Received: 17 June 2019 | Revised: 6 February 2020 | Accepted: 29 February 2020

DOI: 10.1111/tbed.13535

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Transboundary and Emerging Diseases WILEY

ORIGINAL ARTICLE

A molecular and pathological study of peste des petits ruminants virus (PPRV) from field outbreaks in Palestine, 2017–2019

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Abstract

Peste des petits ruminants (PPR) is a severe, highly contagious and fatal viral disease of small ruminants causing high economic losses. Therefore, the disease was targeted for eradication by 2030. The aim of this study was to investigate for the first time the molecular and pathological characterization of the circulating PPR virus (PPRV) in sheep and goat in Palestine. Samples were collected from suspected necropsy cases of sheep and goats during recent outbreaks in two provinces in Palestine between 2017 and 2019. In this study, severe PPR outbreaks occurred in sheep and goats causing typical lesions which include erosive and ulcerative stomatitis, bronchointerstitial pneumonia and severe enteritis. For the molecular investigation of PPRV, suspected animals were examined for the presence of PPRV by RT-PCR. PPRV genome was detected in all samples. Subsequently, two samples were used for N gene sequencing and phylogenetic analysis of PPRV isolates. The nucleotide sequence and phylogenetic analysis indicated that the Palestinian PPRV isolates were genetically clustered within the lineage IV isolates of the virus among populations of sheep and goats which most prevalent in Asia, the Middle East and recently Africa. Further analysis showed that the Palestinian isolates were closely related to those described in Turkey and Iraq, suggesting a common origin of PPRV isolates in the region. This information is critical to understand the molecular epidemiology of this disease in the region and helps to develop appropriate control measures for eradication of this disease.

KEYWORDS

goat, Palestine, petits ruminants disease, phylogenetic analysis, sheep

1 | INTRODUCTION

Peste des petits ruminants disease (PPR) is a highly contagious disease affecting primarily small ruminants such as sheep and goats and currently considered as one of the most important animal transboundary diseases in small ruminants in developing countries (Banyard et al., 2010; Baron, Parida, & Oura, 2011). The disease is caused by peste des petits ruminants virus (PPRV), a single-stranded negative-sense RNA virus that belongs to the small ruminant morbillivirus subfamily of the *Paramyxoviridae* family (Amarasinghe et al., 2017). PPRV genome encodes eight proteins: the nucleocapsid protein (N), the phosphor-protein (P), the matrix protein (M), the fusion protein (F), the haemagglutinin protein (H), the polymerase protein (L) and the two non-structural proteins, C and V (Banyard et al., 2010). PPRV was first described in 1942. (Gargadennec & Lalanne, 1942). The virus was thought to be an antigenic variant of the rinderpest virus (RPV) (Mornet, Orue, & Gilbert, 1956). However, the biological and physicochemical characteristics of the PPRV indicated

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that the agent is considered as a distinct entity of the *Morbillivirus* of *Paramyxoviridae* (Gibbs, Taylor, Lawman, & Bryant, 1979). Since then, the virus has been reported in many countries in West and Central Africa, Arabia, the Middle East and Southern Asia (Nanda et al., 1996; Shaila, Purushothaman, Bhavasar, Venugopal, & Venkatesan, 1989). In addition, PPRV is reported globally with increasing frequency (Banyard, Wang, & Parida, 2014).

Although PPR has been targeted by the World Organization for Animal Health (OIE) and the Food and Agriculture Organization (FAO) as the next viral pathogen to be eradicated by 2030, recent PPR outbreaks have been reported in North Africa and the European part of Turkey (Parida et al., 2016). These recent outbreaks represent a potential threat for the spread of the disease into Europe (Parida et al., 2016).

Genetically, PPRV is divided into four distinct lineages (I-IV) based on the sequence comparison of the F gene, N gene or H gene (Couacy-Hymann et al., 2002; Forsyth & Barrett, 1995; Kumar et al., 2014). Previously, lineages (I-III) presences were restricted in PPR endemic areas in Africa (Parida et al., 2015), whereas lineage (IV) was reported in the Middle East and Asia (Banyard et al., 2010). However, with rapidly rising incidence and virulence of PPRV across Africa, the Middle East and Asia, the Asian lineage (IV) is constantly being reported in Africa (Kwiatek et al., 2011).

Information on the prevalence of PPR is known in neighbouring countries such as Jordan and Israel (Clarke, Mahapatra, Friedgut, Bumbarov, & Parida, 2017; Lefevre, Diallo, Schenkel, Hussein, & Staak, 1991). However, to the best of our knowledge, no previous studies have been performed on PPRV in Palestine. In Palestine, PPR infection outbreaks continued to occur every year (Alzuheir, 2019). As a result, vaccination campaigns have been implemented; however, the incidence rate of PPR infection was not significantly reduced. Therefore, identification of PPRV lineage is essential for understanding the epidemiology and control of this disease. This publication describes the pathological lesions associated with recent PPR outbreaks along with sequencing and neighbourhood-joining phylogenetic analysis of N gene of PPRV from field outbreaks in Palestine.

2 | MATERIALS AND METHODS

2.1 | Clinical samples

Peste des petits ruminants outbreaks investigated in this study occurred in two provinces in Palestine between 2017 and 2019 (Table 1, Figure 1). Tissue samples were obtained from sheep (n = 3) and goats (n = 7) submitted for necropsy from three farms from Nablus and Ramallah provinces as a part of the diagnostic service of the Department of Veterinary Medicine at An-Najah National University. The first and second farms from Nablus province contained only goats with a flock size of 150–300 animals, and the animals were over 4 months of age. Whereas, the third farm from Ramallah province contained sheep in close contact with cattle with a flock size of about 100 animals ranging from a few days to several months of age. All three farms were reported to be unvaccinated by the owners. The clinical suspicion of PPR infection was based on clinical signs which include high fever (>40°C), severe depression and anorexia, signs of pneumonia (dyspnoea and coughing with serous to mucopurulent nasal discharge), mouth lesions and evidence of severe enteritis (diarrhoea and dehydration). Samples were kept at the central diagnostic laboratory of the Department of Veterinary Medicine at An-Najah National University—Nablus for further analysis.

2.2 | Histopathology

After necropsy, obtained samples were fixed in 10% neutral-buffered formalin, routinely embedded in paraffin wax and stained with haematoxylin and eosin for histopathological examination following standard procedures (Bancroft & Stevens, 1990).

2.3 | RNA extraction, reverse transcription-polymerase chain reaction (RT-PCR) and sequencing

Tissue specimens were stored in phosphate-buffered saline (PBS) for RNA extraction and subsequent RT-PCR amplification. Total RNA was extracted from 25 mg of the intestine and lung of animals showing clinical signs using a Geneaid Total RNA Mini Kit (Geneaid Biotech) as per the manufacturer's protocols. RNA was reverse-transcribed and the PCR was performed using specific primers in one-step reverse transcription PCR using qScript XLT 1-Step RT-PCR (QuantaBio) according to the manufacturer's instructions. Primers selected on the N protein gene sequence were used as described by (Kwiatek et al., 2007). These primers allow the amplification of 351 bp to the 3' end of N gene of PPRV. The amplification cycle consisted of an initial denaturation at 94°C, for 2 min, followed by 30 cycles of 94°C for 30 s, 55°C for 30 s and 72°C for 30 s. The final cycle had an extension time of 72°C for 5 min. The amplified PCR

TABLE 1 Details of the samples employed in this study

Number	Date of collection	Province	Animal species	Number of the dead animals during the outbreak
1	May 2017	Nablus	Goat	63
2	April 2018	Nablus	Goat	40
3	February 2019	Ramallah	Sheep	14

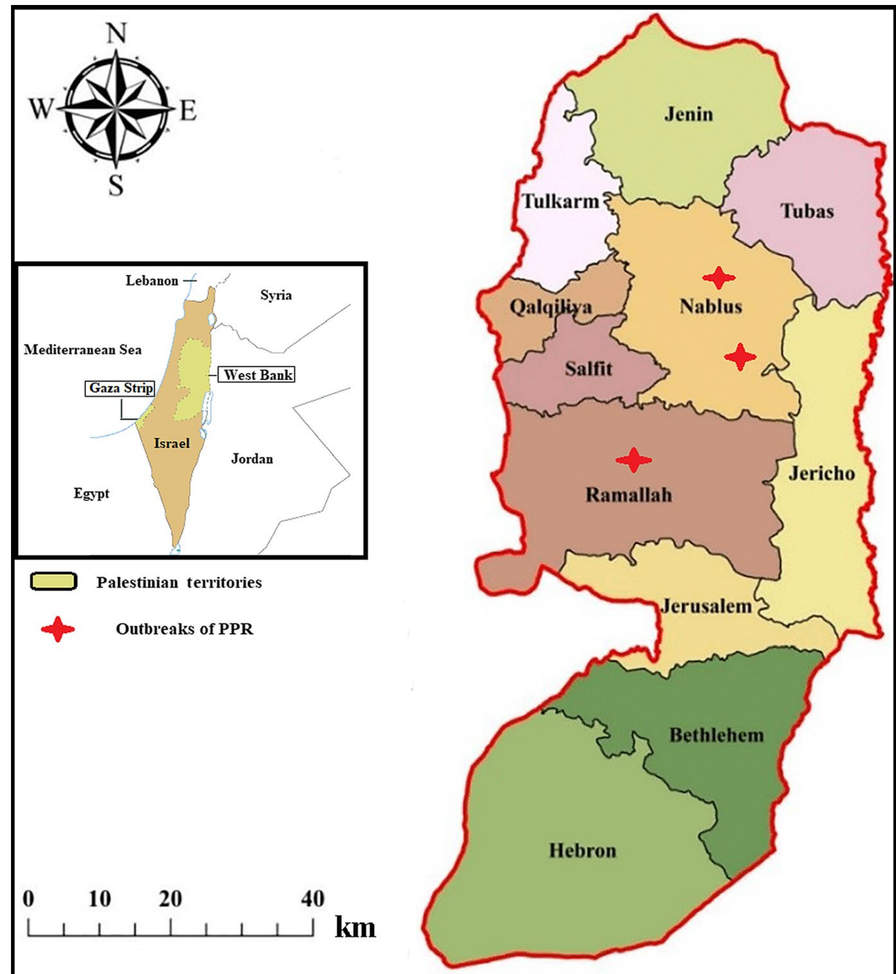

FIGURE 1 Map of Palestinian districts showing reported PPR outbreaks in the West Bank where samples of this study were taken

products were analysed by electrophoresis on a 1% agarose gel with ethidium bromide. Unpurified PCR products of the N gene (351 bp, obtained from NP3, NP4 primers) with the specific size were submitted to Sanger sequencing (Syntezza Bioscience, IDT) with the forward primers.

2.4 | Sequence analysis

The nucleotide sequence results were retrieved by Finch TV 1.4 (https://finchtv.software.informer.com/1.4/) software, aligned and edited with Vector NTI 9.1.0® (Invitrogen), compared with selected reference PPRV isolates of the different linage and geographical distribution. The phylogenetic tree was constructed by the neighbour-joining method using the MEGA 10.0.5 ® software (Kumar, Nei, Dudley, & Tamura, 2008) with 1,000 bootstrap replication. To assess the phylogenetic clustering and relationship among PPRV, the N gene sequences were aligned by ClustalW method (Kumar et al., 2008; Thompson, Gibson, & Higgins, 2003) and the phylogenetic tree was constructed using the neighbour-joining method with bootstrap values were determined by 1,000 replicates to assess the confidence level of each branch pattern using MEGA 10.0.5 ® software (Kumar et al., 2008).

3 | RESULTS

3.1 | Gross and histopathology

At necropsy, all submitted goats from the first and second farms were in good body condition, whereas lambs from the third farm were severely dehydrated and thin. The most common gross lesion observed (10/10) was erosive and ulcerative stomatitis with multiple 1- to 2-mm mucosal erosions and ulcers within the buccal cavity mainly on the soft and hard palate and tongue. Pathological changes observed in the small intestine were varied from distended intestinal loops with watery contents (3/10) to hyperaemic mucosa with blood-tinged intestinal contents (7/10). Mesenteric lymph nodes were enlarged and oedematous. In the respiratory tract, lungs in all cases from three outbreaks (10/10) were enlarged and oedematous with noticeable rib imprints on the pleural surfaces. In addition to this, most submitted cases (7/10) showed consolidation of anteroventral lobes with areas of fibrinous pleuritis. Moreover, flakes of mucopurulent discharge within the upper respiratory tract were seen. No additional lesions were observed.

Histologically, within the large and small intestine, the mucosa was infiltrated with large numbers of inflammatory cells

consisting of lymphocytes, macrophages and some neutrophils with severe loss of lining simple columnar epithelium in animals from all three outbreaks. The lumen of intestinal crypts was filled with moderate numbers of neutrophils and necrotic cell debris. Additionally, affected lambs from the third outbreak showed numerous rounds to ovoid basophilic structures measuring approximately 4–6 m in diameter were noted adherent to the mucosal surfaces of intestinal enterocytes (*Cryptosporidium parvum*). In the affected lungs from all animals, lung parenchyma, alveolar wall and interstitium were severely infiltrated with inflammatory cells consisting of lymphocytes with typical syncytial cells and occasional intranuclear eosinophilic inclusion bodies within syncytial cells and hyperplastic bronchial epithelium. Erosions and ulcers in the oral cavity from all infected animals showed multifocal hydropic degeneration and necrosis of stratum granulosum with mononuclear cell infiltration.

3.2 | Molecular characterization of PPRV isolated from tissue samples obtained from fields outbreaks

The reverse transcription PCR was performed on the extracted RNA from diseased animals showing clinical signs associated with PPR, and the products were analysed by agarose gel electrophoresis. All tissue samples (lung and intestine) obtained from sheep and goats from three outbreaks in Nablus and Ramallah provinces showed positive PCR reactions of correct sizes for the N gene-specific fragments. The expected size of the PPRV N gene amplicons was 351 bp (including primers) in agreement with the published nucleotide sequences (Kwiatek et al., 2007). One PCR product was excluded as the sequence result contains a non-informative background and was not clean for further analysis. Phylogenetic analysis was performed on 245 and 285 bp fragment of the N gene from two animals, each from different outbreaks. The sequences showed a 99.18% nucleotide homology were sent to GenBank and deposited under accession numbers MN813964 and MN813965.

For the phylogenetic tree construction; the Palestinian N gene nucleotide sequences and other reference PPRV isolates cover the four lineages from different geographical locations that were retrieved from the GenBank database. The phylogenetic analysis using the ClustalW method showing the clustering of the four lineages (Thompson et al., 2003).

Using BLAST program of the NCBI (Zhang, Schwartz, Wagner, & Miller, 2000), the obtained nucleotide sequences showed 97.93% nucleotide sequence identity with small ruminant *Morbillivirus* isolate TR/KONYA(Cumra)/2017/Sheep foetus/2825 detected in aborted sheep foetus in Turkey in (GenBank: MG744255.1) and small ruminant *Morbillivirus* isolate S4 isolated from goat in Iraq (GenBank: LT882725.1). All of these isolates are genotypically classified as lineage IV PPRV. Interestingly, strain sequences originating from Israel (DQ840173 and DQ840181), Iran (DQ840186) and Saudi Arabia (DQ840196) farther away in the linage IV clade (Figure 2).

4 | DISCUSSION

Small ruminants have a significant socio-economic value in Palestine. With approximately 730,894 sheep and 215,335 goats, they considered as the most abundant farm animal in Palestine (Palestinian central bureau of statistics, 2017). The rearing systems in Palestine include both intensive and extensive systems of production where small ruminants are kept in farms in rural areas with constant trading between farms (Palestinian central bureau of statistics, 2017).

Since the first report of PPR in Israel 1993 (Parida et al., 2015), several PPR outbreaks in sheep and goats were reported to the OIE in different locations within the country almost every year (Alzuheir, 2019). The presence of PPRV was confirmed in Palestine by using conventional RT-PCR by the local Palestinian veterinary services. Despite this, no available information exists regarding the virus circulating within the country due to the lack of available sequence data. This study is the first to carry out sequencing and sequence analyses of N gene from PPR viruses involved in the recent disease outbreak (2017–2019) in Palestine. In addition, we investigated the pathological and molecular characterization from three outbreaks in Palestine. In our study, all samples were taken from three outbreaks that occurred in two different provinces in Palestine during the period 2017–2019. Samples were collected from carcasses of sheep and goats with a history of typical clinical signs of the disease including pneumonia and diarrhoea as well as the typical gross and histological lesions. The pathological features reported in Palestinian outbreaks were similar to those described elsewhere (Chowdhury, Bhuiyan, Rahman, Siddique, & Islam, 2014; Kul, Kabakci, Atmaca, & Ozkul, 2007). Death in PPRV infected animals is attributed to dehydration resulted from enterocolitis and/or respiratory failure associated with pneumonia (Fentahun & Woldie, 2012). In our study, 100% of suspected animals tested with RT-PCR for PPRV infection were confirmed as positive. This can be attributed to the specific lesion for PPRV infection demonstrated in dead animals. For a better understanding of PPRV strains circulating in the region, phylogenetic analysis was performed based on the N gene partial sequence which has been previously identified as the best candidate to discriminate between different strains in comparison with F and H genes (Kwiatek et al., 2007). Sequence comparison showed a high level of homology (99.18%) of the circulating viruses suggesting that these viruses do not undergo rapid genetic changes in the N gene. This finding is in agreement with other studies (Güler, Şevik, & Hasöksüz, 2014; Liu et al., 2017). The phylogenetic tree constructed from N gene sequences available in GenBank and the partial sequence of the PPRV isolates from Palestine (Figure 2) clearly shows that the circulating PPRV in Palestine belongs to lineage IV which is most abundant PPRV lineage circulating in the Middle East, Asia and recently in Africa (Abd El-Rahim, Sharawi, Barakat, & El-Nahas, 2010; Fakri et al., 2016; Wang et al., 2009). Furthermore, this analysis reveals a strong genetic relationship with PPRV isolates from Turkey and Iraq which were reported in 2017, and this close

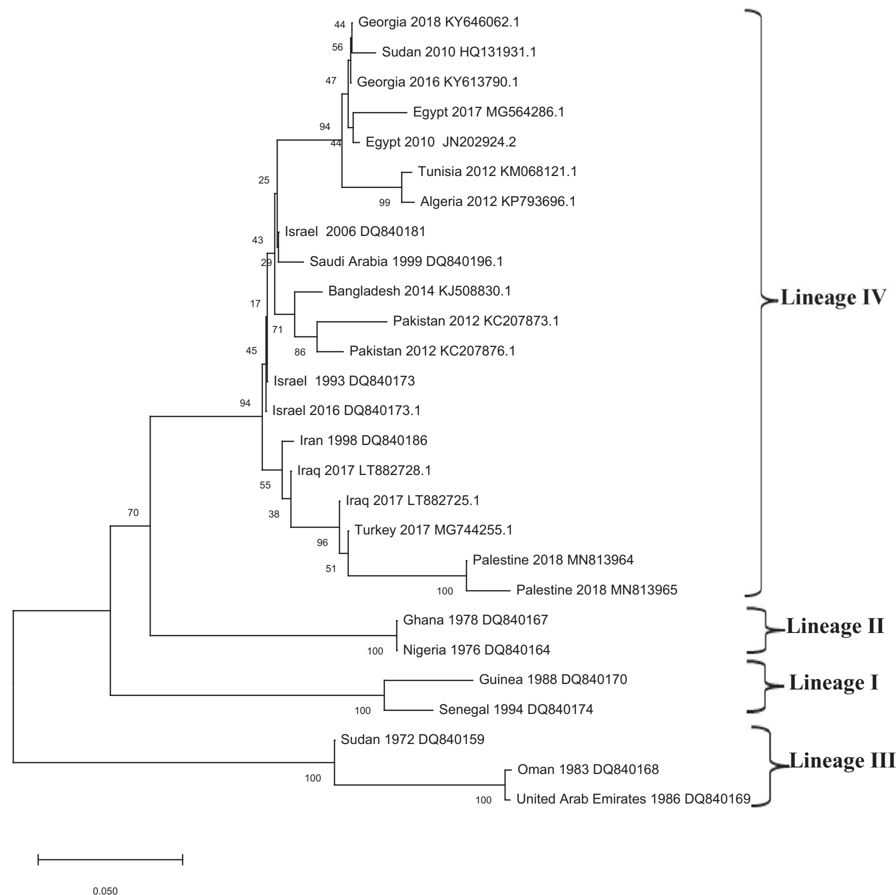

FIGURE 2 Phylogenetic analysis using the MEGAX software of partial nucleotide sequences from the amplified products of the PPRV N protein gene with different lineages occurring worldwide. The sequences from Palestine are shown with number code (2018 strain 1 and 2018 strain 2). The numbers indicate the bootstrap values calculated from 1,000 bootstrap replicates. The different lineages are shown

proximity of reported sequences despite the absence of common borders between Palestine and these countries suggests that the virus introduction could be occurred through trading of infected animals or their infected products (Parida et al., 2016). On the other hand, despite the legal and illegal small ruminant's trade between Palestine and Israel, the Palestinian PPRV isolates were distantly clustered from previously reported strains in Israel (Clarke et al., 2017). However, this cannot exclude the presence of other PPRV sequences currently circulating in Palestine indicating multiple introductions of PPRV into the country (Clarke et al., 2017). All of this may suggest a missing rout that may facilitate the circulating and emergences of PPRV in the region. As there are many PPR outbreaks for which there is unknown historical sequence data as well as the shortage of sequence data available from Palestine and the surrounding countries, determination of the exact routs of PPR transmission into Palestine is a challenge.

Control of PPR in Palestine is based on vaccination of susceptible adult animals as well as lambs and kids in farms where PPR outbreaks were reported using the live attenuated vaccine from PPRV strain Nigeria 75/1. However, many field PPR outbreaks are commonly misdiagnosed due to failure to submit the appropriate samples or the secondary infection such as pneumonic pasteurellosis which can be easily mistaken for PPR lesions (Smith & Sherman, 2009). In addition, the continued occurrence of PPR outbreaks in Palestine could be also attributed to other factors such as the restriction of vaccine administration by the Veterinary Services at the Ministry of Agriculture as well as the low vaccination rate (9%) which were reported during 2005–2017 (Alzuheir, 2019). All these factors together with uncontrolled movements of infected animals and their products across the country from infected to uninfected farms with the absence of a national mass vaccination programme may be responsible for the repeated PPRV outbreaks in Palestine.

In conclusion, PPR is endemic in Palestine with several PPR outbreaks occurring every year with limited information on the genetic nature of PPRV in these outbreaks. The described PPRV sequence in recent Palestinian outbreaks increases the information regarding PPRV strain in the country. However, future studies are needed to be performed at a national level to develop a complete picture of the circulating virus in the country in order to develop effective control measures to eradicate PPR in Palestine.

ACKNOWLEDGMENTS

This study was financially supported by An-Najah National University. The authors are grateful to Ms. R. Daibes, R. Daamaeh and I. Al-Qadi for excellent technical assistance.

ETHICS STATEMENT

Samples were obtained from cases sent to the Veterinary Clinic at the Department of Veterinary Medicine, An-Najah National University, for the diagnosis of the disease under the usual veterinary service work in Palestine. Approval for the use of samples in the research was obtained from the owners. In addition, the tissue samples were collected from dead animals only.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data available within the article or its supplementary materials (https://doi.org/10.1111/tbed.13535).

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REFERENCES

Abd El-Rahim, I. H., Sharawi, S. S., Barakat, M. R., & El-Nahas, E. M. (2010). An outbreak of peste des petits ruminants in migratory flocks of sheep and goats in Egypt in 2006. *Revue Scientifique Et Technique*, *29*, 655–662.

Alzuheir, I. (2019). Epidemiological study of Peste des Petits ruminants in sheep and goat during 2005–2017 in Palestine. *World's Veterinary Journal*, *9*, 70–75.

Amarasinghe, G. K., Bào, Y., Basler, C. F., Bavari, S., Beer, M., Bejerman, N., … Kuhn, J. H. (2017). Taxonomy of the order Mononegavirales: Update 2017. *Archives of Virology*, *162*, 2493–2504. https://doi.org/10.1007/s00705-017-3311-7

Bancroft, J. D., & Stevens, A. (1990). *Theory and practice of histological techniques* (3rd ed.). New York, NY: Churchill Livingstone, Edinburgh.

Banyard, A. C., Parida, S., Batten, C., Oura, C., Kwiatek, O., & Libeau, G. (2010). Global distribution of peste des petits ruminants virus and prospects for improved diagnosis and control. *Journal of General Virology*, *91*, 2885–2897. https://doi.org/10.1099/vir.0.025841-0

Banyard, A. C., Wang, Z., & Parida, S. (2014). Peste des petits ruminants virus, eastern Asia. *Emerging Infectious Diseases*, *20*, 2176–2178. https://doi.org/10.3201/eid2012.140907

Baron, M. D., Parida, S., & Oura, C. A. (2011). Peste des petits ruminants: A suitable candidate for eradication? *Veterinary Record*, *169*, 16–21. https://doi.org/10.1136/vr.d3947

Chowdhury, E. H., Bhuiyan, A. R., Rahman, M. M., Siddique, M. S., & Islam, M. R. (2014). Natural peste des petits ruminants virus infection in Black Bengal goats: Virological, pathological and immunohistochemical investigation. *BMC Veterinary Research*, *10*, 263. https://doi.org/10.1186/s12917-014-0263-y

Clarke, B., Mahapatra, M., Friedgut, O., Bumbarov, V., & Parida, S. (2017). Persistence of Lineage IV Peste-des-petits ruminants virus within Israel since 1993: An evolutionary perspective. *PLoS ONE*, *12*, e0177028. https://doi.org/10.1371/journal.pone.0177028

Couacy-Hymann, E., Roger, F., Hurard, C., Guillou, J. P., Libeau, G., & Diallo, A. (2002). Rapid and sensitive detection of peste des petits ruminants virus by a polymerase chain reaction assay. *The Journal of Virological Methods*, *100*, 17–25. https://doi.org/10.1016/S0166-0934(01)00386-X

Fakri, F., Embarki, T., Parida, S., Bamouh, Z., Jazouli, M., Mahapatra, M., … Elharrak, M. (2016). Re-emergence of Peste des Petits Ruminants virus in 2015 in Morocco: Molecular characterization and experimental infection in Alpine goats. *Veterinary Microbiology*, *197*, 137–141. https://doi.org/10.1016/j.vetmic.2016.11.006

Fentahun, T., & Woldie, M. (2012). Reviewon Peste Des Petits Ruminants (PPR). *European Journal of Applied Sciences*, *4*, 160–167. https://doi.org/10.5829/idosi.ejas.2012.4.4.66151

Forsyth, M. A., & Barrett, T. (1995). Evaluation of polymerase chain reaction for the detection and characterisation of rinderpest and peste des petits ruminants viruses for epidemiological studies. *Virus Research*, *39*, 151–163. https://doi.org/10.1016/0168-1702(95)00076-3

Gargadennec, L., & Lalanne, A. (1942). Peste Des Petits Ruminants [in French]. *Bulletin Des Services Zootechniques Et Des Epizzoties De L'afrique Occidentale Francaise*, *5*, 16–21.

Gibbs, E. P., Taylor, W. P., Lawman, M. J., & Bryant, J. (1979). Classification of peste des petits ruminants virus as the fourth member of the genus Morbillivirus. *Intervirology*, *11*, 268–274. https://doi.org/10.1159/000149044

Güler, L., Şevik, M., & Hasöksüz, M. (2014). Phylogenetic analysis of peste des petits ruminants virus from outbreaks in Turkey during 2008–2012. *Turkish Journal of Biology*, *38*, 671–678. https://doi.org/10.3906/biy-1401-28

Kul, O., Kabakci, N., Atmaca, H. T., & Ozkul, A. (2007). Natural peste des petits ruminants virus infection: Novel pathologic findings resembling other morbillivirus infections. *Veterinary Pathology*, *44*, 479–486. https://doi.org/10.1354/vp.44-4-479

Kumar, K. S., Babu, A., Sundarapandian, G., Roy, P., Thangavelu, A., Kumar, K. S., … Parida, S. (2014). Molecular characterisation of lineage IV peste des petits ruminants virus using multi gene sequence data. *Veterinary Microbiology*, *174*, 39–49. https://doi.org/10.1016/j.vetmic.2014.08.031

Kumar, S., Nei, M., Dudley, J., & Tamura, K. (2008). MEGA: A biologist-centric software for evolutionary analysis of DNA and protein sequences. *Briefings in Bioinformatics*, *9*, 299–306. https://doi.org/10.1093/bib/bbn017

Kwiatek, O., Ali, Y. H., Saeed, I. K., Khalafalla, A. I., Mohamed, O. I., Obeida, A. A., … Libeau, G. (2011). Asian lineage of peste des petits ruminants virus, Africa. *Emerging Infectious Diseases*, *17*, 1223–1231. https://doi.org/10.3201/eid1707.101216

Kwiatek, O., Minet, C., Grillet, C., Hurard, C., Carlsson, E., Karimov, B., … Libeau, G. (2007). Peste des petits ruminants (PPR) outbreak in Tajikistan. *Journal of Comparative Pathology*, *136*, 111–119. https://doi.org/10.1016/j.jcpa.2006.12.002

Lefevre, P. C., Diallo, A., Schenkel, F., Hussein, S., & Staak, G. (1991). Serological evidence of peste des petits ruminants in Jordan. *Veterinary Record*, *128*, 110. https://doi.org/10.1136/vr.128.5.110

Liu, Y., Yang, B., Li, X., Wang, X., Zhang, L., Li, F., & Zhao, L. (2017). Confirmation and Sequence analysis of N gene of PPRV in South Xinjiang, China. *Arquivo Brasileiro De Medicina Veterinária E Zootecnia*, *69*, 1105–1113. https://doi.org/10.1590/1678-4162-9277

Mornet, P., Orue, J., & Gilbert, Y. (1956). Unicity and plasticity of cattle plague virus; a natural virus adapted to small ruminants. *Comptes Rendus Hebdomadaires Des Seances De L'academie Des Sciences*, *242*, 2886.

Nanda, Y. P., Chatterjee, A., Purohit, A. K., Diallo, A., Innui, K., Sharma, R. N., … Taylor, W. P. (1996). The isolation of peste des petits ruminants virus from northern India. *Veterinary Microbiology*, *51*, 207–216. https://doi.org/10.1016/0378-1135(96)00025-9

Palestinian Central Bureau of Statistics. (2017). *Livestock survey, 2013 – Main results* (p. 63). Ramallah, Palestine: Palestinian Central Bureau of Statistics.

Parida, S., Muniraju, M., Altan, E., Baazizi, R., Raj, G. D., & Mahapatra, M. (2016). Emergence of PPR and its threat to Europe. *Small Rumin Res*, *142*, 16–21. https://doi.org/10.1016/j.smallrumres.2016.02.018

Parida, S., Muniraju, M., Mahapatra, M., Muthuchelvan, D., Buczkowski, H., & Banyard, A. (2015). Peste des petits ruminants. *Veterinary Microbiology*, *181*, 90–106. https://doi.org/10.1016/j.vetmic.2015.08.009

Shaila, M. S., Purushothaman, V., Bhavasar, D., Venugopal, K., & Venkatesan, R. A. (1989). Peste des petits ruminants of sheep in India. *Veterinary Record*, *125*, 602. https://doi.org/10.1136/vr.125.24.602-a

Smith, M. C., & Sherman, D. M. (2009). *Digestive system: Goat medicine* (2nd ed.). Hoboken, NJ: John Wiley & Sons.

Thompson, J. D., Gibson, T. J., & Higgins, D. G. (2003). Multiple sequence alignment using ClustalW and ClustalX. *Current Protocols in Bioinformatics*, *2.3.*, 1–2.3. 22. https://doi.org/10.1002/0471250953.bi0203s00

Wang, Z., Bao, J., Wu, X., Liu, Y., Li, L., Liu, C., … Wang, J. (2009). Peste des petits ruminants virus in Tibet, China. *Emerging Infectious Diseases*, *15*, 299–301. https://doi.org/10.3201/eid1502.080817

Zhang, Z., Schwartz, S., Wagner, L., & Miller, W. (2000). A greedy algorithm for aligning DNA sequences. *Journal of Computational Biology : A Journal of Computational Molecular Cell Biology*, *7*, 203–214. https://doi.org/10.1089/10665270050081478

How to cite this article: Fayyad A, Alzuheir I, Jalboush N. A molecular and pathological study of peste des petits ruminants virus (PPRV) from field outbreaks in Palestine, 2017–2019. *Transbound Emerg Dis*. 2020;00:1–7. https://doi.org/10.1111/tbed.13535