



Emergence of PPR and its threat to Europe

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ABSTRACT

PPR is an important infectious viral disease of domestic and wild small ruminants, that threatens the food security and sustainable livelihood of farmers across Africa, the Middle East and Asia. Europe is free of the disease except in Thrace (European part of Turkey) and Israel where outbreaks occur. Following the successful eradication of RPV, PPR has been targeted by the OIE and FAO as the next viral pathogen to be eradicated by 2030. However, the recent outbreaks in Northern Africa and Thrace (European part of Turkey) represent a significant threat to mainland Europe, as a source of disease spread. We have discussed here the emergence of PPR worldwide since its discovery with particular reference to the recent outbreaks in Northern Africa and Thrace, and the potential for spread of the disease into Europe.

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1. Introduction

PPR (goat plague) is an important infectious viral disease of domestic and wild small ruminants that threatens the food security and sustainable livelihood of farmers across Africa, the Middle East and Asia (Banyard et al., 2010; Libeau et al., 2014; Parida et al., 2015). PPR is emerging in new regions of the world and is causing great economic losses (Perry et al., 2002; Singh et al., 2014; Banyard et al., 2014). The causative agent, peste des petits ruminants virus (PPRV) belongs to the family *Paramyxoviridae*, genus *Morbillivirus* under order *Mononegavirales* alongside other important viral pathogens such as rinderpest virus (RPV), measles virus (MV) and canine distemper virus (CDV) (Fig. 1). The incubation period of PPR is typically 4–6 days although it may range from between 3 and 10 days. At the acute stage of disease, animals show a pyrexia response of up to 41 °C that may last for 3–5 days, depression, anorexia and dryness in the muzzle. Watery nasal and lachrymal discharges gradually become mucopurulent with excessive salivation. Erosive lesions formed in the oral cavity may become necrotic. In the later stage of infection, animals develop diarrhoea leading to severe dehydration and a persistent

cough with laboured abdominal breathing. This clinical condition may last for 14 days before recovery from infection although it can lead to death during the acute stage of infection. The morbidity rate can reach 100% with a high case fatality with the acute form of disease. The above described clinical signs and mortality can vary considerably depending on the virulence of virus (OIE, 2012). Live attenuated vaccines are in use in endemic areas that provide long term immunity in sheep and goats. Following the successful eradication of RPV, PPR has been targeted by the OIE and FAO as the next viral pathogen to be eradicated by 2030 (FAO, 2014).

1.1. Emergence of PPR

The first report of PPR was made in 1942 in the Ivory Coast (Gargadennec and Lalanne, 1942). Before 1942, it is likely that PPR would have been confused with rinderpest (Parida et al., 2015). After the first recognition of PPR in the Ivory Coast, increased awareness led to further reports in neighbouring countries in the order, Senegal, Chad, Togo, Benin, Ghana, Nigeria, Oman, Sudan, Saudi Arabia, India, Jordan, Israel, Ethiopia, Kenya, Uganda and Pakistan (Sen et al., 2010). This detection appeared to demonstrate the geographic spread of disease from West Africa to East Africa, and then to the Middle East and Asia (Muniraju et al., 2014). Since then PPRV is considered to be endemic across Africa, the Middle East and Asia (Banyard et al., 2010; Dhar et al., 2002; Kwiatek et al., 2011).

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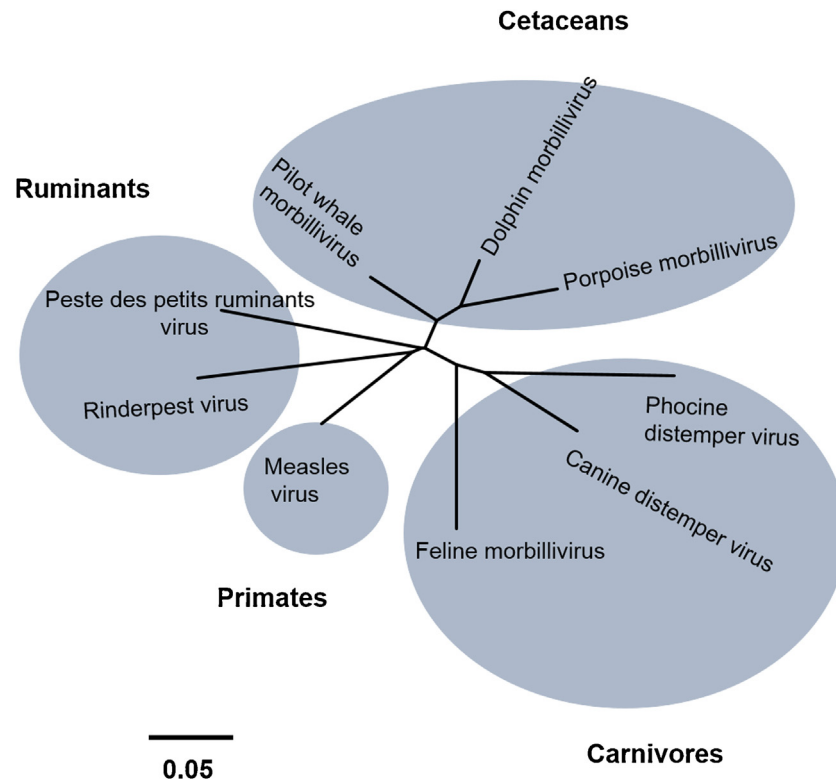


Fig 1. Un-rooted neighbour-joining tree showing the relationships between the different morbilliviruses. Cited from Parida et al., 2015. *Veterinary Microbiology*, 14; 181 (1–2); 90–106.

In recent years PPRV has extended its boundaries southwards in Africa as far as southern Tanzania (2008), Zambia (2015) and the Democratic Republic of Congo and Angola (2012). PPR outbreaks have also been reported across North Africa including Tunisia (2006), Morocco (2008 and 2015) and Algeria (2011 and 2016). Alongside this, within the European part of Turkey approximately twelve laboratory confirmed PPR outbreaks in sheep and goats were reported during 2012 (Parida et al., 2015). The first occurrence of PPR has been reported in Georgia in the month of February, 2016 (OIE report). In East Asia, the virus spread to Tibet (2007) and has recently been reported all over China (2013–2014) (FAO, 2013). In central Asia the disease is prevalent in many countries and the virus detected in Kazakhstan (2014) was found to be genetically similar to the virus circulating in the mainland of China but was distinct to the virus isolated from 2007 outbreaks in Tibetan part of China (Kock et al., 2015).

PPRV exists as a single serotype but at the genetic level is divided into four distinct lineages. This lineage differentiation is based on partial genome sequence of either the N or the F gene (Couacy-Hymann et al., 2002; Forsyth and Barrett, 1995; Banyard et al., 2010; Senthil Kumar et al., 2014). Historically, lineages I–III were found in Africa and were numbered according to the apparent spread of virus from West Africa (I and II) to East Africa (III). Lineage IV was mainly restricted to the Middle East and Asia with a few exceptions of Lineage III in Yemen and Oman and mixed lineages of III and IV in UAE and Qatar. However, lineage IV has now established its presence all across the PPR endemic areas with frequent outbreaks in Africa (Kwiatk et al., 2011; Parida et al., 2015).

A recent full genome phylogeographic analysis has indicated that Nigeria was the geographic origin of the most recent common ancestor of PPRV (Muniraju et al., 2014). The estimated median time to most recent common ancestor (TMRCA) of PPRV was estimated to be \approx 1900 (95% HPD 1805–1962) (Parida et al., 2015). Furthermore, geographic origins of the most recent common ancestor of

PPRV lineages I, II, and III were predicted to originate within Africa; lineage IV likely emerged in India suggesting that the origin of PPRV was in western Africa, which then spread to eastern Africa, the Middle East, and Asia. Recently 33 full PPR genome sequences became available for PPRV and considering all these available sequences (Genbank upto the end of November, 2015) a neighbour-joining phylogenetic tree was constructed (Fig. 2). From this data it is clear that lineage IV has established its presence not only in Asia and the Middle East, but also in recent years in many PPR endemic countries in Africa (Fig. 3). From the Phylogenetic analysis two clear groups (Asian and African origin) of lineage IV viruses are observed. Asian lineage IV viruses are again distributed into two subgroups, the Indian vaccine strain (Sungri 96) and Izatanagar strains are found to be more closely related to the circulating strains in Turkey whilst Chinese strains form a separate cluster. The China (Tibetan) outbreak viruses from 2007 are more closely related to the viruses circulating in India whilst the viruses characterised from the 2014 outbreaks across China are more closely related to the viruses from Kazakhstan (Kock et al., 2015).

Lineage II viruses have been mainly reported in north-west, western and central Africa and recently in Tanzania, East Africa (Mahapatra et al., 2015; Parida et al., 2015). Lineage I virus although historically reported in West Africa has not been detected for several years (since 1997). Lineage III viruses are currently circulating in East African countries and have been historically reported in Oman and in the UAE in the Middle East.

1.2. Transmission of disease and threat to Europe

PPRV is transmitted to close in-contact susceptible animals through exhaled aerosols, particularly during coughing, or through clinical excretions (lachrymal, nasal, saliva and faeces). Virus may be spread over large distances through the movement of infected animals for trade or for during migration, particularly those

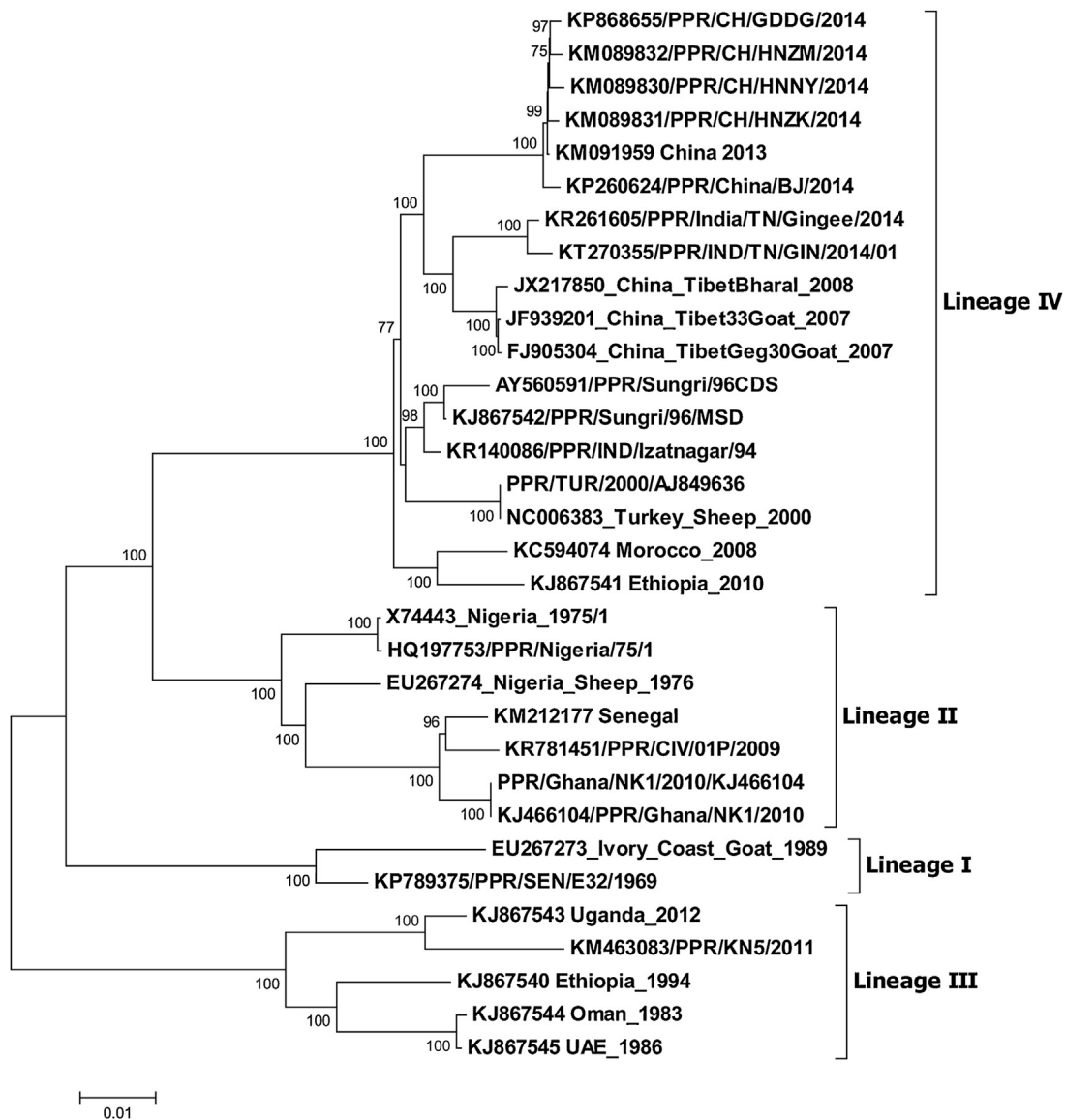


Fig. 2. Phylogenetic analysis of circulating PPR viruses. Neighbour-joining tree was constructed using nucleotide sequences of the full genome of PPRV showing the relationships between the PPR viruses circulating in Asia, the Middle East and Africa. The percentage of replicate trees in which the associated taxa clustered together in the bootstrap test (10,000 replicates) is shown next to the branches. The GenBank accession number of each sequence is shown in the taxon name.

animals incubating the disease without clinical manifestations. PPRV is temperature labile and is readily inactivated outside its host in a dry environment. Infected and recovered animals develop life-long protective immunity with no carrier state (Hamdy et al., 1976). However, virus can spread in animals as a mild virulent form that can later lead to severe disease where transmission occurs to naive susceptible populations (Couacy-Hymann et al., 2007). There is also a possibility that the virus may be transmitted through fomites. Host factors like age, sex, breed and season may also play a role in disease outbreaks. Sheep and goats are the primary hosts for PPRV with a few reports of disease outbreaks in camels (Khalafalla et al., 2010; Kwiatek et al., 2011; Roger et al., 2001; Saeed et al., 2004). Cattle (Anderson and McKay, 1994; Lembo et al., 2013; Sen et al., 2014; Abubakar et al., 2015), buffalo (Govindarajan et al., 1997) and pigs (Nawathe and Taylor, 1979) develop a subclinical infection with PPRV, but are not thought to be capable of excreting virus and contributing to the epidemiology of the disease. PPRV infection in wildlife, mainly living under semi-free range conditions, has also been reported, though the exact role of wildlife in the epidemiology of PPR disease remains to be clarified. A recent sero-survey at

the interface of wildlife and domestic small ruminants in Serengeti eco-system in Tanzania revealed spill-over of virus from domestic infected animals to wildlife, which needs further investigation (Mahapatra et al., 2015).

The recent reports of PPR in countries previously considered free and the reoccurrence of PPR in countries which had experienced PPR earlier, are mainly thought to be caused by the introduction of new live sheep and goats incubating the disease or illegal movements of infected small ruminants or their products from endemic countries to free countries. An example of this is the outbreaks reported in Angola where PPR outbreak was experienced for the first time in 2012 after importing sheep and goats from Democratic Republic of Congo. Situations like this, alongside the recent PPR outbreaks in North African countries (Morocco, Algeria, Mauritania and Tunisia) as well as PPR outbreaks in Marmara region of Turkey, particularly in the European part of Turkey (Thrace region), represent threats for incursion of PPR into mainland of Europe. Morocco was free from PPR until 2008. It is believed that the disease was introduced through the movement of live infected animals due to intense trade in northern Morocco immediately before the

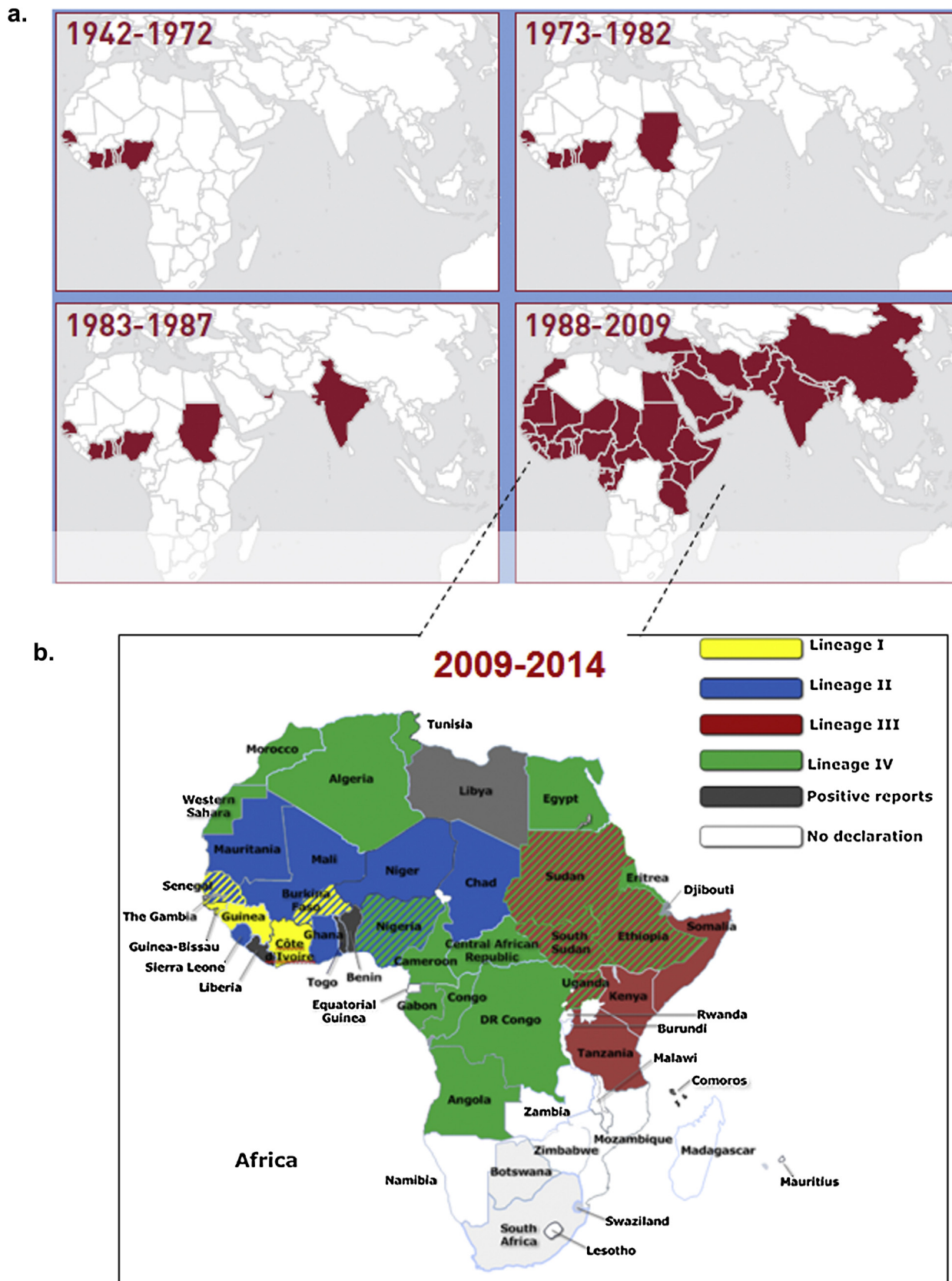


Fig. 3. Global spread of Peste des petits ruminants virus from its first detection in 1942–2014, including lineage distribution; (a) adopted from Food and Agriculture Organisation; (b) recent circulations of Peste des petits ruminants virus in Africa, drawn by using smart draw software. Cited from Parida et al., 2015. *Veterinary Microbiology*, 14; 181; (1–2); 90–106.

EID al-Adha festival. Due to mass vaccination campaigns during 2009–2011, PPR outbreaks were not seen until June 2015 when the PPR outbreaks reoccurred. A serosurveillance study in young animals (less than 8 months of age) in 2012 revealed no antibody prevalence against PPR virus and therefore, it is believed that circulation of PPR virus had ceased. The cause of recent outbreaks in Morocco is unknown but is believed to be due to the transboundary movement of infected animals. Mauritania has been endemic for PPR since the 1980s (Le Jan et al., 1987). PPR outbreaks in Mauritania in 2012 were shown to be caused by Lineage II PPRV, similar to the Senegalese outbreak. The 2010 PPR outbreaks in the Sahrawi Territories of Algeria were caused by a Lineage IV virus (De Nardi et al., 2012). The most recent reported outbreaks were seen in Ghardaia province of Algeria during 2013 (Kardjadj et al., 2015) and reoccurred in February 2016, indicating continuous circulation of PPRV in Algeria. The southern desert areas bordering Sahel countries favour the movement of infected animals for the spread of PPR in Algeria. A recent serological survey in Algeria in 2012 showed a PPRV seroprevalence of 68.8% (Baazizi et al., 2015). In Tunisia, serological evidence was first reported in 2006 (Ayari-Fakhfakh et al., 2011) and clinical cases were described in 2012 and 2013 (Soufien et al., 2014). It is believed that the uncontrolled introduction of PPRV infected animals through southern border of Tunisia resulted in these new outbreaks.

Although seroprevalence against PPR virus was reported earlier in Turkey, the first clinical outbreak in Southeastern Anatolia, Eastern Anatolia and Mediterranean region of Turkey was not reported until 1999. The movement of live infected animals facilitated the rapid spread of disease to other parts of the country. In Thrace, PPRV infection was reported in Istanbul for the first time in 2000 and Istanbul was followed by Edirne (on the border with Greece) in 2004 and Kırklareli (on the border with Bulgaria) in 2006. Since then outbreaks have been reported annually in Kırklareli except during 2009 and 2014. There were a total of 12 outbreaks recorded in the Thrace region during 2011 and three during 2012. During 2013 only one outbreak was reported in Kırklareli in the Thrace region. These events pose big threats to Europe for the incursion of PPRV. In addition to the illegal movement of infected animals or animal products, direct connectivity of this endemic region to mainland Europe is another potential route of transmission. Turkey hosts the same community of wild ruminants as Europe including, representatives from the Caprinae (wild goats, chamois) and Cervidae. Wild goats are of high interest as the ancestor of the domestic goats; Cervidae [roe deer (*Capreolus capreolus*), red deer (*Cervus elaphus*)] are of importance because they are the most widespread and abundant wild ruminants in Europe. These wild ruminants may serve as a bridging species for PPRV transmission between distant populations of infected and healthy domestic sheep and goat populations. In Kurdistan, wild goats (*Capra aegagrus*) were reported to be clinically infected (Hoffmann et al., 2012) and in United States of America, white-tailed deer (*Odocoileus virginianus*) were experimentally infected (Handy and Dardiri, 1976) by PPRV.

Importing live animals from countries endemic for sheep and goat pox is forbidden. Therefore the legal importation of sheep and goats from North African countries and Turkey to Europe is not possible. However some movements of live sheep and goats were recorded in Eurostat and the UN COMTRADE data base in 2011 that showed animal movements from Turkey to Bulgaria and Hungary. A large number of sheep and goats used to be transferred within Turkey, particularly from southern and western Turkey that are endemic with PPR to free areas like Istanbul and Thrace. According to UN COMTRADE data base, the largest number of animal movement occurs between East African countries and the Middle East. Although North African countries generally do not import live sheep and goats, an informal trade of animal movement through the borders is ongoing, which favours the spread of PPR. Although the

movement of sheep and goats from these North African countries to Europe is forbidden, movement of live animals from Spain, France, Italy and Romania to Morocco, Algeria and Tunisia are registered, particularly towards the end of religious festivals like Ramadan and the month preceding Hajj. Therefore, there is a chance of PPR spread from North African countries to the European countries through fomites, potentially through returning trucks used to deliver live sheep and goats to the North African countries. Therefore proper cleaning and disinfection of vehicles transporting livestock is required before returning vehicles to European countries. Another threat of disease spread may occur through the uncontrolled movement of infected animal products, i.e. in the luggage of passengers as all luggages are not checked at the airports or other ports of entry. As the virus can survive for 2–3 days in meat, there is a possibility of introduction of the disease in PPR free countries. Similarly, small ruminants may be smuggled in vehicles into Europe for domestic consumption (Miller et al., 2009). The illegal movement of infected animal products represents a further potential threat for disease spread to PPR free countries.

2. Conclusions

Preventive measures employed in an uninfected area includes the restriction of animal importation from disease-endemic regions. Disease can be efficiently controlled by isolating and slaughtering infected animals, disinfection of the premises and the quarantine of animals suspected of carrying infection without showing clinical signs/symptoms. Prophylactic immunisation in suspected animal populations or areas is also recommended within endemic countries. Immunisation is carried out with the availability of excellent live attenuated vaccines that elicit a protective immunity that is maintained for at least three years (Diallo et al., 2007; Sen et al., 2010; Singh et al., 2010). The major obstacle to PPR control in endemic countries is the requirement for frequent immunisations, at least every 3 years. Due to the high turnover of small ruminant populations, vaccination of younger animals (4 months old) is recommended (Balamurugan et al., 2012).

In conclusion, there are threats for the potential spread of PPR into mainland Europe from North Africa and Turkey. The risk of disease spread to Europe or other PPR free countries could be reduced by: raising awareness of the disease; training farmers and veterinary staff to identify the disease; increasing knowledge of the transportation of animals and animal products; implementation of adequate biosecurity measures at relevant borders including potentially implementing surveillance at borders and maintaining stocks of vaccine, preferably that can be differentiated from infection.

Conflict of interest

Authors have no conflict of interest.

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