction of new live animals</li> <li>• Illegal movement of animals</li> </ul>	Involved a herd of 55 sheep/goats brought from the Democratic Republic of the Congo, despite the prohibition of imports from PPR-affected countries. Positive serological results were picked up in a farm during routine surveillance. No clinical evidence suggestive of the disease has been found so far in the area. Surveillance activities have been intensified in all of the seven provinces bordering neighbouring infected countries and vaccination is planned and will take place within the next 2–3 weeks. Note by the OIE Animal Health Information Department: since this is the first time that PPR is identified in the history of the country, the reason for notification as first occurrence applies to the country, but the event is in fact circumscribed to a zone	<ul style="list-style-type: none"> <li>• Quarantine</li> <li>• Movement control inside the country</li> <li>• Screening</li> <li>• Zoning</li> <li>• Disinfection of infected premises/establishment(s)</li> <li>• No vaccination (to be applied)</li> <li>• No treatment of affected animals</li> </ul>
Bhutan	2014	<ul style="list-style-type: none"> <li>• Introduction of new live animals</li> <li>• Illegal movement of animals</li> </ul>		<ul style="list-style-type: none"> <li>• Movement control inside the country</li> <li>• Disinfection of infected premises/establishment(s)</li> <li>• Dipping/spraying</li> <li>• Vaccination prohibited</li> <li>• Treatment of affected animals (antimicrobials/vitamins)</li> </ul>
Comoros	2012 and continuing	<ul style="list-style-type: none"> <li>• Introduction of new live animals</li> <li>• Contact with infected animal(s) at grazing/watering</li> </ul>	Losses range from 50 to 80 % in some outbreaks. Affected and dead animals showed the following signs: fever, anorexia, nasal discharge, lacrimation, salivation, profuse and foul-smelling diarrhoea, difficulties for breathing with cough and, finally, death in 3 to 5 days	<ul style="list-style-type: none"> <li>• No vaccination</li> <li>• No treatment of affected animals</li> </ul>
Congo (Republic of the)	2005, endemic 2009	<ul style="list-style-type: none"> <li>• Unknown or inconclusive</li> </ul>		<ul style="list-style-type: none"> <li>• Quarantine</li> <li>• Movement control inside the country</li> <li>• Zoning</li> <li>• Disinfection of infected</li> </ul>

Country	Year of outbreaks	Source of the outbreak(s) or origin of infection	Epidemiological comments	Control measure applied
				<ul style="list-style-type: none"> <li>premises/establishment(s)</li> <li>• Modified stamping out</li> <li>• No vaccination</li> <li>• No treatment of affected animals</li> </ul>
Kenya	2006, endemic 2007	<ul style="list-style-type: none"> <li>• Illegal movement of animals</li> <li>• Contact with infected animal(s) at grazing/watering</li> </ul>		<ul style="list-style-type: none"> <li>• Quarantine</li> <li>• Movement control inside the country</li> <li>• Screening</li> <li>• Vaccination in response to the outbreak(s)</li> <li>• Treatment of affected animals (antibiotics)</li> </ul>
Mali	2004, endemic 2005	<ul style="list-style-type: none"> <li>• Unknown or inconclusive</li> </ul>	Clinical observations: diarrhoea, lacrimation, salivation, nasal discharge. Post-mortem observations: abscess and lung congestion	<ul style="list-style-type: none"> <li>• Quarantine</li> <li>• Movement control inside the country</li> <li>• Vaccination in response to the outbreak(s)</li> <li>• No treatment of affected animals</li> </ul>
Tajikistan	2013 and continuing	<ul style="list-style-type: none"> <li>• Illegal movement of animals</li> <li>• Animals in transit</li> </ul>		<ul style="list-style-type: none"> <li>• Quarantine</li> <li>• Movement control inside the country</li> <li>• Screening</li> <li>• Zoning</li> <li>• Vaccination in response to the outbreak(s)</li> <li>• Disinfection of infected premises/establishment(s)</li> <li>• Dipping/spraying</li> <li>• Modified stamping out</li> <li>• No treatment of affected animals</li> </ul>
Uganda	2007, endemic	<ul style="list-style-type: none"> <li>• Unknown or inconclusive</li> </ul>	Note by the Animal Health Information Department: this is the first laboratory confirmation of PPR in Uganda	<ul style="list-style-type: none"> <li>• No vaccination</li> <li>• Treatment of affected animals to prevent secondary infections</li> <li>To be applied: <ul style="list-style-type: none"> <li>• Control of wildlife reservoirs</li> </ul> </li> </ul>

Country	Year of outbreaks	Source of the outbreak(s) or origin of infection	Epidemiological comments	Control measure applied
				<ul style="list-style-type: none"><li>• Quarantine</li><li>• Movement control inside the country</li><li>• Screening</li><li>• Zoning</li><li>• Vaccination in response to the outbreak(s)</li></ul>

### **3. Mapping of animal movements in the regions of concern and other countries of the Mediterranean Basin and Black Sea**

#### **3.1. Data and methodologies**

In section 3, movements of live sheep and goats and meat from extra-EU countries in the Mediterranean Basin and Black Sea area to EU MSs, and inside the affected EU MSs and Turkey, are presented with the use of flow maps. The data underlying the maps originates from trade records from Eurostat and the UN COMTRADE database, from records from border inspection posts (TRACES system) and from data from national authorities of Turkey as an example of a neighbouring country affected by PPR.

Furthermore, the risk of PPR spread linked to nomadic movement patterns at the regional level and political unrest and related movements of people and refugees, especially in Middle Eastern countries, has been considered.

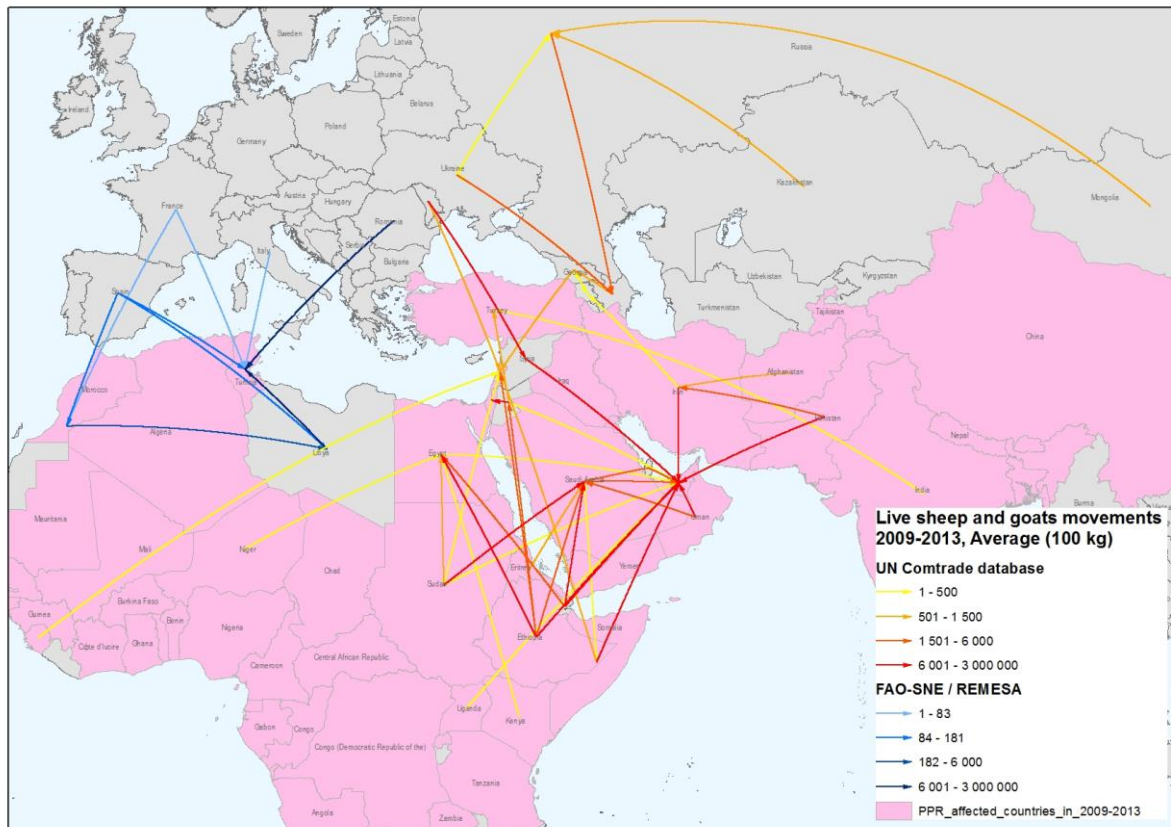
#### **3.2. Import/export of live animals from third countries to Member States**

According to EU animal health legislation, the import of live animals from countries where sheep pox and goat pox is endemic is forbidden; therefore, since sheep and goat pox is endemic in Northern African countries, Turkey and other Middle Eastern countries facing the Mediterranean Sea, there should be no import of live animals into the EU. Nevertheless, some discrepancies were noticed when commercial data (Eurostat) were compared with veterinary border checks (TRACES system). For example, some movements of live sheep and goats were registered in Eurostat in 2011 from Turkey to Bulgaria (269.2 tonnes of live animals) and from Turkey to Hungary (48.7 tonnes of live animals) and in the UN COMTRADE database, with amounts 50 times higher. These discrepancies could be explained because of the different data collection systems in place at Eurostat (data collection through MS Ministry of Finances) and TRACES (data collection according to veterinary checks taken place at EU borders as defined by EU public or animal health legislation). In the case of live animal import, TRACES data could be considered more accurate; nevertheless, proper validity cross-checks should be carried out. It may be worthwhile to verify the consistency between different data sources and reporting systems for animal movement.

#### **3.3. Movement of sheep and goats among African and Middle Eastern countries**

The commerce of small ruminants is the most likely reason for the spread of PPR across borders. In order to provide insights into the animal movements in areas where PPR is endemic and from where possible risk of introduction to Europe or neighbouring countries, the trade movements of live sheep and goats between African and Middle East countries as registered in the UN COMTRADE database are displayed in Figure 12.

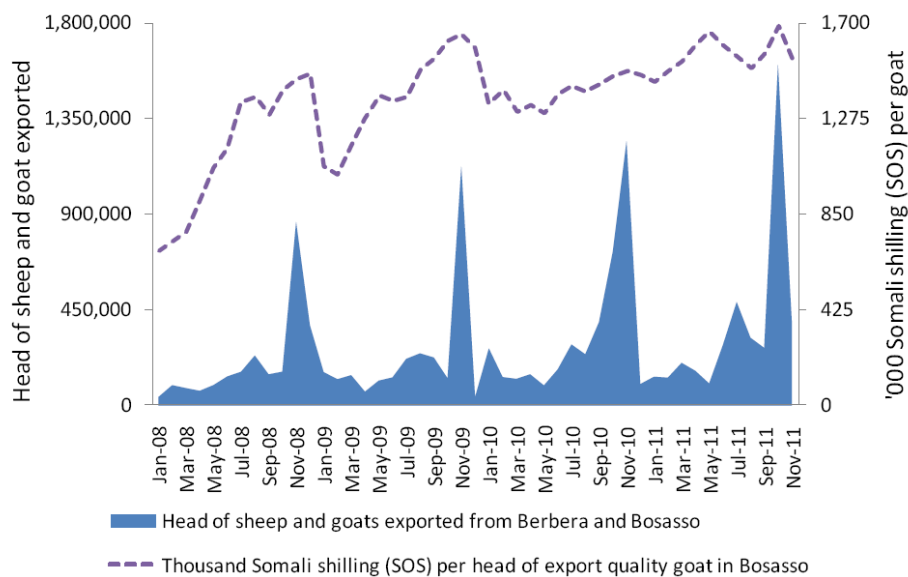




Data source: UN COMTRADE and FAO-SNE/REMESA. Countries affected by PPR in 2009–2013 are displayed in pink.

**Figure 12:** Movements of live sheep and goats among African and Middle East countries displayed as average amount during the period 2009–2013

It is evident from the map in Figure 12 that the biggest movements are from East Africa towards the Arabian Peninsula. The consumption and importance of these products in Muslim countries reaches its peak during religious festivities which encourage trade and commerce of the animals in the region. Such movements for trade or to meet grazing and watering needs are poorly regulated. Although the countries of North Africa are net importers of live sheep and goats, there remains an informal trade pattern across the borders that may, at a given point, favour the spread of PPR (or other transboundary animal diseases) to other countries of the region or to countries of southern Europe. The large population of sheep and goats around the Mediterranean, notably Morocco and Algeria, with more than 22 million sheep and goats each, is an important factor that should be taken into consideration, given the economic impact that this disease represents (Sanz-Alvarez et al., 2008). According to data collected by REMESA, there are movements of live sheep and goats from European countries to Morocco, Algeria and Tunisia, especially from Spain, France and Italy and higher amounts from Eastern European such as Romania. These countries are net importers of live sheep and goats, in particular in certain periods such as the end of Ramadan. The increase in livestock movements during the holy month celebrations of Ramadan and Eid al-Adha can negatively affect the containment of the disease. Livestock exports have some year-round demand, but the seasonality is such that there is a minor increase in demand in preparation for Ramadan and there is a substantial spike in demand, especially for sheep, in the month preceding the Hajj, owing to high demand for sheep or cattle to sacrifice on Eid al-Adha (Figure 13). This annual spike in demand drives price movements and demand patterns, especially for sheep, across the Horn of Africa (FEWS-NET, 2012).

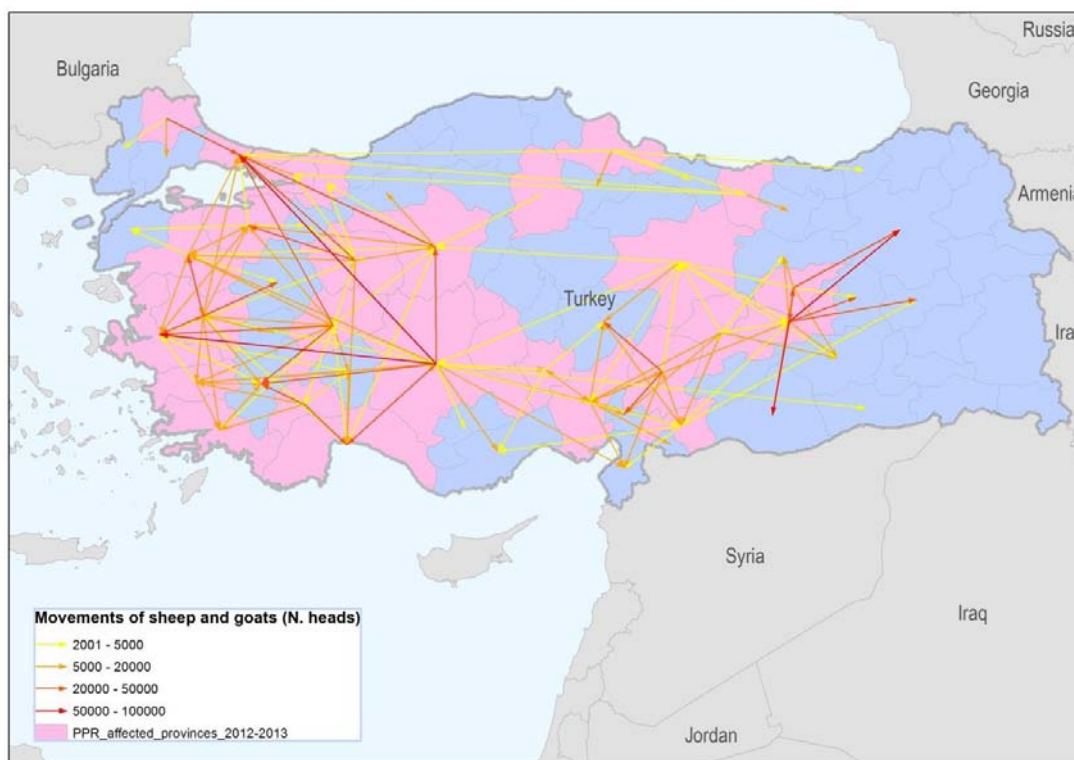


Source: Food Security and Nutrition Analysis Unit (FSNAU)/FEWS NET

**Figure 13:** Volume of goats and sheep exported from Somalia to the Arabian Peninsula in 2008–2011 and prices of export quality goats

### 3.4. Animal movement inside Turkey

In the following map (Figure 14), the movements of small ruminants inside Turkey are presented. The movement data were obtained by the Ministry of Agriculture of Turkey. The PPR-affected provinces are also displayed.



Source: Turkish Ministry of Agriculture and OIE, modified

**Figure 14:** Animal movement in Turkey in 2012 related to the trade of small ruminants from provinces affected by PPR in 2012–2013 (in pink)

In Figure 14, it is evident that several thousands of small ruminants, up to 100 000 heads a year, are usually shipped from PPR-affected provinces in Turkey, including from provinces in southern and western Turkey that have been affected by PPR and which are a long distance from other provinces in western Turkey including Thrace and Istanbul. This highlights the importance of promptly setting movement restrictions when PPR cases are detected to limit the spread of the disease.

### 3.5. Uncontrolled movements of animals and animal products

The EU has issued several directives and regulations pertaining to the import of animals and products of animal origin and veterinary controls on importation;<sup>5</sup> however, uncontrolled movements of animals and animal products still occur worldwide and may favour the spread of transboundary diseases if infected animals or contaminated animal products are introduced into naive countries or areas.

The illegal transport of live animals are linked to several drivers at the socio-economical (poverty, urbanisation, demographic change), political (unrests) or geographical (e.g. droughts, remote areas) levels. This may not necessarily be valid for the illegal movement of small amounts of animal products in passengers' luggage or that is sent by mail, as they occur frequently throughout the world (Beutlich et al., 2014; Nagy et al., 2014; Rodríguez-Lázaro et al., 2014; Schoder et al., 2014).

#### 3.5.1. Uncontrolled movement of live animals in Northern Africa

The network of REMESA, in particular the working group of RESEPSA, which deals with the socio-economic impact of transboundary animal diseases in Northern Africa, has analysed the problem of uncontrolled movements of animals in Northern Africa. In this region, most uncontrolled movements of animals occur by land transport; in fact, this region is characterised by very long borders in the desert that vary between 460 km (Tunisia–Libya) and 1 560 km (Morocco–Mauritania). This remains a major constraint for the control services. The flow of uncontrolled movements of animals across the border is difficult to estimate and depends on several factors (price changes, relationships between families, climates, Ramadan, etc.). This type of movement occurs along the east–west axis between Northern African countries, but also from south to north across the borders with Niger, Chad, Mali, Senegal and Sudan, which represent a serious risk of the transmission of infectious diseases, as these countries have worse health situations than the countries in North Africa. These events are also a commercial constraint because they limit legal trade with other countries (Oueslati, 2012).

The quantitative estimation of illegal animal movements is not simple, as, by definition, illegal activities are not recorded. The movements detected by the border police represent an underestimation and this information is not easy to access, as it is not centralised across countries, or, if any international system is in place, the reporting may not be complete. An attempt to provide figures for this type of movement could be done by screening the local press. According to data provided by the management of the Tunisian Customs, during the months of August and September 2013, customs officials seized 24 goats in Medenine in south-east Tunisia.<sup>6</sup> In September 2014 in Algeria, in the province of Tebessa, the gendarmerie succeeded in stopping the attempted illegal entry into Tunisia of 115 heads of goats.<sup>7</sup> These are, of course, anecdotal reports and any extrapolation would lead to erroneous figures, unless a systematic reporting system is put in place. At the moment, the real number of these movements remains unknown, but is certainly much higher.

<sup>5</sup> Council Directive 91/496 on veterinary checks on animals entering the EU from third countries; Council Directive 97/78/EC on organisation of veterinary checks on products entering EU from third countries; Commission Decision 2003/623 about development of an integrated computerised veterinary system known as Traces; Commission Regulation 136/2004 on the procedures for veterinary checks at EU BIPs on products imported from third countries; Commission Decision 2007/275 on lists of animals and products to be subject to controls at Border Inspection Posts (BIPs); Commission Regulation 206/2009 on the introduction into the Community of personal consignments of products of animal origin.

<sup>6</sup> African manager news, 13th October 2013, available online: <http://www.africanmanager.com/156689.html>

<sup>7</sup> Libertè, 3rd September 2014, available online: <http://www.liberte-algerie.com/algerie-profonde/breves-de-l-est-227733>

### 3.5.2. Illegal movement of products of animal origin

Most of the EU regulations mainly refer to commercial trade in food and food products and usually only in large quantities.<sup>8</sup> Nonetheless, some food products are exempted from customs control. Among these products are those intended for personal consumption, which are potentially present in traveller luggage, those sent by post in small volumes to individuals and those sent as trade samples. These food products of animal origin may represent a pathway of introduction of transmissible disease of humans and livestock.

For the illegal movement of animal products, some estimations have been done, especially for the transport of meat or dairy product in passengers' luggage that can be checked by samples at customs in big transportation hubs such as airports or railway stations (Beutlich et al., 2014; Schoder et al., 2014). This study by Schoder et al. (2014) was conducted at the airport in Vienna, where, in a period of six months, spot checks were made on the luggage of 61 355 passengers from 240 flights from non-EU countries; 1 473 products of animal origin were confiscated (6 229 kg, 86.4 % from countries at high risk of communicable animal diseases). Of these, 43.7 % were meat products, with a total weight of 452.9 kg. Of these products, 70 % originated from China, Turkey and Egypt, where PPR occurs. These animal species could be determined in 57.6 % of all cases owing to missing or incomplete labelling, and only two samples comprised mutton or lamb.

Beutlich et al. (2014) performed microbiological analysis on a total of 663 food items seized from 296 passengers arriving in Germany from 35 different departure countries, half of which were meat or meat products including a whole lamb carcass. The majority of confiscated items (51 %) originated from Turkey (where PPR is present) and Russia.

### 3.6. Animal movements related to transhumance of small ruminant flocks

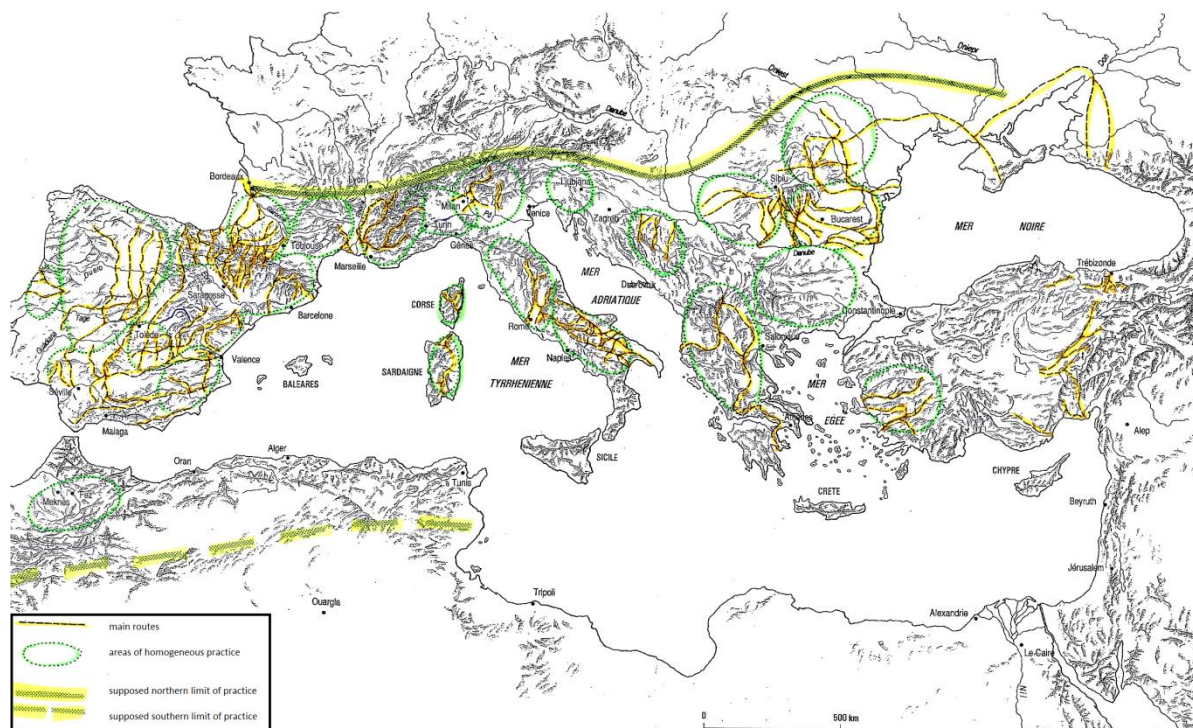
The understanding of nomadic movement patterns at the regional level could be considered as a necessary element to provide insights about potential spread of animal disease.

In many countries, including Europe, transhumance (a traditional livestock practice based on the movement of livestock between winter and summer pastures) and pastoralism (the practice of mobile livestock raising by use of extensive grazing on rangelands) are husbandry practices used to take advantage of the characteristic instability of rangeland environments, characterised by economic rationality and ecological sustainability (Krätli et al., 2013). In mountain areas of many European countries, although they have shown a declining trend in the last decade, transhumance is still largely practised (Figure 15), and its advantages in playing a significant role in conserving biodiversity and in sustainably using marginal areas are broadly described (Halstead, 1987; Ruiz, 2001; Olea and Mateo-Tomás, 2009).

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<sup>8</sup> European Commission International Affairs—Import Conditions (2012), available online: [http://ec.europa.eu/food/international/trade/index\\_en.htm](http://ec.europa.eu/food/international/trade/index_en.htm)





Source: modified from Duclos and Pitte (1994)

**Figure 15:** Main transhumance routes in the Mediterranean area

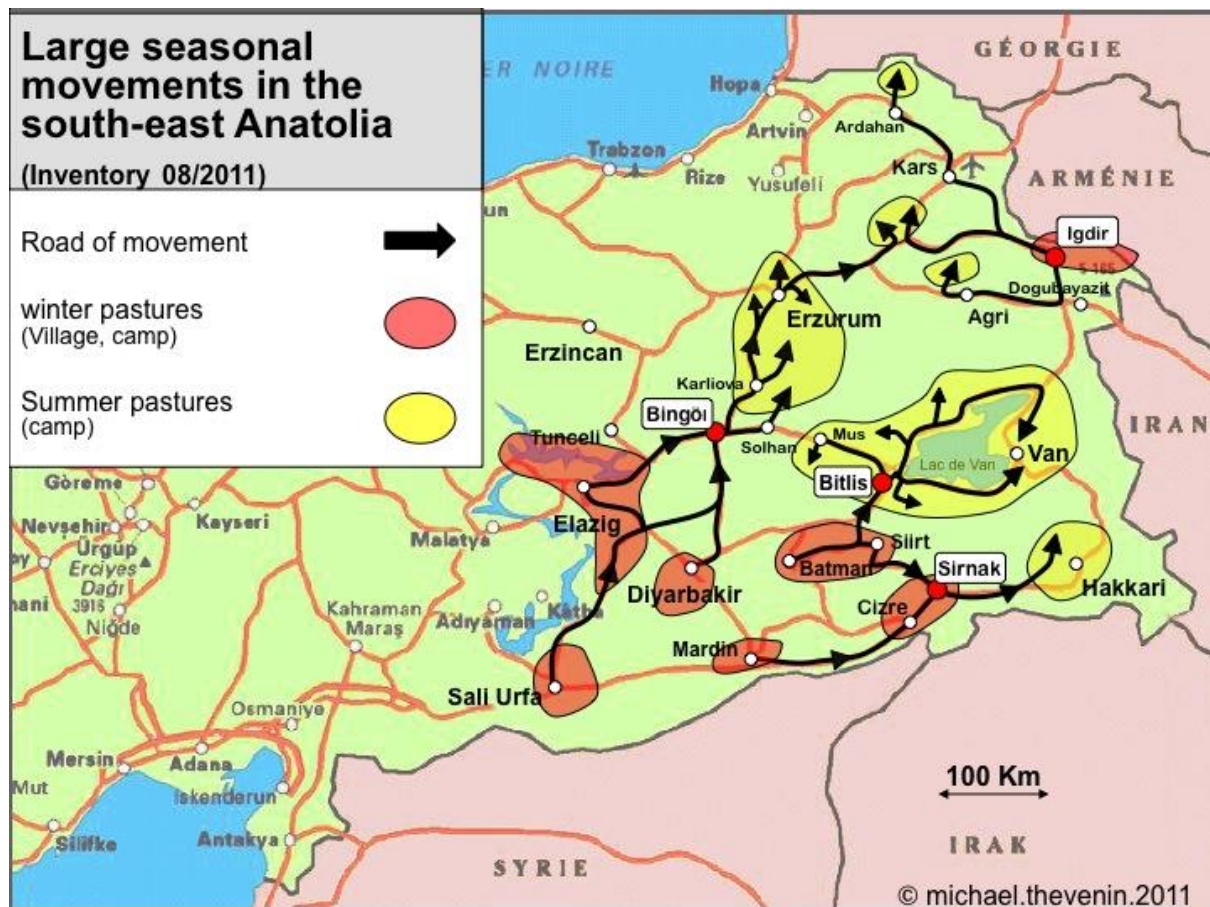
Through strategic mobility, transhumance and pastoralism find an asset in the existence of dynamic variability in the drylands, where sedentary agriculture or mixed farming are not suitable practices. When this balance is disrupted by external factors and the mobility of pastoralists is impeded because of, for example, unstable political situations, wars, etc., decreased and constrained access to pasture and water resources, impeded livestock movements and limited access to veterinary services become some of the key factors contributing to increasing prevalence and persistence of livestock diseases in nomadic systems (Bett et al., 2009).

Moreover, considering the lessons learnt in the Saharan–Sahel context, pastoralism has been internationally recognised as one of the best stabilisation strategies for remote and unstable areas (Plateforme Regional Tchadienne, 2013); thus, it could be seen as a practice to be promoted especially in critical situations such as the current ones in the Middle East (see section 3.7). Furthermore, the support to pastoralism may lead to socio-economic advantages, since it may keep or open employment chances and reduce land abandoning and urbanisation of rural communities with the potential related social problems.

Different patterns of migratory movement of small ruminants are present in Turkey, where 29 million sheep and 9 million goats are present and where husbandry practices range from modern breeding to transhumant nomadic and semi-nomadic pastoralism. Apart from being one of the most ancient cultures linked to different tribes, the transhumant practice, if properly supported, guarantees sustainable use of rangelands, especially in south-eastern Turkey, also considering that pastures occupy around 27 % of the total land surface of Turkey (Thevenin, 2011).

Pastoralism in Turkey has two forms: village-based sedentary pastoralism and pastoralism with vertical or horizontal movements. The latter may take the form of local transhumance, with livestock based in the villages of the plain and ascending the mountain pastures in summer, or, in contrast, with sheep based in mountain villages in the summer that come down during the winter onto lowland pastures. Large seasonal movements may comprise regional or inter-regional trips, sometimes with double seasonal migration, and may extend from the winter pastures in south Turkey at the border

with Syria to the summer pastures across the country and along the border with Iran, Armenia and Georgia (Figure 16).



Source: (Thevenin, 2011)

**Figure 16:** Large seasonal animal movements in south-eastern Turkey

It is possible that such long-distance movements of sheep and goats may have repercussions in the spreading of disease throughout the countries and across borders. Therefore, since these farming systems should be supported for the economic, environmental and political reasons explained above, proper veterinary care and improved surveillance should be in place for transhumant and nomadic farmers along the migratory routes.

### 3.7. Socio-political drivers

Times of political unrest could increase the potential risk of transboundary livestock diseases spreading into other countries, particularly those that border the affected countries. This is mainly driven by disruption of veterinary and public health services and of trade and movement routes, insecurity, massive displacement of refugees across borders and/or internally displaced people, and impeded access to pasture, water and feeds. The situation in the Middle East with the current civil war in Syria and the recent crisis in Iraq, and the instability in Ukraine, is a relevant example of such crisis that may contribute to the potential spread of livestock disease across EU borders.

A mission report by FAO (FAO, 2013) provides useful insights about the impact of the Syrian crisis on the livestock sector.

The Syria crisis has compounded the already difficult economic situation in the majority of Syria's neighbouring countries (i.e. Egypt, Iraq, Jordan, Lebanon and Turkey). Exports, tourism and transportation have all been negatively affected by the interruption of trade routes and the deterioration



of regional and national security. The Syria crisis is affecting all sectors in neighbouring countries, but its impact on the agriculture and food sectors is particularly important, as this is the main source of income for a significant proportion of the population, particularly for the poorest and most vulnerable communities in rural areas.

The Syrian veterinary services collapsed in 2012. Uncontrolled livestock movements have increased significantly, and Turkey is the only country with a relatively strict border control system with the slaughtering of all non-registered animals. Nevertheless, this remains a challenge owing to the 900-km long border shared with Syria. Animals are crossing into Turkey, with cases of PPR, bovine tuberculosis and brucellosis reportedly confirmed in captured animals. In Gaziantep Province in 2012, 13 cases of rabies were reported at the border with Syria, which have never been observed before. Foot-and-mouth disease, PPR, bluetongue, brucellosis and lumpy skin disease in animals and cutaneous leishmaniasis and tuberculosis in refugee camps have all been unofficially reported in the Syrian border areas of Iraq, Jordan and Lebanon (FAO, 2013).

Unvaccinated live animals are being legally imported or are illegally crossing into Iraq, Jordan and Lebanon, sometimes without quarantine, for sale on the open market and to slaughterhouses throughout those countries (e.g. FAO reported that 300 000 goats were illegally imported from Syria into Jordan in 2012). Moreover, nomadic Bedouins and agro-pastoralists from the Syrian border areas Iraq, Jordan and Lebanon can no longer access free or subsidised Syrian vaccines and animal feeds, and the disruption of traditional transhumance routes has led to overgrazing, land degradation and animal suffering and concentration, thus increasing the risk of disease transmission (FAO, 2013).

The UN Refugee Agency UNHCR estimated that 6.5 million people had been displaced in Syria, while more than 3 million refugees had fled to countries such as Lebanon (1.14 million), Jordan (608 000) and Turkey (815 000) since the start of the Syria crisis (UNHCR communication, 29th August 2014). This massive immigration of refugees, both legal and illegal, may represent a risk of both human and animal disease transmission, by refugees being potentially active or passive carriers of pathogens.

A further impact of the Syria crisis on animal and human health is the safety and quality of animal feeds and animal source foods. Illegal trade of unsafe foods and animal feeds is being practised owing to the disruption of regulatory systems, border inspection posts and law enforcements in Syria and is possibly the case in neighbouring countries owing to insecurity in border areas.

#### **4. Possible pathways of introduction of PPR into the EU and ranking on the basis of their level of risk, with a view to enhancing preparedness and prevention**

##### **4.1. Data and methodologies**

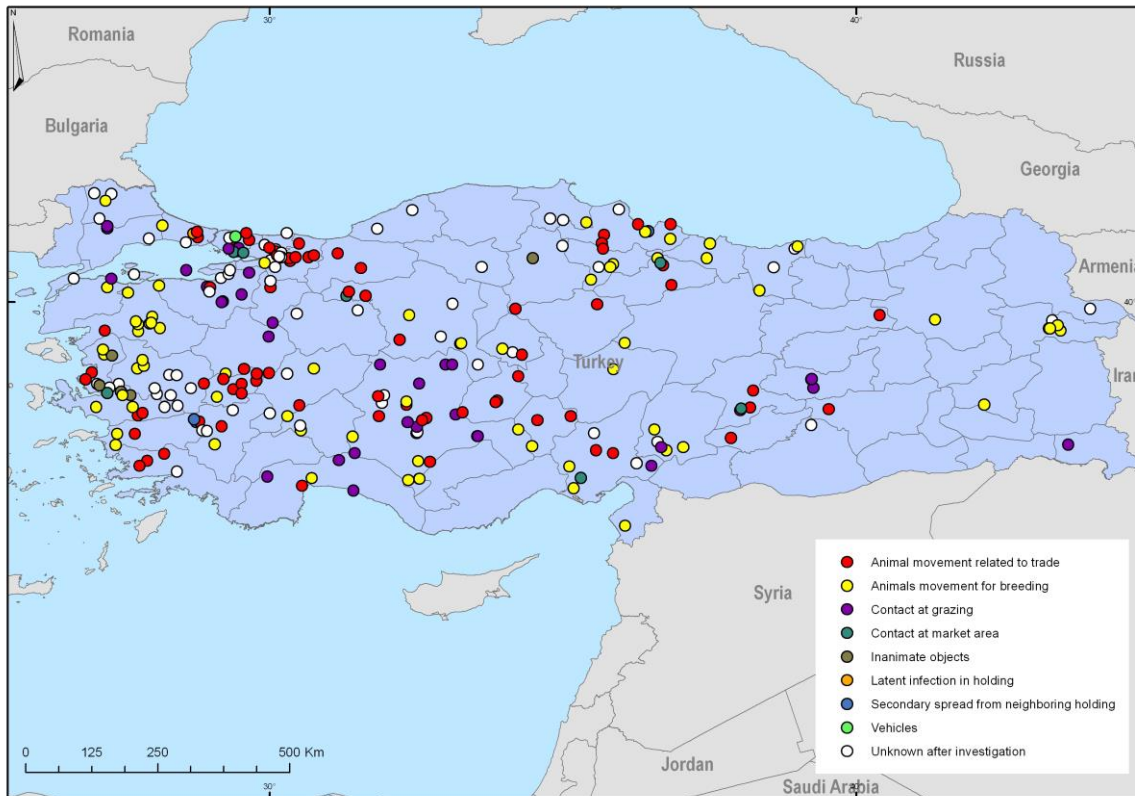
The main potential pathways of introduction of PPR from endemic countries into the EU are identified on the basis of the possible ways of transmission and virus survival in various matrices (see section 1.1.2), literature evidence, epidemiological information reported to OIE and the Animal Disease Notification System (ADNS), expert knowledge and reports of affected countries based on field evidence.

##### **4.1.1. Introduction of PPR through live animals**

Council Directive 92/65/EEC of 13 July 1992 laying down animal health requirements governing trade in and imports into the Community of animals, semen, ova and embryos identifies PPR as a notifiable disease and requires that trade in Bovidae, including small ruminants, and their products be subject to specific health requirements. Owing to the measures in force at the EU borders for trade in animals and animal products, any introduction of PPRV into the EU in live animals can only be directly or indirectly illegal, and this may occur because customs controls can never be exhaustive. The precise quantification of attempted smuggling of small ruminants is not available. However, it is possible that live small ruminants for domestic consumption could be smuggled into Europe by vehicles (Miller et al., 2009).

According to the outbreak investigation conducted in countries where PPR occurred in 2014 (OIE reports of the immediate notifications and follow-up of the PPR outbreaks in 2014 in Angola, Bhutan, China, Comoros, Democratic Republic of the Congo, Kenya, Mali, Tajikistan and Uganda, see section 2.2), the epidemiological information reported there showed the introduction of live animals (e.g. illegal movement of animals, contact at grazing or watering) as the most probable introduction pathway for PPR.

From the outbreak notification to the ADNS system, information was reported about the origin of some of the PPR outbreaks that have occurred since 2006 up to 2014. The information provided to ADNS is reported by central veterinary structures at the national level, which collect the information from veterinarians who work at the provincial and district levels. In Figure 17, the location of the outbreaks according to their presumed source is displayed.



Data source: ADNS

**Figure 17:** Outbreaks of PPR in Turkey displayed according to presumed disease origin

From the map shown in Figure 17, the most frequent source of outbreaks appears to be related to the movement of animals (red and yellow dots).

In most cases, animal dealers or the purchase of infected animals were considered to be the source of the infection. In Turkey, there are animal dealers who collect and transport animals from farm to farm, sometimes during the incubation period of disease. This happens in particular during the lambing season and before the period of Eid al-Adha, when amounts of live animals are moved from the areas of rearing, e.g. central and eastern Turkey, towards the bigger urban centres.

Finally, according to the answers provided in the questionnaire distributed to the REMESA network, the possible introduction of PPR into Algeria and Tunisia is mostly related to transboundary uncontrolled movement of infected animals (see section 2.1).

#### 4.1.2. Introduction of PPR through animal products

For meat or meat products to serve as vehicles for the introduction of animal disease, a number of criteria must be met: (i) the disease must be present in the country of origin; (ii) the disease must be present in the particular animal slaughtered (or the carcass must have become contaminated during the butchering process); (iii) the diseased meat must pass inspection procedures, unless illegal slaughtering takes place; (iv) the pathogen in the meat must survive storage and processing and be present at an infectious dose; and (v) the pathogen must be present in the tissues to be shipped. After introduction, in order to infect further animals, the infected products should enter in contact with susceptible animals. The same steps are valid for dairy products.

In order to be a source of PPRV, a product of animal origin should come from an animal that was infected and that had a period of viraemia allowing contamination of tissues, and the PPRV should

survive the treatment that the product undergo. A previous risk assessment performed in France considered achieving all of these events unlikely (Miller et al., 2009). Furthermore, for the further spread of the disease, the infected meat should come into contact with susceptible sheep and goats, which is also unlikely.

The legal transport of products of animal origin for human consumption to the EU should fulfil a series of requirements that make the risk of slaughtering a PPR-infected animal that is to be shipped to the EU extremely low. According to EU regulations, PPR is included in the list of diseases of relevance to trade of products of animal origin and for which control measures have been introduced under Community legislation, as in Directive 2002/99/EC<sup>9</sup> laying down the animal health rules for the production, processing, distribution and introduction of products of animal origin for human consumption. In particular, products of animal origin for human consumption that are obtained from animals which do not come from territories subject to animal health restrictions regarding PPR, in the case of meat and meat products, should not be slaughtered in an establishment in which there are infected animals or animals suspected of being infected with PPR. Alternatively, a list of heat-based treatments is provided in the same Directive to eliminate PPRV in meat and milk.

Nevertheless, for illegally carried products of animal origin, the situation may be different, particularly if PPRV is carried intentionally, e.g. for bioterrorism purposes. There are few controls regarding checked baggage of passengers, and so the amount of products of animal origin imported illegally remains high (see section 3.5.2). In addition, the Food and Veterinary Office (FVO) has repeatedly pointed out the lack of awareness among travellers to the risks involved in the introduction of animal products. A lack of coordination between customs and veterinary services is also identified as a weakness of the system of import controls (FVO, 2006). Regarding the survival of PPRV to processing and storage of, for example, meat, as explained in section 1.1.3, PPRV may survive in fresh or chilled meat for some days, although the pH values of meat after maturation (5.6–5.8) are not optimal for PPRV survival. In the case of frozen or salted meat, it is reported for similarity that RPV can remain infective for several months if frozen before the pH drop of rigor mortis (MacDiarmid and Thompson, 1997), although this is not a condition that normally occurs in the processing of lamb or mutton meat, and still needs to be proven for PPRV. This evidence is mainly extrapolated from RPV behaviour, and there is no clear evidence about PPRV survival in meat; therefore, real-scale experiments (in slaughtering plants) should be conducted on this. Regarding PPRV survival in milk or dairy products obtained from infected animals, no information is available.

In conclusion, the introduction of PPRV into the EU through infected animal products may potentially occur, in particular when illegally or intentionally carried to spread the virus (e.g. bioterrorism), although the risk is low and the further spread of PPR via this route is unlikely.

#### **4.1.3. Introduction of PPR through fomites**

The introduction of PPRV is theoretically possible by vehicles that transport livestock being contaminated with any infectious material. In the EU, these pathways may occur when livestock trucks return to the EU after the delivery of animals in infected areas or farms. Exports of small ruminants from the EU to PPR-infected areas exist, e.g. to African countries (see section 2), such as from Spain to Northern Africa. On the way back to the EU, the vehicles could be PPRV carriers if no proper biosecurity measures are in place (e.g. cleaning and disinfection, in particular of livestock vehicles and

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<sup>9</sup> Council Directive 2002/99/EC of 16 December 2002 laying down the animal health rules governing the production, processing, distribution and introduction of products of animal origin for human consumption addresses all stages of the production, processing and distribution within the Union and the introduction from third countries of products of animal origin intended for human consumption.

vessels, as in EC Decision 2014/689<sup>10</sup>), as the virus could survive for one to three days (data reported for RPV, not for PPRV; see section 1.1.3 of the present opinion).

#### **4.2. Pathway ranking**

From the discussion above, the different pathways of PPR introduction can be ranked in the following order:

1. The introduction of infected sheep and goats is largely the most efficient pathway to introduce PPR into a country. For the EU, this would occur mainly by illegal transport of infected animals (e.g. carried in private vehicles).
2. The introduction of PPRV into the EU through infected animal products may occur, in particular for meat or meat products when illegally and intentionally carried (e.g. bioterrorism), although the risk is low and the further spread of PPR via this route is unlikely.
3. Less likely is the introduction of PPRV via fomites into the EU, which may occur when vehicles carrying livestock return to the EU after the delivery of animals in infected areas or farms and where no biosecurity measures are applied.

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<sup>10</sup> Commission Implementing Decision of 29 September 2014 on measures to prevent the introduction into the Union of the foot-and-mouth disease virus from Algeria, Libya, Morocco and Tunisia. OJ L 287/27.

## 5. Risk of introduction of PPR into the EU through illegal import of animals

A quantitative approach to estimate the likelihood of introduction of an infectious disease agent into a disease-free country through the movement of animals is essential to assess the risk of introduction of such a disease agent. The movement of animals has been considered to be the main risk factor for the introduction of several infectious diseases into disease-free areas.

The European legislation identifies PPR as a notifiable disease and requires that the trade of Bovidae and their products be subject to specific health requirements. Moreover EU legislation sets a series of animal health rules governing the production, processing, distribution and introduction of products of animal origin for human consumption and addresses all stages of production, processing and distribution within the EU and the introduction from third countries of products of animal origin intended for human consumption. Therefore, an introduction of PPR by live animals into the EU would be possible only through illegal import.

The approach followed here is to establish the likelihood of introduction of PPR into Europe and is based on probability theory as reported in a previous EFSA scientific report (EFSA, 2012). The objective of the assessment is to propose an approach to assess the risk of introduction of PPR into a free area via import. In particular, this approach should help in the evaluation of the likelihood of the introduction of PPR. The approach should focus on the relevant animal species that could be potentially introduced into Europe.

In order to estimate the probability of introduction into the EU via the illegal movement of animals, it was considered that no test are applied to the animal moved; thus, using the binomial distribution, the probability that all animals are free of PPR in a shipment of size  $N$ , where  $\rho$  is the probability of being infectious, will be:

$$P(x = 0) = \binom{n}{0} \rho^0 (1 - \rho)^{N-0} = (1 - \rho)^N \quad (1)$$

The intention is to know if at least one is PPR infectious, rather than to know the probability that all moved animals are PPR free; thus, it is of interest to calculate the following probability:

$$P(x > 0) = P(x \geq 1) = 1 - P(x = 0) = 1 - (1 - \rho)^N \quad (2)$$

On the other hand, it is of interest to estimate the prevalence of infectious animals in a population with a reported seroprevalence. Considering a Susceptible Infected Removed (SIR) model to describe disease transmission, it is possible to establish a relationship between infectious prevalence and seroprevalence at equilibrium. The relationship is derived from the following differential equation:

$$\frac{dR}{dt} = \text{RecoveryRate} \times I - \text{InverseDurationImmunity} * R \quad (3)$$

where  $R$  and  $I$  refer to the recovered and infectious cases, respectively, and

$$\text{RecoveryRate} = \frac{1}{\text{Mean Infectious Period}}$$

The mean infectious period was considered to be 14 days and

$$\text{InverseDurationImmunity} = \frac{1}{\text{Mean Duration of Immunity}}$$

which in this case is considered to be the lifespan of the host (two years), but shorter or longer periods might be considered if needed. Hence, at equilibrium:



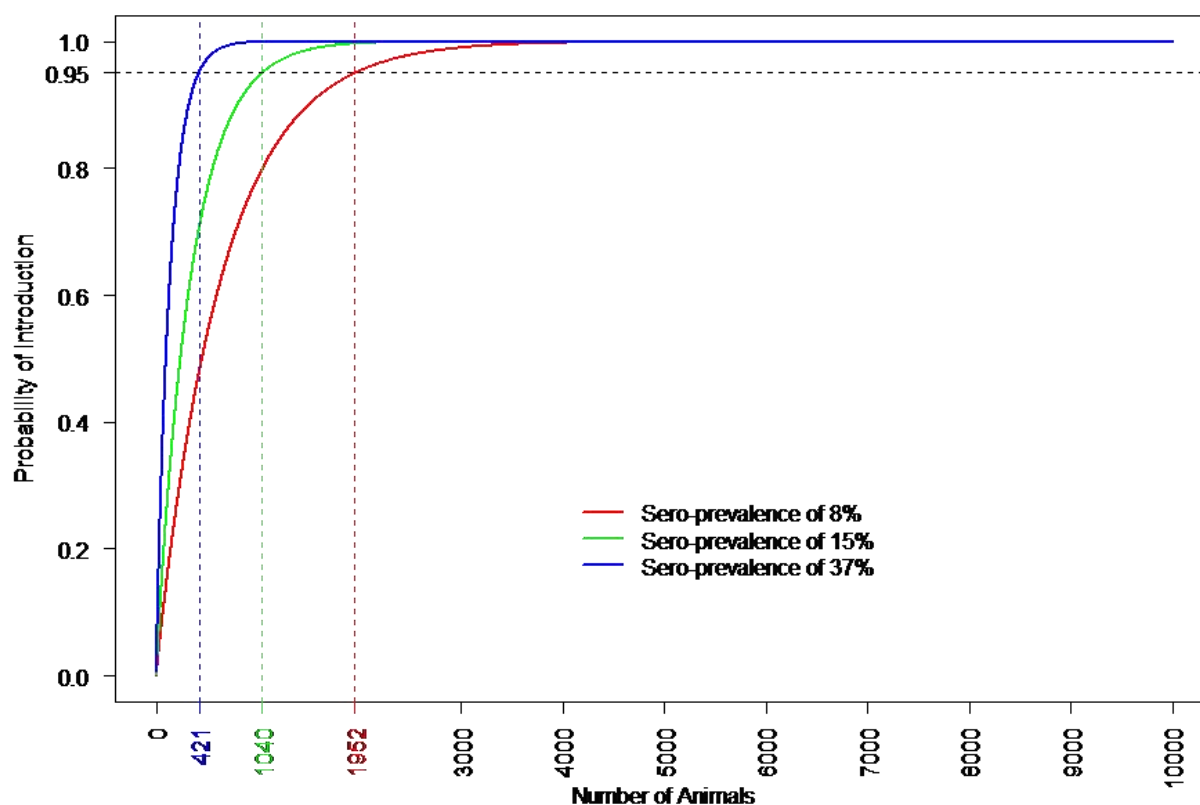
$$I = \frac{\text{Mean Infectious Period} \times \text{Sero-Prevalence}}{\text{Mean Duration of Immunity}} \quad (4)$$

In order to estimate the probability of introduction, equation (4) should be inserted into equation (2) as follows:

$$P(x > 0) = 1 - \left(1 - \frac{\text{Mean Infectious Period} \times \text{Seroprevalence}}{\text{Mean Duration of Immunity}}\right)^N \quad (5)$$

The probability of the introduction of PPR into the EU could be then calculated using equation (5) for different levels of seroprevalence in the area of origin (0.08, 0.15 and 0.37), including different numbers of illegal animals moved into the EU (1 to 10 000).

The results obtained are presented in Figure 18. If seroprevalence is 37 %, the number of animals to be moved so that the probability of introduction is greater than 0.95 or lower than 0.05 to introduce PPR into Europe would be 421 and 8, respectively. On the other hand, if seroprevalence is 8 %, the number of animal needed to be moved would be 1 952 and 34, respectively.



**Figure 18:** Probability of introduction for different seroprevalence levels as well as the number of animals moved

## **6. Speed of propagation of PPR**

Since data regarding the transmission of PPR are limited, and field data on transmission in European or comparable conditions are also lacking, the evaluation of the speed of propagation needs to be restricted to an evaluation of propagation under very different conditions followed by a discussion on how this could be translated into a European setting.

Suitable data on transmission outside of Europe is also limited, because vaccination is often applied, and thus transmission under non-vaccinated conditions cannot be derived from these sources. Data are available from a few African countries and are used to derive estimates on transmission rates and the spatial scale of spread.

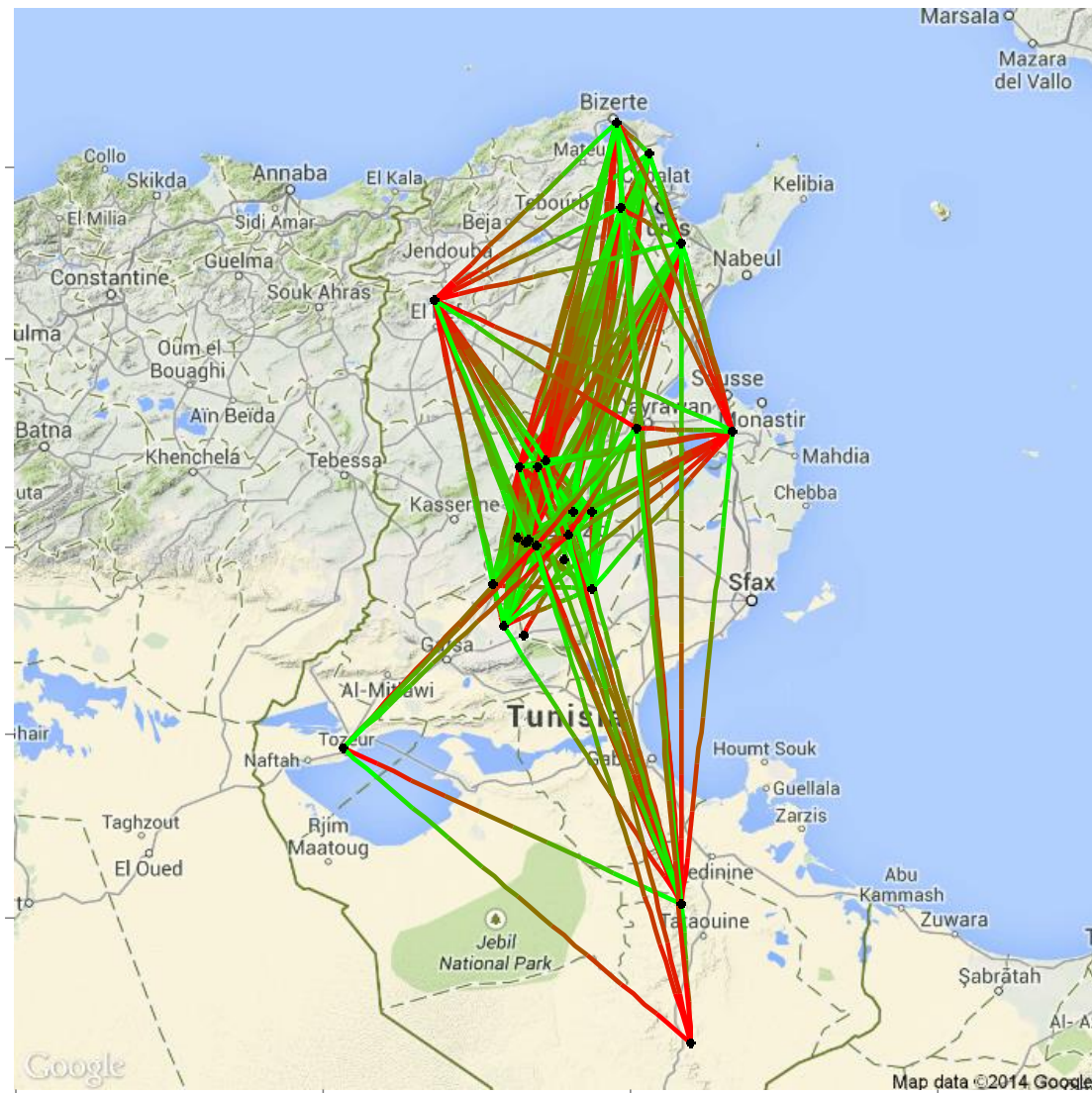
Tunisia was chosen as a case study because vaccination was not applied and the population could be considered mostly PPR naive. Moreover, in some aspects, the climate and farming systems are similar to those in some southern European countries.

Furthermore, data from Senegal have been evaluated to derive a within-herd estimate of the basic reproduction ratio of infection. Again, this concerns unvaccinated herds that experienced a major epidemic outbreak, suggesting the population was mostly naive before the outbreak.

### **6.1. Case study: Tunisia**

Outbreak data of PPR in Tunisia as reported to OIE (WAHID) were used to plot temporal and spatial links between outbreaks and to derive an estimate of the potential ranges of speed of propagation.

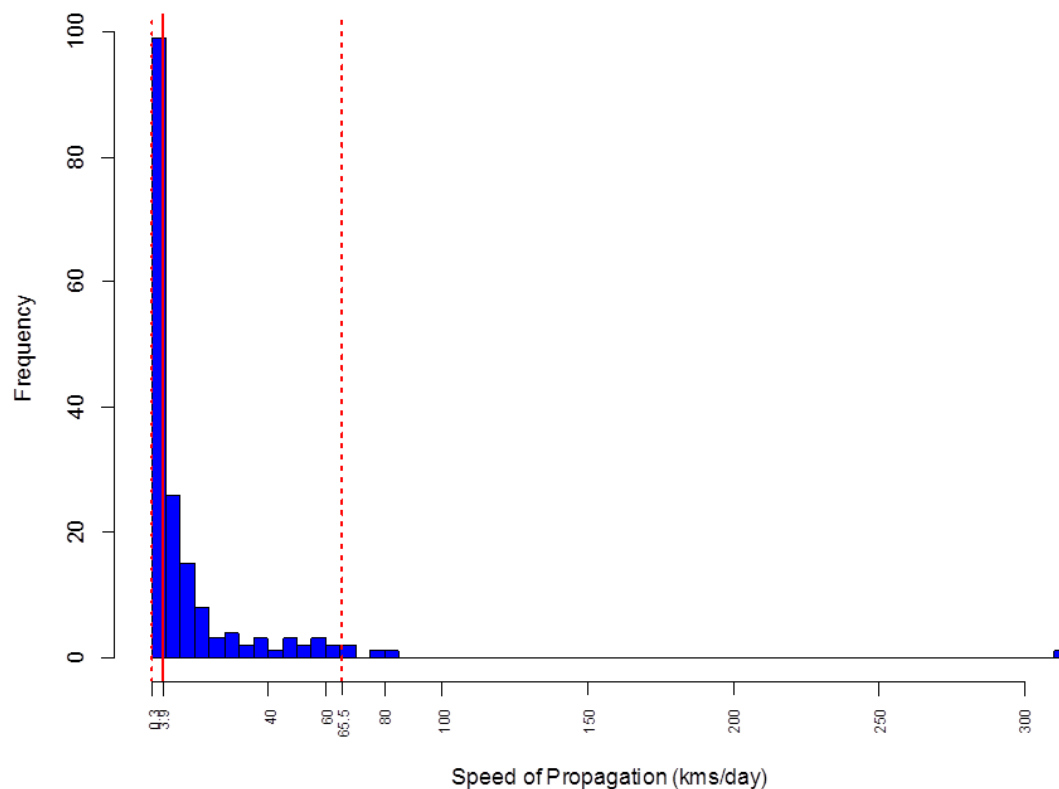
Figure 19 shows the scenario in which each outbreak, sorted by time, could cause any of the other subsequent outbreaks that occur up to three months later. The underlying assumption here is that transboundary movement of infected animals is not a possible source of the outbreaks. The reason for this assumption is that there are no available data about these movements, although these may be the most likely source of introduction of PPR.



The gradient of colours from green to red indicates potential sources for subsequent outbreaks, considering that they have occurred up to three months after their reporting, and accounting for potential delays in reporting and diseases epidemiology

**Figure 19:** Flow diagram showing links between outbreaks sorted by time that could cause any of the other subsequent outbreaks that occur up to three months later

The histogram of the speed of propagation derived from the spatial pattern of outbreaks is given in Figure 20, with the vertical lines representing the lower, median and upper bounds for a 95 % CI. The median speed of propagation was estimated to be 3.9 km/day, with a 95 % CI of 0.3 to 65.5 km/day. These results should be interpreted with caution and should be seen as indicative without direct extrapolation to the whole of the EU situation.



**Figure 20:** Median, lower and upper bounds (95 % CI) of the speed of propagation of PPR, using outbreak data from Tunisia in 2012

Translating the histogram of the speed of propagation into the speed of a spatially moving epidemic wave leads to a pattern where slower and frequent speed determines the overall speed of the wave, while incidental long-distance transmissions (from the tail of the histogram) can create new epidemics further away. Based on this pattern, for Tunisia, the speed of propagation of an epidemic would probably be below 10 km per day. Translating this pattern into a European situation is not straight forward and will depend on farm density and animal transports. In the European regions that are comparable to Tunisia, similar speeds of propagation of the infection can be expected. Generally, in Europe, control measures are expected to isolate local infection sources and monitor the area around the source. In such conditions, rare long-distance transmissions would be more important than the more common short-distance transmissions, because they may escape more intensive monitoring. The information from Tunisia should not be considered relevant for Europe directly, but can be used to estimate the speed and spatial scale at which infection can be expected to spread. The travelling frequency and distance of small ruminants in Europe should be determined and taken into account. While sheep and goats very frequently travel short distances, their transportation over longer distances may be less common in Europe than in Tunisia. If, however, a similar pattern would also apply to Europe, an epidemic of PPR might be hard to stop, as the spatial spread might be substantial, at the detection of the first outbreak. Such a situation might occur particularly in regions densely populated with sheep and with low goat densities, owing to longer delays in clinical detection in sheep, assuming that the local sheep breeds in Europe are also less susceptible to PPRV than goats, as appears to be the case in Northern Africa (Hammouchi et al., 2012).

## 6.2. Case study: Senegal

The case of Senegal represents a situation where no vaccination was implemented in the study area, and where information was available on epidemiological investigation that has been conducted by

national authorities in collaboration with CIRAD. Virological and serological data were collected in collaboration with the Directorate of Veterinary Services (DSV, Dakar), the National Veterinary Diagnostic and Research Laboratory (ISRA-LNERV, Dakar) and CIRAD.

Data were collected in PPR outbreaks observed in goat herds that were initially found positive using RT-PCR.

Following this initial outbreak survey, a subsequent farm visit was organised two to three months later, i.e. at the end of the epidemic phase of PPRV transmission. The assumption of fade-out due to depletion of susceptible animals at the time of this second visit looked realistic. Obvious clinical cases in the animals were not observed. Moreover, neighbouring farms experienced PPR outbreaks during the following months, indicating that PPRV was still circulating in the neighbourhood. During this second visit, goats older than one year were randomly sampled in each herd, with blood taken from the jugular vein. Sera were tested with ELISA. Results are listed below in Table 3.

**Table 3:** Seropositivity to PPR by ELISA in goats in Senegal randomly sampled in 12 herds

<i>ID of farm</i>	<i>Sample size</i>	<i>Positive ELISA results</i>	<i>Latitude</i>	<i>Longitude</i>	<i>Community</i>
101	13	11	14.1301	-13.2313	Mba Sam
102	9	8	14.1301	-13.2313	Mba Sam
103	6	6	14.1301	-13.2313	Mba Sam
106	10	9	14.0385	-16.2303	Ndiatane
107	10	10	14.0813	-16.217	Ndiafate
202	7	7	14.0787	-16.4815	Soum
205	8	8	14.0567	-16.34	Keur Moda
206	8	8	14.0567	-16.34	Keur Moda
301	10	10	15.0754	-16.6	Sakh
305	7	5	15.6149	-16.2449	Louga
306	7	7	15.6149	-16.2449	Louga
401	10	10	13.6864	-14.0426	Sare Houro

Because farming systems, animal phenotype and animal density were very similar in these sampled herds, the data can be pooled to get a single estimated  $R_0$ . Of course, this estimate only makes sense in the specific context of goat farming in this area of Senegal.

De Koeijer et al. (1998) derives a method to estimate the basic reproduction number  $R_0$  for infections such as PPR, i.e. with high mortality, in animal species that live in herds:

$$R_0 = (1 - f)\ln(y)/\ln(x)$$

where  $f$  is the mortality of infected animals (attack rate),  $y$  is the fraction of the initial population that escapes from infection and  $x$  is the fraction of the population that survives the epidemic.

This formula can be used with an attack rate estimate for mortality in infected goats, which was estimated as 33 % (Grech-Angelini, 2012). Using the pooled seroprevalence data, 99 out of 105 of the sampled goats tested positive, and 6 were negative. Thus, it can be expected that, for every 105 animals that survived the epidemic, a total of  $99/(1 - f) = 148$  animals had initially become infected and, of these 148 animals, 49 died ( $99 * f/(1 - f)$ ).

Given:

$$x = 105/148 = 0.71$$

$$y = 6/148 = 0.039$$

$$\ln(x) = -0.38 \text{ and } \ln(y) = -3.24,$$

$$R_0 = 6.3$$

These results confirm that the within-herd transmission of PPR is very high, and an epidemic outbreak will lead to infection of most animals in a herd. This is in agreement with other epidemiological studies about PPR in Tanzania and Pakistan, where the estimated value of  $R_0$  was 6.8 and 4.0, respectively (Zahur et al., 2009; Kivaria et al., 2013).

Owing to the high within-herd transmission, the between-herd transmission will also be difficult to control, as this is affected by the infectiousness in the herd, in combination with the contact rate to other herds. The latter is very variable within Europe. Common grazing is a major risk source and should be avoided when PPR is detected in the “neighbourhood”.



## **7. Risk of PPR becoming endemic in the animal population in the EU**

Endemicity of PPR is the long-term persistence of the infection in an area (i.e. for several years). Long-term presence of an infection in an (already) affected area can occur if new susceptible animals arise prior to infection fade-out in that area. Therefore, long-term persistence requires infectiousness to be maintained for a sufficiently long time along the infection chain of multiple hosts or delayed/intermittent infectiousness of individual infected hosts or external reservoirs.

Therefore, endemicity can be enhanced by (i) a very long latent or long infectious period (i.e. carriers, reactivation), (ii) persistence of the infection in a reservoir population other than the one at risk (often wildlife and often with mild clinical disease) or (iii) persistence of the infectious agent itself in the environment (including biological vectors).

For actively controlled infections, a fourth situation may occur where fast spread of an infection in the high-risk period before detection in that area is followed by reduction of the infection owing to control measures. For such infections, prolonged perpetuation of the infection can occur either as a result of insufficient control and disinfection in the affected area or because of a high frequency of “spatial escape” from the control region, i.e. long-distance transmission, which escapes from the tracing efforts.

PPR shows strong epidemic behaviour followed by fade-out. Depending on the level of spatial spread, the infection can move between herds and in space, and can return to previously infected areas when the susceptibility has increased sufficiently. In areas with very large numbers of herds, the infection is not sufficiently fast for purely epidemic behaviour and endemicity can be expected without the use of control measures. Specific persistence mechanisms are not known for PPR. The fourth situation is the only one that is really relevant for PPR in Europe. If the long-distance transmissions (see also section 6) before detection are sufficiently frequent, the infection might persist that way. If this is indeed the case, it can be solved by (if possible) more efforts put into the tracing of potentially infectious contacts before detection of the epidemic and by using a larger surveillance zone.

For PPR, there is no data available which is sufficiently comparable and suitable for extrapolation to the EU situation. Thus, beyond these qualitative appreciations, it is not possible to make a quantitative assessment of the risk of endemicity. For PPR, data regarding transmission from Africa are too limited to properly evaluate the situation to date. An attempt at gathering and evaluating such data should be supported (financially) to obtain better data. Extrapolation of such information should be realistic, but it would take too much time to fit within the frame of this opinion.

If they occur, epidemics within the EU should be closely documented and data should be collected to enable the evaluation of the probabilities of these potential risks of the persistence of the infection. This information can subsequently be used to evaluate the need for extended control measures. The required control measures may differ between regions owing to animal and herd densities, but also to local animal husbandry practices.

## 8. Impact of PPR in endemic and free areas

### 8.1. Impact in endemic areas

PPR is particularly a constraint in less developed areas where livelihoods highly depend on livestock, and especially on sheep and goats, for their survival (FAO, 2013). Sheep and particularly goats contribute significantly to the nutrition and cash income of small farmers in Africa and South Asia, the two regions of the world with the largest concentration (about 72.90 %) of people living in poverty (Alcigir et al., 1996; Tatar and Alkan, 1999; Guler et al., 2014). The International Livestock Research Institute (ILRI), Nairobi, Kenya, has identified PPR as one of the priority animal diseases whose control should be considered for poverty alleviation in Western Africa and South Asia, and this highlights the economic importance of PPR (Alcigir et al., 1996; Tatar and Alkan, 1999; Guler et al., 2014).

PPR is still a poorly recognised disease, particularly with regard to epidemiological features such as transmission dynamics under different production systems. A great deal more research into this aspect of the disease is urgently required (Perry et al., 2002).

After the successful global rinderpest eradication programme in cattle, national and international organisations, such as FAO and OIE, have started important initiatives to control PPR (see section 9.5).

According to FAO estimates, the morbidity, mortality, production losses and treatment costs of PPR altogether are likely to cause an economic loss of around USD 2.9 billion/year during 2012–2017 in the South Asian Association for Regional Cooperation (SAARC) region and, of these countries, in India alone it would be USD 2.5 billion/year (Yener et al., 2004). FAO estimated that the global small ruminant population is about 2 billion animals, 71.5 % (1.45 billion) of which are at risk of PPR.<sup>11</sup> If the vaccination cost per animal was around USD 0.3, in the worst-case scenario of a three-year mass vaccination (and being sure to cover the naïve replaced animals for a further two years), a total of around 4.35 billion vaccine doses would be required at the cost of USD 1.3 billion.

#### 8.1.1. Impact in Turkey

Concerning the situation in Turkey, as a result of aetiological, serological, pathological and immunopathological studies done between 1994 and 2009 in Turkey, it was determined that PPR disease is endemic in Turkey (Ozkul et al., 2002; Gulyaz and Ozkul, 2005; Albayrak and Alkan, 2009; Albayrak and Gür, 2010). According to these studies, PPR prevalence was found to be 28 %, morbidity was found to be 80–100 % and mortality was found to reach up to 80 % in relation to age of infected animals. The payment for compensation is fixed at EUR 170 for adult animals and EUR 68 for young animals. The cost calculation for vaccines and vaccination in Turkey is EUR 8.5/100 heads for the vaccine cost, EUR 5.1/100 heads per day and for transport and EUR 1.7/100 heads for farmers, coming to a total of EUR 15.3/100 heads.

#### 8.1.2. Senegal

A study was performed to estimate the decrease of goats herds' productivity, and therefore of profitability, due to the disease in Senegal (Grech-Angelini, 2012). A retrospective cohort including 27 goat herds was reconstituted and studied over a period of 12 months. From this cohort, 12 herds were exposed to the PPRV. With this retrospective approach, which is based on farmers' interviews, demographical and zootechnical data were gathered to compare the productivity of the two groups. Exposed herds have seen their mortality rate multiplied by three. Females from the exposed group underwent four times more abortions and the rates of parturition and fecundity dropped by 30 %. At the same time, the off-take rate of animals, from which farmers' revenues depend on, decreased by 50 %. This study is the first to quantify the effect of PPR on the productivity of livestock. Despite

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<sup>11</sup> FAOSTAT.

some biases resulting from the retrospective method, it seems clear that, in areas affected by the disease, profitability coming from goat breeding has been reduced to almost nil.

The impact of PPR, which is still recognised as the biggest killer of flocks of small ruminants in Senegal, had never been studied in any country where it occurs. The retrospective survey in Senegal is the first to quantify the actual losses due to exposure to PPRV. The results showed that PPR heavily drove down productivity and thus reduced the farming profitability to virtually zero. As goats are often a secondary source of income for rural Senegalese populations, any change in that income has important implications. For other countries, where the goat population is central to the socio-economic life of the people (e.g. in the Democratic Republic of the Congo), PPR is similar to a natural disaster threatening the food security of the population. Therefore, the results of the study aimed to encourage policy makers and economic operators to invest in the vaccination of livestock and also to impress upon recalcitrant farmers the utility of this vaccination. With vaccination coverage of less than 30 % and without a collective awareness, Senegal may be not ready to control PPR.

In the medium term, the different rates calculated in this study should contribute to the construction of a transmission model of PPR in Senegal type SIR (susceptible, infected, removed). This model should include parameters of sheep populations and genetic data on the virus strain circulating in Senegal. This would allow a better understanding to be gained of the epidemiology of PPR in Senegal.

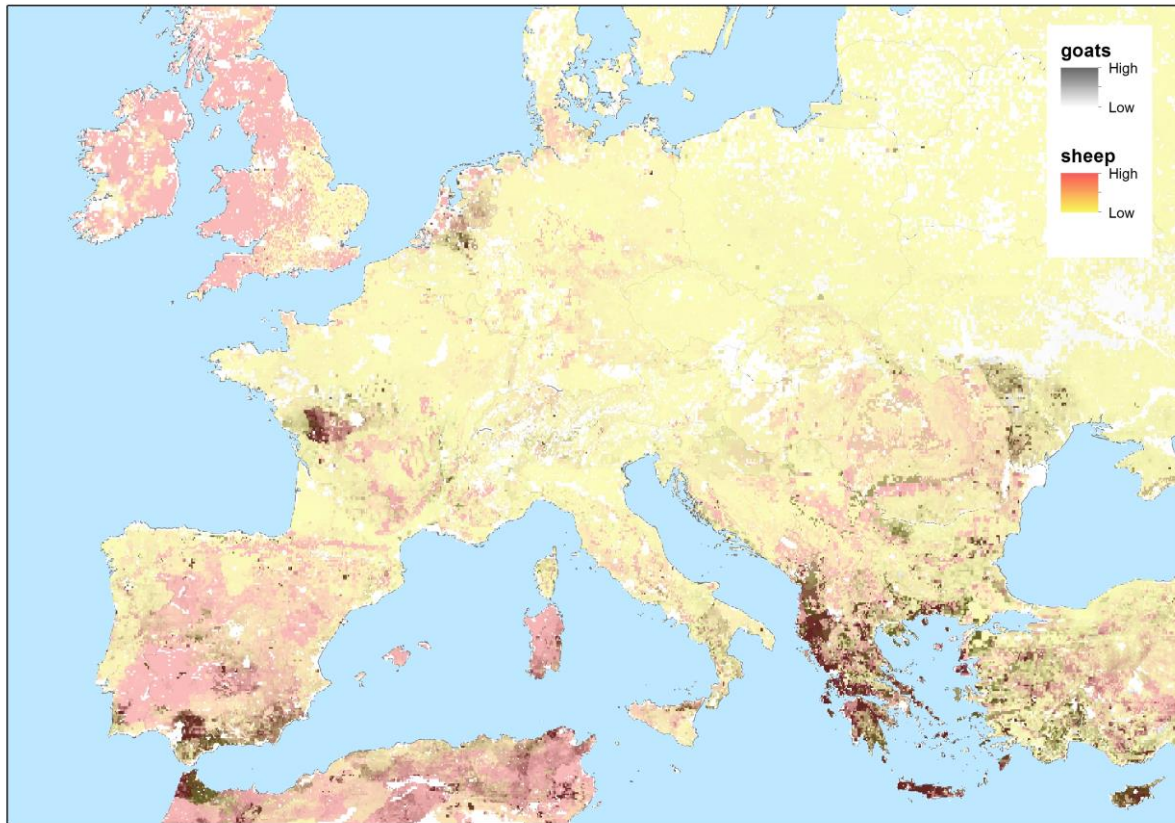
## **8.2. Impact in free areas**

From worldwide experience in endemic areas, it can be assumed that European goats would be more susceptible than sheep. Indications of this come from exotic (alpine) goats breeds in Morocco, including an experimental infection study (El Harrak et al., 2012; Hammouchi et al., 2012). In addition, recently, an experimental infection study was conducted by Wernike et al. (2014), in which goats of German breeds infected with a Kurdish PPRV isolate showed more severe symptoms than sheep.

In some European countries (e.g. France), there are goat feedlots that contain hundreds or thousands of goats, especially kids. The impact of the potential PPR introduction in such feedlots would be enormous.

Moreover, sheep are highly mobile (via trade) in Europe. Silent or semi-silent PPR transmission in sheep, like in Morocco, might result in widespread infection within weeks and thus heavy losses or at least economic costs (stamping out). This occurred in Morocco, where the level of genetic selection in sheep breeds is not high: the situation was of low cumulative incidence, and there was a moderate fatality rate, leading to several months before PPR was diagnosed. In that situation, the sheep population was fully naive to PPRV and, thus, had no previous immunity.

There might be a risk that, if PPR enters areas in the EU with dense sheep populations but low goat densities, it would start circulating and leading to widespread infection before being detected. Since European goat breeds are highly susceptible to PPRV (Hammouchi et al., 2012), this situation might occur in areas densely populated with sheep, and without important goat populations. Figure 21 shows that this population pattern occurs in several European areas, particularly in Great Britain and the Republic of Ireland.



Data source: FAO GeoNetwork

**Figure 21:** Sheep and goat densities in Europe

Therefore, it would be important to assess the susceptibility of major European sheep breeds to PPRV, for a better risk assessment. In addition, it might be important to check if European cattle breeds can be infected and if the virus can be eliminated.

## **9. Availability, effectiveness and feasibility of the main disease prevention and control measures**

### **9.1. Data and methodologies**

The information used in this section derives from a literature search, field evidence and expert knowledge.

Several studies for validating diagnostic tests for *Capripoxvirus* infections have been conducted and published. Experimental, retrospective and observational studies performed worldwide were identified for domestic small ruminants through mapping the collected evidence, and the extracted information on assay development and validation criteria are summarised in Appendix A.

### **9.2. Diagnostic tools**

#### **9.2.1. Clinical diagnosis**

The clinical disease resembles rinderpest in cattle. It is usually acute and characterised by pyrexia, serous ocular and nasal discharges, diarrhoea and pneumonia, and erosive lesions on different mucous membranes, particularly in the mouth.

Because of the respiratory signs, PPR can be confused with contagious caprine pleuropneumonia (CCPP) or pasteurellosis. In many cases, pasteurellosis is a secondary infection of PPR, a consequence of the immunosuppression that is induced by the PPRV (OIE, 2013).

#### **9.2.2. Laboratory techniques**

A literature review was performed in order to address the ToRs on the availability of diagnostic techniques for PPR (protocol in Appendix A). In the 243 papers selected for revision following the criteria listed in Appendix A (Study Eligibility Form), 41 diagnostic tests were named. Eight of them are detailed in the relevant chapter of the OIE manual which was recently revised in 2013, where the available tests for PPR infection are described (OIE, 2013). What follows here is an outline of the most used diagnostic tools.

The most basic test is agar gel immunodiffusion (AGID), which is a very simple and inexpensive test that can be performed even in the field. It is still in use in a number of laboratories in the developing world where they can generate their own test antisera.

More common are various forms of PCR and ELISA. There is only one commercially available immunocapture ELISA (Libeau et al., 1994). It is reliable test for the virus where local technology cannot perform molecular techniques, although it is not as sensitive as PCR. The test can detect 100.6 TCID<sub>50</sub> (the 50 % tissue culture infectious dose)/well of PPRV, and the results are obtained in approximately two hours.

Well-established gel-based PCRs using targets in the N gene (Couacy-Hymann et al., 2002) or F gene (Shaïla et al., 1996) are in common use, requiring no patented materials. A large number of real-time PCR methods have been published, one of which has been commercialised as a kit. The real-time assays are generally more sensitive than the gel-based assays.

A field test for PPRV has recently been developed at Pirbright Institute. This commercially available test is essentially a lateral flow device-formatted immunocapture test, and has been trialled in the field and found to be functional (submitted for publication).

For animals that recover from disease, the viral antigen or genome cannot usually be detected in samples more than two weeks after the initial rise in temperature. For the detection of antibodies of the virus, the test used is either AGID or competitive ELISA. Specific competitive ELISA techniques for



PPRV (Anderson and McKay, 1994; Libeau et al., 1995) have been developed and used in the field, and are commercially available.

### 9.2.3. Virus isolation and propagation

Primary lamb kidney cells (LK) have proven to be one of the best for isolating PPRV (Gilbert and Monnier, 1962; Taylor and Abegunde, 1979). However, because of the ease of use of Vero cells, many isolates of PPRV have first been propagated in these cells (Hamdy et al., 1976). Either LK cells or Vero cells are recommended for PPRV isolation (OIE, 2013). More recently, it has become preferable to use one of the signalling lymphocyte activation molecule (SLAM)-expressing cell lines available. The discovery that the highly conserved host cell membrane protein CD150, also known as SLAM, is the normal receptor for all kinds of morbilliviruses (Tatsuo et al., 2000; Tatsuo et al., 2001; Baron, 2005; Birch et al., 2013) prompted the creation of modified tissue culture lines expressing SLAM; these lines have proven to be the most useful for propagating wild-type PPRV isolates. The modified Vero line expressing canine SLAM has been superior to equivalent Vero cells expressing human or bovine SLAM, but any of these cells, or CV1 cells expressing goat SLAM, can be used (Adombi et al., 2011). Vero cells expressing the canine SLAM molecule (Vero/dog-SLAM) were generated by von Messling et al. (2003) and have also recently been used in an experimental infection by Wernike et al. (2014).

PPRV does not form plaques in the normally accepted sense of causing cell lysis or rapid cell death in tissue culture, so titration is usually carried out by measuring the TCID<sub>50</sub> (Anderson et al., 1996). Regions of infected cells can be observed in the light microscope by the cytopathic effect (cell rounding or distortion, syncytium formation).

## 9.3. Vaccines and vaccination

### 9.3.1. Vaccines

In the absence of homologous vaccines, and owing to the close relationship between the two viruses, the attenuated tissue culture rinderpest vaccine has been used for a long time to protect small ruminants against PPR. At the end of 1980s, a PPRV strain was successfully attenuated by serial passages into Vero cells (Diallo, 2002). In 1998, OIE endorsed the use of homologous vaccines in countries that have decided to follow the 'OIE pathway' for epidemiological surveillance for rinderpest in order to avoid confusion when serological surveys are performed.

Sheep and goats vaccinated with the attenuated strain of PPR or that recover from PPR develop an active immunity against the disease for at least three years (OIE, 2013). It is assumed that this protection is actually lifelong, as RPV vaccine was observed to protect cattle (a naturally longer living species) for up to 10 years (Plowright and Taylor, 1967). For this reason, animals that have recovered from PPRV infection are assumed to be protected from the disease for life, although there are no published studies on attempts to re-infect animals that have recovered from the disease.

After vaccination with standard PPRV vaccines, antibodies can be detected clearly at 14 days post vaccination (Baron, MD, unpublished). Full protection is achieved from standard doses of vaccine at three weeks post vaccination (OIE, 2013). There are no studies investigating if protection can be achieved earlier with higher doses of vaccine.

Homologous PPR vaccines are available, which are cell culture-attenuated strains of natural PPRV, such as the common Nigeria 75/1 strain, and have been used extensively in Africa and the Middle East to suppress outbreaks (Diallo et al., 1989; Sen et al., 2010). Currently, there are six available PPR vaccine strains: the PPRV Nigeria 75/1 (India, lineage II; isolate of goat origin); the PPR Sungri 96 (India, lineage IV; isolate of goat origin); Arasur 87 (India, lineage IV; isolate of sheep origin); Coimbatore 97 (India, lineage IV; isolate of goat origin); Titu (Bangladesh, lineage IV; isolate of goat origin); and 45G37/35-K PPR Vaccine (Kazakhstan, lineage IV).



The vaccine strain Nigeria 75/1 confers a strong immunity against all known PPRV genotypes (to date, a single serotype has been described). With the prescribed vaccine dose of  $10^{2.5}$  TCID<sub>50</sub>, a single injection confers an economic lifelong immunity (lasting at least five years).

The other similar PPRV vaccines developed in India (Sungri 96, Coimbatore 97 and Arasur 87) have been proven to be effective in terms of protection (Saravanan et al., 2010). In the study done by (Saravanan et al., 2010), the potency of these vaccines was tested in accordance with OIE guidelines by challenging 18 sheep and 32 goats, and their efficacies were evaluated using PPR competitive ELISA. The results showed that the sheep challenge virus was less potent than the goat virus; however, the challenge studies in sheep and goats with species-specific challenge viruses confirmed the efficacy of the vaccine in terms of the level of protection afforded.

A number of vaccine manufacturers in Africa and the Middle East produce vaccines based on the original Nigeria 75/1 vaccine, and their effectiveness has been tested in several studies (Diallo et al., 2007; Intizar et al., 2009; Saravanan et al., 2010). Several African, Middle Eastern and Asian (India, China) laboratories produce the PPR vaccine, although it should be submitted to external quality control before its use in vaccination campaigns.

An important point is that there is no indication of lineage-specific pathology or protection. The most commonly used PPRV vaccine is based on a lineage II isolate (Nigeria 75/1), but protects animals against all known isolates of PPRV, in all parts of the world in which it has so far been used (from Africa through to China).

Since only live, attenuated vaccines are available, this poses the question of their safety. Regarding the Nigeria 75/1 strain, reversion experiments were done. No reversion was observed after three back passages in animals (Diallo, 2014). Besides the lack of reversion, no side effect has been observed, whatever the physiological status of the host. In particular, no increased abortion rate was noticed in vaccinated pregnant ewes and goats during field experiments conducted in several African countries (more than 40 000 vaccinated animals, and 35 000 control animals). Moreover, after more than 25 years of use in field situations, including vaccination in PPR outbreaks, no incident was noticed. Indeed, the example of PPR control in Morocco is demonstrative of the safety and quality of this vaccine.

The available PPR vaccines do not support the DIVA principle. The only vaccines in use are attenuated forms of the virus, and there is no consistent difference in antibody responses to these viruses and wild-type forms of the virus. Possible DIVA vaccines based on recombinant viruses are good vaccine candidates. One vaccine based on recombinant adenoviruses has shown to be promising but has not undergone extensive or long-term testing (Herbert et al., 2014). Thermostable vaccines and other strategies for improving the stability of PPR vaccines are under study (Sarkar et al., 2003; Siddique et al., 2006; Riyesh et al., 2011; Silva et al., 2014). Preliminary results on recombinant capripox-based PPR vaccines indicate that they can protect against both capripox and PPR, but they are not yet validated for field use (Diallo et al., 2002; Berhe et al., 2003; Caufour et al., 2014).

Some research has been done on synthetic short interfering RNAs (siRNAs) which might kill the virus while preserving the serological status of treated animals (Servan de Almeida et al., 2007). As live attenuated vaccines cannot protect the animals before 14 days post vaccination, in emergencies, it would be desirable to have efficient therapeutics for virus control. However, these are still at the experimental level and are far from practical use.

Killed vaccines are not available and, owing to the immunological response to PPRV, would not be fully effective. This may also be the reason why no experiments for the development of PPR killed vaccines and for testing their protection have been done. Furthermore, from the experience of rinderpest eradication, it was shown that killed rinderpest preparations (e.g. heated blood or other body fluids from infected animals) were ineffective at giving protection. In addition, some data about the measles vaccines show that the inactivated measles vaccines, in addition to giving only transient

protection, could lead to increased virulence of subsequent infection (atypical disease in about 20 % of cases), which is one of the reasons why killed measles vaccines are not used.

### 9.3.2. Vaccination in endemic areas

In regions and countries where the disease is endemic in nature, the most commonly employed control method is controlling the disease by increasing the immunity level through extensive vaccination campaigns. Although different attenuated strains are in use in vaccination activities, the attenuated Nigeria 75/1 is the only vaccine strain approved by OIE.

Concerning the coverage needed, in the epidemiological study performed by Kivaria et al. (2013), the incursion, persistence and spread of the virus in Tanzania were investigated. The computed overall effective reproductive number was between 2.3 and 6.83, varying with the different study areas, which would correspond to a range of vaccination coverage between 56 and 85 %. This is in agreement with what was computed in the example in Senegal, analysed in section 6.2 of the present opinion ( $R_0 = 6.3$ , thus vaccination coverage = 84 %), and with what was found in Pakistan ( $R_0 = 6.83$ , vaccination coverage 85 %) (Zahur et al., 2009). This would mean that achieving elimination of the PPRV from flocks in those pastoralist conditions would require significant effort and development of highly effective intervention tools.

#### 9.3.2.1. Vaccination as performed in Turkey

Vaccine production is also performed with attenuated Nigeria 75/1 strain in Turkey. Owing to the variation in susceptibility to PPRV with environmental conditions, it is compulsory to secure the cold chain in the transfer and administration of vaccines. Animals vaccinated against PPR at the right age and under suitable conditions are reported to acquire an immunity period of four years, although the immunity acquired through the vaccine is regarded to be maintained for the whole course of an animal's lifetime. Despite the reports indicating that a single administration of the vaccine is sufficient for healthy adult animals, it is recommended for animals younger than one year of age to administer a second vaccine in the sixth month following the first vaccine administered in the fourth month.

#### 9.3.2.2. Vaccination as performed in Northern Africa countries

The experience gained in Morocco shows that PPR can be controlled in endemic areas, such as Northern Africa, through mass vaccination campaigns implemented at the national level, provided that adequate means are available and correctly implemented. After the vaccination campaigns, the epidemiological situation needs to be assessed. No viral circulation could be observed among young unvaccinated animals, and a good immune protection rate was achieved in vaccinated adults.

### 9.3.3. Vaccination in free areas

Although the present PPR vaccine is safe and effective, as shown by years of use in the field in various PPR-endemic countries, the main drawback for use in the EU is the lack of DIVA vaccines and diagnostic tests. The routine use of vaccination prevents effective serosurveillance, and makes it impossible to maintain a status of freedom from PPRV according to OIE.

Consequently, as also prescribed by the EU animal health regulation, EU countries would implement the culling of infected flocks to eliminate PPR infection, rather than vaccination.

A possible exception might be the case of low-noise PPRV circulation in sheep populations leading to widespread infection before a diagnosis is made, as was probably the case in Morocco when PPR emerged in 2008 (see section 8.2). Moreover, former experiences with foot-and-mouth disease virus showed that the social acceptability of wide-scale stamping out could be poor.

Finally, a further limitation for the use of vaccination is the lack of producers of PPR vaccines within the EU, and the global production of vaccines is likely to be needed for many years as the global PPR eradication campaign is initiated.

#### **9.4. OIE–FAO global PPR control strategy**

A global PPR control strategy is being developed within the Global Framework for the Progressive Control of Transboundary Animal Diseases, a joint initiative between OIE and FAO (Elsawalhy et al., 2010; Baron et al., 2011). The strategy that led to the successful eradication of rinderpest will serve as a model for PPR eradication programmes.

The overall objective of the global PPR control strategy is to contribute to poverty alleviation and improve livelihoods in developing countries, and to protect and further develop the global and regional trade in animals and animal products. The specific objective is to improve PPR and other transboundary disease control in regions where diseases are endemic.

The PPR control component of the strategy aims to reduce the burden of PPR on animal production not only in developing countries, but also in PPR-free countries. Reducing PPR at the source in PPR-endemic countries is therefore a shared interest and is considered a global public good.

The PPR strategy will include several components such as specific improvement of global PPR control, strengthening veterinary services, and improving the prevention and control of other major diseases of livestock. This means that the strategy will combine vertical (disease-specific) and transversal (horizontal) approaches.

The technical issues that support a progressive PPR control and eradication strategy include the fact that PPR has one serotype, that there is no carrier state after infection and that there is no known reservoir other than domestic small ruminants, although this should be clarified. Moreover, many of the tools required for progressive control are already available: diagnostic tests, cheap vaccines with lifelong immunity after a single dose and thermostable vaccines are under study.

The conditions for successful control and eradication include the effectiveness of the following: veterinary services, surveillance programmes, laboratories, appropriate legislation, sustainable animal health delivery systems including vaccine delivery, and the involvement of all veterinary actors in the field. Difficulties are considered to be the following: access to all remote areas, small village and small ruminant holders and the cost recovery issue. Interdisciplinary approaches and socio-economic analysis will be also needed.

The strategies will depend on the available tools (surveillance systems, diagnostic laboratories, vaccines, etc.), on the PPR epidemiological situation of each area (endemic or free, production systems, socio-economic systems) and on the socio-economic national and regional contexts such as veterinary services, legislation, social context (attitudes, behaviour, culture, politics and institutions).

#### **9.5. Assessment of effectiveness of control measures for PPR: lessons learnt from experiences at the country level**

##### **9.5.1. Turkey**

According to what was reported to OIE in 2014, the control measures against PPR put in place in Turkey include general surveillance, screening, vaccination, precaution at the borders, movement control within the country and zoning. In the case of outbreaks in holdings, cordon, quarantine, isolation and disinfection operations are set. It is forbidden to remove sheep, goats, cattle, buffaloes or poultry, as well as hay, grass or animal materials from quarantined areas. Camel, equid and cattle skins are allowed to be removed only after disinfection. Contact animals kept around disease outbreaks are made subject to homologous PPR vaccination starting from the centre and extending to the periphery of the area surrounding the outbreaks.

For the regions of Turkey where the disease is endemic, the main components of disease control are extensive vaccination, control of diseased or suspect animals and training activities aimed at strengthening active and passive clinical surveillance.

Although no fully clear data are available in relation to the current disease situation throughout the country, the disease is considered widespread in Turkey. There are inadequacies in disease notification, although Thrace is in a better position than Anatolia in terms of control measures against not only PPR, but also other transmissible diseases.

Since currently available diagnostic methods do not allow for the distinction of whether a PPRV antibody-positive result is induced by prior to vaccination or by infection, clinical surveillance and serosurveillance must be implemented to establish that PPRV is not in circulation. The sample for serological surveillance activities must be selected from unvaccinated animals without any maternal antibodies.

The aim is to vaccinate all susceptible animals older than three months and to ensure the provision of an immunity rate of at least 80 % in and among herds for PPR control and eradication.

Seromonitoring activities must be performed to measure the efficiency of vaccination and to designate the future strategy in regions of extensive vaccination.

A stronger focus must be placed on clinical surveillance during vaccination and following the end of vaccination. In addition, passive surveillance must also be strengthened by placing more emphasis on activities aimed at raising consciousness and awareness among people in direct contact with animals including breeders, dealers and caregivers.

The activities can be carried out more easily with a programme planned based on the division of the Turkish territory into regions on the basis of geographical features, effectiveness of the veterinary service, disease risk status and ease of monitoring. Programmes within this context must be monitored regularly and the control and eradication strategy must be improved and amended in accordance with the results obtained from these monitoring efforts. The active and accurate functioning of the identification and registration system is essential for the tracking of the vaccination history of animals.

Besides the monitoring and restriction of animal movements, susceptible materials should also be restricted between disease-free regions or regions actively seeking the recognition of the disease-free status and infected regions.

The following requirements are considered essential for PPR control:

- monitoring and restriction of animal movements, as well as movements of materials susceptible to disease transmission between disease-free regions or regions actively seeking the recognition of the disease-free status and infected regions;
- consequent prohibition of illegal animal movements from other neighbouring countries;
- trans-border cooperation under the governments of Turkey and the neighbouring countries;
- trans-border cooperation of border Provinces, customs and veterinary services;
- movements within the country with health certificates based at least on farm identification;
- permission of movements with respect to the epidemiological status (e.g. free, provisional free, endemic);
- movements of animals via markets only for ear-tagged and registered animals;
- stringent clinical examination of sheep and goats before entering livestock markets, collection points, etc. by official veterinarians (either municipality vets or governmental veterinarians);
- stringent clinical examination of sheep and goats before entering slaughterhouses by official veterinarians (either municipality vets or governmental veterinarians).

### 9.5.2. Morocco

In Morocco, PPR was controlled through mass vaccination, thus providing evidence that PPR control can be achieved if enough resources are available and correctly implemented. However, assiduous vigilance is still needed because the risk of PPR reoccurrence is present given the illegal cross-border livestock movements. Indeed, early detection of such reoccurrence is a necessary condition for rapid response and the effective management of possible outbreaks of PPR. This fragile PPR-free situation in Morocco highlights the importance of designing and actually implementing a regional PPR control strategy, relying on coordinated mass vaccination in infected countries, together with post-vaccination monitoring and efficient active surveillance measures. In particular, a better knowledge of legal and illegal livestock movements is of crucial importance. In this respect, Morocco has started an individual identification programme for small ruminants.



## CONCLUSIONS AND RECOMMENDATIONS

### CONCLUSIONS

#### **ToR 1. Characterise the disease and provide an update on the global occurrence of PPR and changes in the distribution during the last 10 years**

- PPR is a severe non-zoonotic viral disease of small ruminants caused by a *Morbillivirus* closely related to RPV. It is widespread in Africa, the Middle East and Southern Asia. It is one of the priority animal diseases whose control is considered important for poverty alleviation in those regions.
- PPR causes severe disease in its acute form, with fever, respiratory signs, congestion and necrosis of mucous membranes, diarrhoea, abortion and immunosuppression. The case-fatality rate can range from 10 to 90 %, depending on host status, and if animals recover there is no persistent infection or carrier state.
- PPRV resistance is extrapolated from its similarity with RPV. It is considered sensitive to heat, ultraviolet light and pH lower than 5.5 and higher than 10, and it does not survive in the environment, unless in shaded conditions, when it can survive for up to 72 hours. In fresh and chilled meat, it may survive for a few days.
- PPR transmission is essentially via contact with infected animals, or with their fresh secretions or faeces. The virus is found in all kinds of secretions, from approximately 3 to 22 dpi.
- Goats are considered more susceptible than sheep, and in the latter PPR may circulate undetected for some time. Cattle and pigs can be infected with the virus and then develop specific antibodies, but show no clinical signs. Camels and several wild ruminants can be infected and show clinical disease, although their role in the epidemiology of PPR, in particular in excreting the virus, needs to be clarified.

#### **ToR 2. Map the region of concern and other countries of the Mediterranean Basin and Black Sea, displaying identified, or likely, major live animal trade routes**

- The movement of small ruminants related to trade (both legal and illegal) is the most likely reason for the spread of PPR across borders, as it occurs between East Africa and the Arabian Peninsula, where the sudden and large increase in livestock movements owing to religious festivity can negatively affect the containment of the disease.
- Movement of live animals from third countries in the Mediterranean Basin and Black Sea areas into the EU is currently forbidden, according to EU animal health legislation on the import of live animals from countries where PPR is endemic. However, illegal movements of animals cannot be quantified.
- In Turkey, there are a large number of within-country movements of live small ruminants from provinces that have been affected by PPR in 2012–2013.
- The illegal movement of animal products, including meat products, carried by tourists and visitors from countries at high risk of PPR and communicable animal diseases is large and underestimated.

#### **ToR 3. Evaluate all possible pathways of introduction of PPR into the EU, ranking them on the basis of their level of risk, with a view to enhancing preparedness and prevention**

- The introduction of infected animals is largely the most efficient pathway to introduce PPR into a country. In the EU, this would occur mainly by the illegal transport of infected animals.
- The introduction of PPRV into the EU through infected animal products may occur, in particular when introduced illegally or with the intention to spread the virus (e.g.

bioterrorism), although the risk of this is low and the further spread of PPR via this route is unlikely.

- The introduction of PPRV via fomites into the EU is unlikely. This could occur when vehicles carrying livestock return to the EU after the delivery of animals in infected areas or farms and where no biosecurity measures are applied.

#### **ToR 4. Assess the risk of introduction and speed of propagation of PPR into the EU and neighbouring countries**

- The introduction of PPR into the EU may occur via illegal transboundary movement of animals from infected regions. In order to estimate this risk, a model was used to assess the probability of an individual being infectious in a given shipment size. For example, for a level of seroprevalence in the country of origin equal to 37 %, the number of animals that would need to be moved to have a probability of introduction greater than 0.95 or lower than 0.05 to introduce PPR into Europe would be 421 and 8, respectively. On the other hand, if the seroprevalence is 8 %, the number of animals that would need to be moved to introduce PPR with a probability greater than 0.95 or lower than 0.05 would be 1 952 and 34, respectively.
- Outbreak data of PPR in Tunisia as reported to OIE were used to plot temporal and spatial links between outbreaks and to derive an estimate of potential ranges of the speed of propagation. According to this, the median speed of propagation was estimated to be 3.9 km/day, with a 95 % CI of 0.3 to 65.5 km/day. This result should be interpreted with caution without direct extrapolation to the potential epidemiological behaviour of PPR if it entered the EU.
- Because the control measures as applied in the EU would aim at culling infected flocks and restricting movements, the spread of PPR in European situations would depend on farm density, travelling frequency and distance of small ruminants, and the duration of silent spread (high-risk period). In such conditions, the long-distance transmissions would be more important for initiating new epidemics and thus for the spatial spread of the infection.
- The basic reproductive ratio calculated with data from a case study in Senegal and compared with other studies in Tanzania and Pakistan showed that PPR has a high within-flock transmission rate, and an outbreak will lead to infection of most animals in a herd.
- The between-herd transmission would be very variable within Europe, and common grazing grounds would be a major risk source and should be avoided when PPR is detected in the neighbourhood.

#### **ToR 5. Assess the risk of PPR becoming endemic in animal populations in the EU and neighbouring countries**

- Owing to a lack of data regarding PPR transmission in the EU, the international data cannot be extrapolated directly to the European situation to make a quantitative assessment of the risk of endemicity.
- Given the control measures foreseen by the current EU policy, PPR would most likely not become endemic in the EU.
- PPR is endemic in several countries neighbouring or close to the EU.

#### **ToR 6. Assess the impact PPR would have if it were to enter the EU, considering different scenarios as regards the effectiveness of surveillance and control measures**

- PPR is particularly a constraint in less developed areas, in particular of Africa, the Middle East and Asia, where livelihoods highly depend on livestock for their survival, especially on sheep and goats. The small ruminant population in endemic countries is estimated to be 1.4 billion.

- From worldwide experience in endemic areas, it can be assumed that, in the EU, goats would be more susceptible than sheep.
- There might be a risk that, if PPR enters EU areas with dense sheep populations but low goat densities, such as Great Britain or Ireland, it would start circulating without being promptly detected, and would lead to widespread infection.

#### **ToR 7. Briefly review the feasibility, availability and effectiveness of the main disease prevention and control measures**

- Clinical signs of PPR are not disease specific and clinical diagnosis is not reliable; therefore, PPR should be confirmed by laboratory testing.
- The most common laboratory techniques for PPRV detection are PCR and immunocapture ELISA. The latter is of choice where molecular techniques are not available or biological samples are poorly preserved, although this method is not as sensitive as PCR.
- Several commercialised kits for real-time PCR assays are available and these are generally more sensitive than the gel-based assays.
- Among serological tests, the one that is most used is competitive ELISA.
- Live, attenuated PPR vaccines are available, with high safety and efficacy, protecting against all known isolates of PPRV. No PPR vaccines are licensed in the EU.
- The available PPR vaccines do not support the DIVA principle. Possible DIVA vaccines based on recombinant techniques are promising but are still at the experimental stage.
- Inactivated vaccines are not available and, owing to the immunological response to PPRV, would not be fully effective.
- The lessons learnt from the PPR epidemics in Morocco show that PPR can be controlled in areas, such as Northern Africa, through mass vaccination campaigns implemented at the national level, provided that adequate means are available and correctly implemented.
- In endemic areas, assiduous vigilance is needed because there is a risk of PPR reoccurrence, especially with risk factors of continuous introduction such as the illegal cross-border movements of livestock.
- Early detection of (re)occurrence is a necessary condition for rapid response and the effective management of possible outbreaks of PPR.

### **RECOMMENDATIONS**

#### **Preparedness**

- Awareness-raising campaigns and training for farmers and veterinary staff in recognising the disease under field conditions should be considered, especially for regions at higher risk of the introduction of PPR (i.e. those bordering affected regions).
- A better knowledge of legal and illegal livestock and animal product movements should be sought, especially in areas at risk of or affected by PPR.
- Adequate veterinary care and improved surveillance should be in place, in particular for transhumant flocks along the migratory routes in risk areas and especially for long-distance migrations.
- Designing and actually implementing a regional PPR control strategy is recommended, especially in endemic countries or where PPR has occurred and been controlled (e.g. Morocco), relying on coordinated mass vaccination, and post-vaccination monitoring and efficient active surveillance measures.

- If non-biological drivers of transmission of transboundary animal diseases change (e.g. breakdown of veterinary infrastructures, human migration, political unrest), the risk of PPR introduction should be accordingly reassessed. Under this perspective, the cooperation of the EU with endemic countries should be encouraged for the prevention of introduction of PPR and other transboundary animal diseases and to enhance preparedness.

### Control

- If PPR entered the EU, rapid detection, movement restriction, prompt culling of infected herds and disinfection measures should be considered as effective measures in limiting the spread and the impact of the outbreaks.

### Research needs

- The PPR endemicity in countries neighbouring the EU underlines the need for the development of safe, efficient and non-replicating DIVA vaccines against PPRV, as well as an associated diagnostic test.
- The following aspects of PPR should be investigated:
  - the PPRV survival and infectiousness in different matrices (e.g. meat, milk) and under different environmental conditions (e.g. temperature, pH, humidity);
  - the virulence of different virus isolates, the capacity of virus excretion and infectiousness in the same host animals, or a comparison of a single isolate in different host species, including European goats, sheep besides cattle, camels, pig breeds and wild ruminants to PPR, including their potential capacity of excreting the virus;
  - collection and analysis of data on the transmission and spread within and between herds in different situations, including in a situation comparable to the EU;
  - the knowledge on populations, movements and contact patterns of small ruminants;
  - the impact of socio-economic factors on the efficacy and efficiency of vaccination campaigns (e.g. vaccine delivery system) in countries where PPR is endemic.

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## APPENDIX

### Appendix A. Protocol for the literature review on diagnostic tools for peste des petits ruminants (PPR)

#### BACKGROUND

Considering the 7th ToR “briefly review the feasibility, availability and effectiveness of the main disease prevention and control measures (diagnostic tools, biosecurity measures, restrictions on the movement, culling, vaccination)”, it is considered appropriate to review the literature for diagnostic test for Peste des Petits Ruminants (PPR).

#### OBJECTIVE/REVIEW QUESTION

The objective of this literature review is to identify diagnostic tests for PPR.

To achieve this objective, the review question is described below.

Review question:

*What are the diagnostic tests for PPR virus?*

#### FRAMEWORK OF REVIEW QUESTION/AND ELIGIBILITY CRITERIA FOR STUDY SELECTION

A literature review was conducted.

Framework for review question issuing P-I-T:

	<b>Review question:</b> Diagnostic tests for Peste des Petits Ruminants
<b>Years</b>	From 1980 onwards
	<b>Comment/Explanation:</b> This will depend on the availability of the bibliographic databases subscription to (and will be reported in the results of the search process).
<b>Language</b>	Only English will be reviewed
	<b>Comment/Explanation:</b>
<b>Publication type</b>	Only primary research studies
	<b>Comment/Explanation:</b> <ul style="list-style-type: none"> <li>• Reviews (i.e. secondary research studies) will not be included in the review, but they reference lists will be screened as sources of studies.</li> <li>• Letters and editorials will be excluded as normally these do not include any primary research studies.</li> <li>• Patents will be excluded.</li> <li>• No geographical limits.</li> </ul>
<b>Population</b>	Sheep and goats will be the only species considered in the literature search.
	<b>Comment/Explanation:</b> <ul style="list-style-type: none"> <li>• Sheep and goats of all ages, any breed and both genders will be included</li> <li>• Wild small ruminants will be excluded</li> </ul>
<b>Intervention</b>	Direct and indirect diagnostic tests for Peste des Petits Ruminants
	<b>Comment/Explanation:</b>
<b>Target</b>	Peste des Petits Ruminants virus (family Paramyxoviridae, genus Morbillivirus) will be the targeted pathogens.
	<b>Comment/Explanation:</b>

## IDENTIFYING RESEARCH EVIDENCE

The literature search aimed to identify studies on diagnostic test for Peste des Petits Ruminants.

### Information sources

Search strategies included use of electronic search engines for bibliographic databases. Review manuscripts (i.e. secondary research studies) were included in the review.

Due to time constraints only studies in peer-reviewed journals and conference proceedings were included. Book chapters, theses and informally reported or unpublished data were not collated.

Two databases were searched:

- Web of Science
- EBSCO

Experts consider that these databases are enough for the objective of this literature review.

### Search strategy

The following search strategy is proposed:

**Population:** *Sheep or goat\* or ovine or ovi\* or capr\* or "small ruminants"*

**Index Test:** *"diagnostic test" or \*PCR or serolog\* or antibody or "virus isolation" or "direct test" or "indirect test" or immunodiffusion or "immune diffusion" or "AGID" or "\*virus antigen" or ELISA or neutralisation or neutralization or immunoelectrophoresis or CIEP*

**Target:** *"Peste des petits ruminants" or PPRV or Paramyxoviridae or Morbillivirus or "small ruminant pest" or "pest of small ruminants"*

A scoping search identified:

- 1055 papers from Web of Science
- 327 papers from Ebsco

A database of the electronic search results was created with the EndNote X5/Endnote Web program. Duplicate citations (306) were deleted using the automated function and manually when required.

## STUDY SELECTION

Screening of titles and abstracts against inclusion criteria described in the study eligibility form (in ATTACHMENT) was conducted for 1076 papers. Excluded or unclear studies were recorded. Studies that were relevant to the review question were reported and summarized in a tabular format (Table 4).

**STUDY ELIGIBILITY FORM: PERFORMANCE OF DIAGNOSTIC TESTS FOR PESTE DES PETITS RUMINANTS**

All papers with a ‘no’ or ‘unclear’ for any question will be excluded based on title/abstract.

All papers with a ‘yes’ for each question will be included based on title/abstract.

**Reviewer ID:**

**Study ID:**

**Question 1:**

Is the paper in English?

**YES**

**UNCLEAR**

**NO**




**Question 2:**

Is the paper an original publication?




**Question 3:**

Is the paper describing PPR diagnostic tests for sheep and goats?




**Question 4:**

What diagnostic tests is it describing? (Checkbox)

**FINAL DECISION**

**INCLUDE**

**REVIEW**

**EXCLUDE**




**Table 4:** Results of the literature review for diagnostic test for Peste des Petits Ruminants (PPR). Diagnostic tests described in the 2013 OIE Terrestrial Manual are in bold.

Test	Total number of references	Reference ID
<b>Competitive ELISA (cELISA)</b>	101	1, 2, 4, 5, 6, 14, 15, 19, 20, 21, 27, 28, 29, 35, 40, 41, 44, 48, 50, 52, 56, 57, 59, 62, 65, 66, 67, 68, 70, 72, 74, 79, 80, 81, 84, 86, 87, 88, 89, 90, 93, 94, 95, 97, 98, 99, 105, 106, 109, 112, 113, 114, 116, 117, 118, 119, 120, 121, 125, 126, 128, 130, 131, 132, 133, 135, 137, 138, 139, 141, 142, 145, 147, 148, 150, 151, 153, 154, 156, 161, 163, 165, 170, 180, 186, 190, 193, 199, 206, 212, 213, 219, 222, 223, 225, 230, 233, 238, 240, 241, 243
<b>RT-PCR</b>	42	3, 8, 9, 11, 13, 17, 18, 19, 23, 30, 31, 32, 34, 46, 51, 55, 60, 63, 73, 74, 75, 89, 91, 92, 96, 100, 108, 111, 115, 129, 134, 144, 146, 152, 164, 168, 184, 216, 226, 235, 236, 237
<b>Immunocapture ELISA (icELISA)</b>	26	61, 69, 78, 79, 86, 88, 92, 102, 103, 110, 131, 133, 153, 155, 158, 166, 167, 180, 181, 187, 208, 218, 219, 220, 237, 240
Sandwich ELISA (sELISA)	22	16, 17, 24, 37, 50, 55, 58, 71, 74, 76, 89, 101, 111, 140, 149, 151, 154, 155, 216, 221, 239, 242
<b>Real Time RT-PCR (QRT-PCR) (QRT-PCR)</b>	19	3, 5, 7, 10, 12, 33, 36, 38, 53, 69, 73, 123, 214, 215, 217, 219, 224, 228, 232
<b>Virus neutralisation (VN)</b>	14	3, 15, 26, 139, 145, 156, 162, 163, 186, 189, 195, 197, 199, 209
<b>Agar gel immunodiffusion (AGID)</b>	11	61, 95, 106, 112, 127, 158, 166, 188, 200, 223, 229
ELISA (not specified)	11	77, 107, 136, 176, 182, 185, 196, 197, 231, 234, 183



Test	Total number of references	Reference ID
Virus Isolation in cell culture (VI)	10	58, 60, 96, 102, 144, 168, 176, 203, 237, 239
Counter immunoelectrophoresis (CIEP)	10	85, 94, 98, 107, 160, 173, 177, 179, 181, 211
Indirect ELISA	8	43, 44, 47, 82, 83, 125, 135, 212
Agar gel precipitation test	7	122, 171, 177, 181, 189, 211, 196
Sero-neutralisation test (SN)	6	172, 152, 167, 192, 194, 204
Unclear	6	22, 25, 39, 42, 45, 54
Immunohistochemical test	5	64, 104, 124, 159, 191
Haemagglutination test	4	61, 103, 198, 122
Immunofluorescent antibody test	3	178, 237, 183
PCR	3	85, 143, 219
Precipitinogen inhibition test	3	95, 112, 201
Blocking ELISA	2	195, 241
Conventional PCR	2	18, 30
dot ELISA	2	210, 189
Immunoelectrosmophoresis test	2	202, 205
Radioimmunoprecipitation test	2	183, 169
RT-LAMP	2	73, 115
Avidin-biotin-peroxidase immunoperoxidase staining test	1	175
Chromatographic strip test with monoclonal antibody	1	49
Countercurrent immunoelectrosmophoresis	1	207
Fixed-cell ELISA	1	169
Fluorescent antibody technique	1	79
Haemagglutination-inhibition test	1	172
Immunofiltration-based test	1	120
Indirect fluorescent antibody test	1	153
Latex agglutination test	1	110
Monoclonal antibody-based ELISA	1	227

Test	Total number of references	Reference ID
Passive haemagglutination test	1	20
PCR-ELISA	1	157
RT-PCR-ELISA	1	134
Single radial haemolysis test	1	98
Slot blot hybridisation	1	169
Solid phase aggregation of coated erythrocytes test	1	174

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