



Pathomolecular Studies of Porcine Parvovirus in Assam

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ABSTRACT

Background: Porcine parvovirus (PPV) is a key etiological agent of reproductive failure in swine, resulting in considerable economic losses for the pig farming industry.

Methods: The present study was conducted to investigate the molecular prevalence, genetic characteristics, seroprevalence and associated pathological alterations in pigs from Assam, India.

Result: Out of the 82 tissue samples screened by PCR targeting 226 bp specific to VP2 gene encoding capsid protein, 9 (10.97%) were found positive for PPV. These samples consisted mainly of pooled aborted fetal materials. Subsequently, next-generation sequencing of a representative clinical sample was carried out. Phylogenetic analysis of VP1 and NS1 genes revealed a high degree of nucleotide identity (99.77-100%) with contemporary PPV strains from China and South Korea. Phylogenetic clustering suggested that the VP1 sequence belongs to the 27a/27a-like lineage. Gross pathological alterations in aborted fetuses included hydrothorax and visceral congestion, whereas histopathology revealed necrosis in multiple organs and lymphoid depletion, which is suggestive of systemic viral effects. Serological screening of 224 pigs using a commercial ELISA kit divulged a seroprevalence of 23.21%, indicating widespread exposure to PPV among both healthy and diseased animals. These combined findings confirm the circulation of potentially virulent PPV strains in Assam and highlight the need of frequent surveillance, which will help in devising appropriate control strategies to mitigate reproductive losses in the swine population.

Key words: Pathology, Phylogeny, Porcine parvovirus, Seroprevalence, VP1.

INTRODUCTION

Porcine parvovirus 1 (PPV) is an economically significant viral pathogen affecting pigs globally. Porcine parvovirus (PPV) was first isolated from Germany in 1965 as a contaminant in porcine kidney cell culture (Yang *et al.*, 2021). The virus is small, non-enveloped and has a genome of approximately 5 kb composed of negative-sense, single-stranded DNA (Souza *et al.*, 2019). The virus causes SMEDI syndrome, a condition in pigs characterized by stillbirth, mummification, embryonic death and infertility (Tamás *et al.*, 2022). It is considered as one of the major causes contributing to infertility in pigs (Mészáros *et al.*, 2017). Moreover, PPV is also recognized for its role in potentiating the severity of postweaning multisystemic wasting syndrome (PWMS) caused by porcine circovirus 2 (PCV2) (Kim *et al.*, 2006). In addition to PPV1, six novel porcine parvoviruses, designated PPV2 through PPV7, have emerged over the past two decades (Streck and Truyen, 2020). Recently, another novel porcine parvovirus designated PPV8 has been reported from several countries (Chen *et al.*, 2025; Igriczi *et al.*, 2024). The International Committee on Taxonomy of Viruses (ICTV) recently revised the classification of porcine parvoviruses, assigning PPV1 within the genus Protoparvovirus as Ungulate protoparvovirus 1 (Karuppannan and Opriessnig, 2018).

The approximately 5 kb genome of PPV contains two open reading frames (ORFs): ORF1, located at the 5' end, encodes three non-structural proteins (NS1, NS2 and NS3) critical for the replication cycle, while ORF2, found at the 3' end, encodes three structural proteins (VP1, VP2 and VP3)

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(Bergeron *et al.*, 1993). VP1 and VP2 differ solely in their amino-terminal regions, with both proteins produced via alternative splicing of a single gene, while VP3 is generated through post-translational processing of VP2 (Simpson *et al.*, 2002). The role of structural proteins, of which VP2 is the major one, is helping the virus to establish infection by recognizing and binding to the host cell (Simpson *et al.*, 2002). VP2, the major protective antigen, contains the primary neutralizing epitope that elicits the production of neutralizing antibodies (Souza *et al.*, 2019).

The significance of PPV has been documented from some parts of India, with studies showing its prevalence and impact on pig health. The first report of PPV in India

was by Sharma and Saikumar in 2010, where the pathogen was associated with reproductive failure and neonatal mortality in crossbred pigs. The first documented occurrence and molecular characterization of PPV in southern India was reported in domestic and wild pig populations from Kerala (Aishwarya *et al.*, 2016). Another study reported a PPV positivity rate of 14.3% in aborted and stillborn pig fetuses from Tamil Nadu and Kerala (Parthiban *et al.*, 2022). Lalrinkima *et al.* (2022) found a sero-positivity of 74.7% for PPV in organized farms of Tamil Nadu. Research by Kaur *et al.* (2016) found a seroprevalence of 41.1% and confirmed the presence of PPV in fetus, placenta and female reproductive samples in Punjab. In northeastern India, a previous study reported the presence of PPV in 14.8% of tissue samples from aborted fetuses (Pegu *et al.*, 2017a). Additionally, a seropositivity of 10.15% for PPV was also found in the northeastern states (Pegu *et al.*, 2017b). Parvoviruses, unlike other DNA viruses, have relatively higher evolution rates similar to that of RNA viruses. It is estimated to range from 10^{-5} to 10^{-4} substitution sites⁻¹ year⁻¹ (Oh *et al.*, 2017). Also, reproductive conditions such as abortions, stillbirths, mummifications, *etc.* occur at an alarming rate in northeast India, where majority of the pig population of the country resides. Virulent PPV1 lineages can cross national borders and pose a significant economic threat to global swine production. Recent phylogeographic studies have highlighted the pace at which these emerging strains can circulate across regions (Franzo *et al.*, 2023). So, it becomes imperative to monitor such pathogens in the field so that appropriate diagnostic tools and control strategies can be devised. So, the present study deals with the identification of PPV in reproductive disorder related cases and surveillance for the presence of antibodies against PPV.

MATERIALS AND METHODS

Clinical cases and sampling

The present work was carried out at ICAR-National Research Centre on Pig, Guwahati, Assam. Sample collection was done from different parts of Assam in 2022-2024. A total of 82 tissue samples of fetuses including lungs, liver, lymph nodes and kidney were aseptically collected from abortion, still births, mummification and respiratory ailment cases of pigs (Fig 1). Tissue samples were processed to prepare a 10% (w/v) suspension in phosphate-buffered saline (PBS; pH 7.2), followed by homogenization with sterile sand using a pestle and mortar. The homogenized material was centrifuged at 6000 rpm for 10 mins to allow the tissue debris to settle. Supernatant from each tissue was separated and used for further steps.

DNA isolation and PCR confirmation

DNA was isolated from the supernatant of each tissue sample, as described above, using the Qiagen DNeasy Blood and Tissue Kit according to the manufacturer's instructions. The extracted DNA was then used as a

template in PCR to confirm the presence of PPV in the samples. PCR amplification was carried out using the published primer pairs 5'-CCAGCAGCTAACACAAGAAAAGGTTATCAC-3' (forward) and 5'-GTCCATGTTGGTAATCCATTGTAAATC-3' (reverse) as described previously by Arnauld *et al.*, 1998. Briefly, the PCR reaction mixture comprised 10 µL of EmeraldAmp PCR Master Mix (Takara Bio), 1 µL each of the forward and reverse primers, 1 µL of extracted template DNA and nuclease-free water to make up a final volume of 20 µL. The PCR cycling conditions were as follows: an initial denaturation at 98°C for 2 minutes, followed by 30 cycles of denaturation at 98°C for 10 seconds, annealing at 55°C for 30 seconds and extension at 72°C for 20 seconds, with a final extension step at 72°C for 5 minutes. Upon completion, the PCR products were subjected to 1% agarose gel electrophoresis for visualization and documentation.

Next generation sequencing and NGS Raw data analysis

Next generation sequencing of one of the positive aborted materials was performed using commercial outsourcing and the data thus obtained was further analyzed. The pair-end (PE) raw reads were checked for quality using FastQC tool version 0.11.9. The poor-quality reads were removed by using trimmomatic software version 11.0.20.1. High quality reads with a phred score of more than 30 were proceeded for alignment via BWA aligner software using PPV reference genome (NCBI Accession no. NC_001718.1) (Li *et al.*, 2009). The mapped reads were extracted in a sorted bam file and used for filtering out viral reads into two fastq files from the raw reads (forward and reverse). Lastly, the obtained fastq PE reads were proceeded for de novo genome assembly to generate the contigs using SPAdes version 3.13.1. The obtained fasta sequences were proceeded for BLAST search to determine the sequence identity and annotation followed by sequence submission to NCBI.

Phylogenetic analysis

A total of 93 PPV genome sequences collected from different countries were retrieved from GenBank for phylogenetic



Fig 1: Aborted fetuses of pigs showing characteristic features of SMEDI syndrome (Stillbirth, Mummification, Embryonic Death and Infertility).

studies. Partial sequences of the genes obtained in the current study were considered for phylogenetic tree construction. Multiple sequence alignment of the genomes was performed using MAFFT software version 7.505 (Kato *et al.*, 2002). The aligned FASTA sequences were used to identify the best-fit substitution model by selecting the one with the lowest Akaike Information Criterion (AIC) score, as implemented in MEGA 11 (Tamura *et al.*, 2021). Maximum Likelihood method using HKY+G+I model with 1000 bootstrap replicates was employed for phylogenetic tree generation in MEGA 11 software. The ultimate tree is generated by midpoint rooting in MEGA 11.

Gross and histopathological studies

All collected tissue samples, including aborted fetal materials, lungs, lymph nodes, liver, kidney and spleen, were grossly examined before further processing. The specimens were carefully inspected for any visible lesions, discoloration, enlargement, hemorrhages and other abnormalities suggestive of disease processes. Representative tissue samples from lungs, lymph nodes, spleen and aborted materials were fixed in 10% formalin for 48 hours. Post-fixation, tissue samples were processed through a series of graded alcohols, cleared in xylene and subsequently embedded in paraffin wax. Serial sections of 4-5 μm thickness were then prepared using a rotary microtome and mounted onto

glass slides for further analysis. The sections were subsequently stained with hematoxylin and eosin (H and E) using standard histological procedures. Stained slides were examined under a light microscope for microscopic lesions and the changes were recorded.

Seroprevalence studies

To study the serological prevalence of PPV in the region, serum samples from both healthy and diseased pigs were

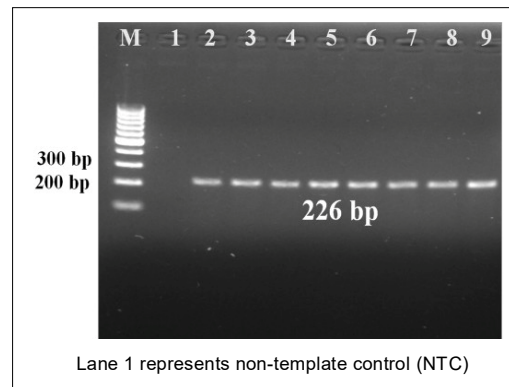


Fig 2: Agarose gel electrophoresis of clinical samples where in an amplicon of 226 bp were obtained in positive samples (Lanes 2-9).

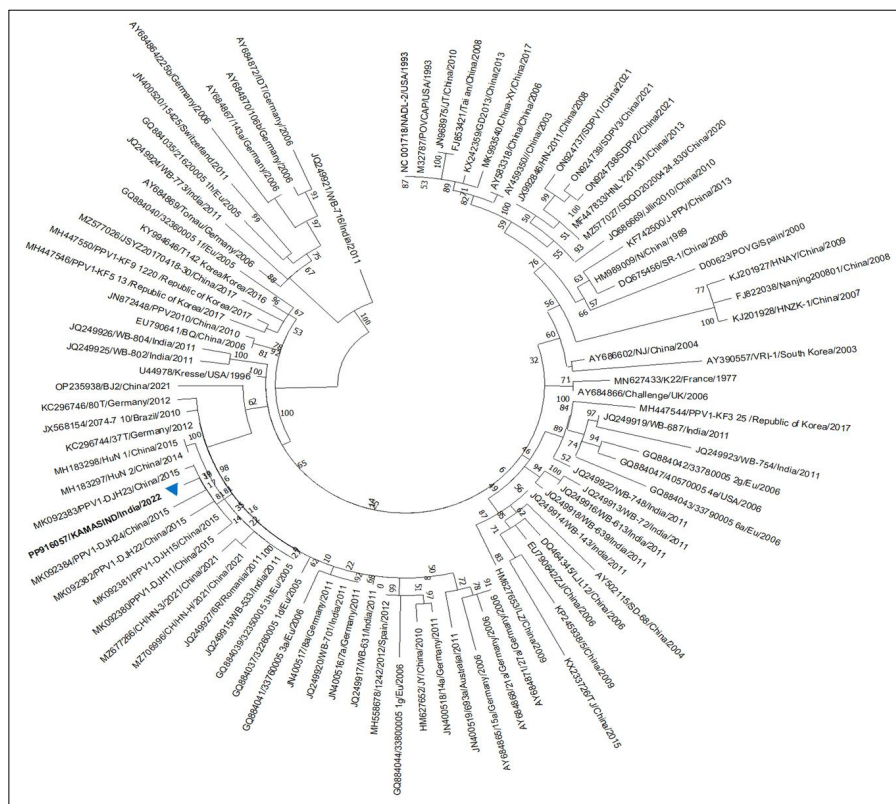


Fig 3: Phylogenetic analysis of the partial nucleotide sequence of capsid protein 1 (VP1 gene, NCBI Accession no. PP916057) along with 93 PPV sequences revealed that the strain under study was grouped with 27a and 27a like strains with 98% of bootstrap support.

collected. Blood samples from 224 pigs were collected during the same period as mentioned before. Approximately 3 ml of blood was obtained from the ear vein of each pig using clot activator tubes and centrifuged at 2,500 rpm for 15 minutes to isolate serum. All experimental procedures were performed with the prior approval of the Institutional Animal Ethics Committee of the Institute. Serum was extracted from each sample and stored at -20°C until analysis. The serum samples were subsequently screened for antibodies against PPV using the PrioCHECK™ Porcine Parvovirus Ab Plate Kit commercial ELISA kit.

RESULTS AND DISCUSSION

PCR Confirmation of PPV

A total of 9 samples out of 82 collected tissue samples were confirmed positive by PCR making 10.97% prevalence of PPV in Assam (Fig 2). The positive samples predominantly came from the pooled aborted cases. Therefore, the molecular prevalence of PPV in the present study was found to be 10.97%. These findings align with earlier studies reporting similar prevalence levels in different Indian states, highlighting the widespread circulation of PPV in pig populations in both southern and northeastern parts of India (Pegu *et al.*, 2017a; Parthiban *et al.*, 2022).

Phylogenetic analysis

In silico analysis of the obtained raw reads generated four contigs with the size of 427, 869, 552 and 487 nts encoding

for 142, 289, 183 and 162 amino acids, respectively. The BLAST analysis revealed that the first sequence possesses 99.77% sequence identity with PPV isolate SDLC202109 (OR452191, China), while the second sequence has 99.88% sequence homology with PPV 1 isolate DJH22 (MK092382, China) and both sequences represent specific regions of *VP1* gene coding for capsid protein 1. The latter two sequences hold 99.82% and 100% sequence similarity with PPV strain 17KWB38 (MT846932, South Korea) and PPV isolate T142_Korea (KY994646, South Korea), respectively and belong to the regions of *NS1* gene coding for non-structural protein 1. Partial sequences of both the genes *viz.* *VP1* and *NS1* are available in NCBI database with the accession numbers of PP916057 and PP916058, respectively. Phylogenetic analysis of the partial nucleotide sequence of capsid protein 1 (*VP1* gene) along with 93 PPV sequences revealed that the strain under study was grouped with 27a and 27a like strains with 98% of bootstrap support (Fig 3). Similarly, for *NS1* gene, the current strain was found to be more closely related to the strain of Germany (27a) (Fig 4). Previously, comparative sequence analysis between the pathogenic and non-pathogenic strains showed that all the observed changes belong to silent mutations (Streck and Truyen, 2020). Further, another study reported that the substitutions were majorly associated with the capsid protein, hence, impacting the receptor binding capacity as well as antigenicity (Streck *et al.*, 2015).

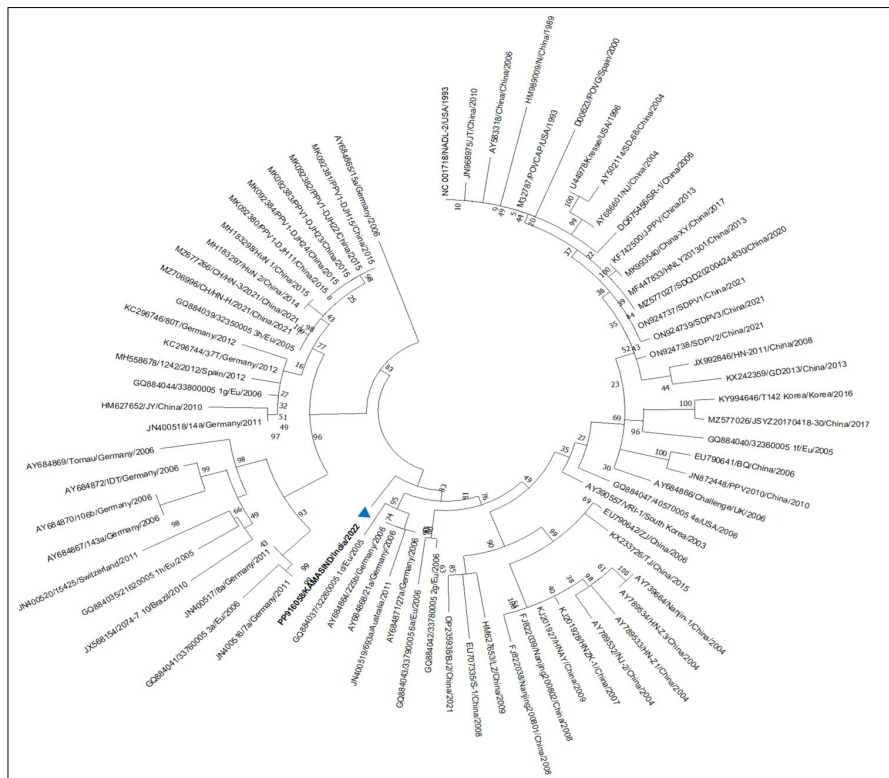


Fig 4: Phylogenetic analysis of the partial nucleotide sequence of non-structural protein 1 (*NS1* gene, NCBI Accession no. PP916058) revealed grouping with 27a and 27a like strains.

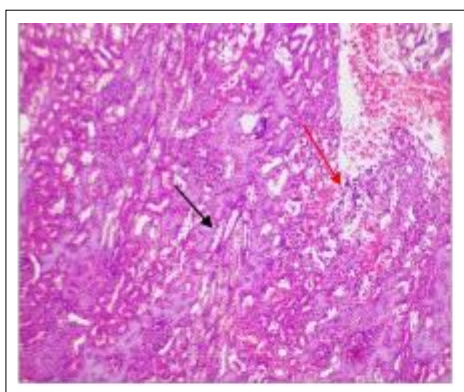


Fig 5a: Histopathological section of fetal pig kidney showing interstitial nephritis with tubular degeneration (black arrow), focal interstitial hemorrhages, and infiltration of mononuclear inflammatory cells (red arrow) H& E, 10X.

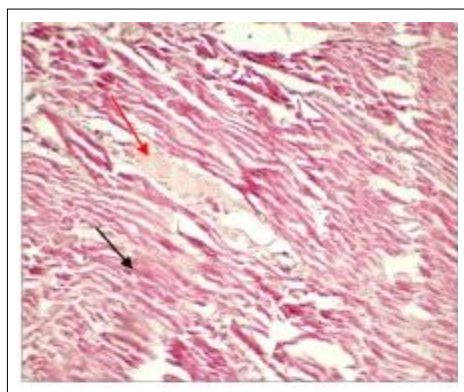


Fig 5b: Histopathological section of fetal pig heart showing myocardial degeneration (black arrow), focal hemorrhages (red arrow) H and E, 10X.

Therefore, in the current study, grouping of *VP1* gene with 27a-like strains might possess a significant evolutionary effect on the viral adaptation as the 27a-like strains have been associated with enhanced pathogenicity and possible vaccine escape due to antigenic variation (Zeeuw *et al.*, 2007). The high sequence conservation across different geographic regions may indicate either global dissemination or a common ancestral lineage, calling for closer molecular surveillance and strain characterization. The need for robust molecular surveillance is further emphasized by recent comprehensive studies, which reveal that a significant portion of PPV-positive samples involve co-infections with multiple emerging parvovirus genotypes (Zhao *et al.*, 2024).

Pathological studies

Gross pathology: In the present study, PPV-infected gilt exhibited significant reproductive disorder, including late-term abortion, stillbirths and the presence of mummified and decomposed fetuses (Fig 1). Gross pathological

changes observed in the infected fetuses included the accumulation of blood-tinged, straw-colored fluid within the body cavities. Additionally, the visceral organs showed signs of moderate congestion and necrotic lesions, which were indicative of the systemic effects of the viral infection on fetal tissues. Reproductive failures in swine due to porcine parvovirus (PPV) infection have been extensively documented in both natural and experimental settings. Natural infections with porcine parvovirus (PPV) are well-established as a primary cause of reproductive failure in swine, especially in immunologically naive gilts and sows. Earlier researchers have reported field outbreaks of PPV leading to embryonic and fetal death, manifested clinically as infertility, mummified fetuses, stillbirths and early abortions, commonly grouped under the term SMEDI syndrome (Pegu *et al.*, 2017a; Lalrinkima *et al.*, 2025).

Histopathological studies: Histopathological examination of the aborted fetuses revealed prominent necrotic changes in the cellular architecture of several developing organs, including the kidneys (Fig 5a), heart (Fig 5b), lymph nodes (Fig 5c) and lungs (Fig 5d). Necrosis was most notable in parenchymal cells, reflecting the virus's affinity for rapidly dividing tissues. In addition, some fetuses exhibited marked lymphoid depletion, particularly within the follicular regions of the lymph nodes, indicating immunosuppression or impaired lymphoid organ development associated with intrauterine viral infection. These findings are in agreement with experimental infections and field reports, which highlight similar histological alterations in fetuses affected by PPV (Joo *et al.*, 1977; Sairam *et al.*, 2019).

Seroprevalence of PPV

A total of 224 serum samples, collected from both healthy and diseased pigs, were screened for the presence of antibodies against PPV using the PrioCHECK™ Porcine Parvovirus Ab ELISA kit. Out of the 224 samples tested, 52 samples (23.21%) were found seropositive, indicating exposure to PPV in the swine population of the region. The seropositive samples were detected in both healthy and diseased animals. The overall seroprevalence suggests widespread circulation of PPV among pigs in Assam. All tests were performed as per the manufacturer's instructions and quality controls provided in the kit yielded expected results, confirming assay validity. Comparable levels of seroprevalence have been reported in other regions of India and Southeast Asia, supporting the notion that PPV remains enzootic in many pig populations (Pegu *et al.*, 2017b; Deka *et al.*, 2021). Some of the studies have detected even higher prevalence (Kaur *et al.*, 2016). The detection of antibodies in apparently healthy pigs further highlights the challenge in early diagnosis and the importance of routine sero-surveillance. In addition, the presence of genetically similar but possibly antigenically divergent strains such as 27a-like variants warrants reviewing current vaccination strategies, especially in breeding herds.

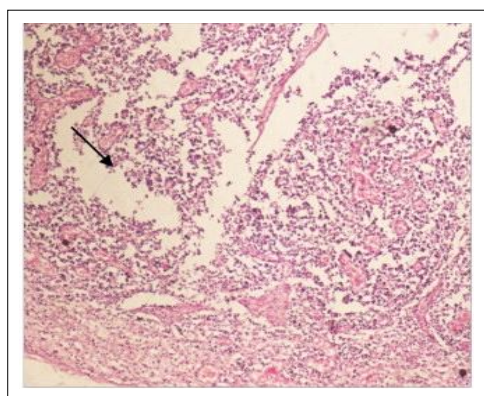


Fig 5c: Histopathological section of fetal pig lymph node showing follicular degeneration in the lymphoid follicle (arrow) H&E, 20X.

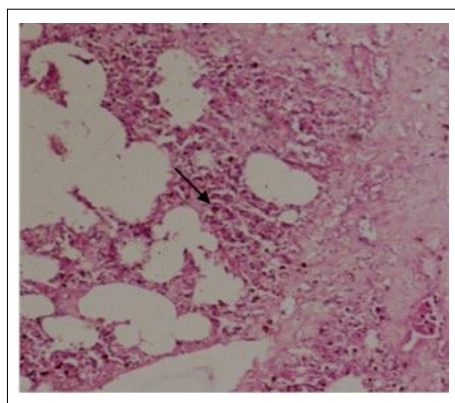


Fig 5d: Histopathological section of fetal pig lung showing interstitial pneumonia with infiltrating cells and edema in the inter alveolar septa (arrow), H&E, 20X.

CONCLUSION

This study provides integrated evidence of the molecular, serological and pathological presence of PPV in pigs in Assam. The molecular detection of PPV, high sequence identity with Asian strains, typical pathological changes in fetuses and widespread seroprevalence altogether emphasize the endemic nature of PPV in the region. Continuous molecular surveillance by using improved diagnostic methods and stringent biosecurity practices can be applied to avoid the reproductive and economic losses caused by this virus. There is also a need for an indigenous vaccine to curb the economic losses caused by this virus.

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Disclaimers

The views and conclusions expressed in this article are solely those of the authors and do not necessarily represent the views of their affiliated institutions. The authors are responsible for the accuracy and completeness of the information provided, but do not accept any liability for any direct or indirect losses resulting from the use of this content.

Informed consent

All animal procedures for experiments were approved by the committee for the purpose of control and supervision of experiments on animals (CCSEA) and handling techniques were approved by the institutional animal ethics committee (IAEC).

Conflict of interest

The authors declare that there are no conflicts of interest regarding the publication of this article. No funding or sponsorship influenced the design of the study, data collection, analysis, decision to publish, or preparation of the manuscript.

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