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Porcine Parvoviruses: Classical and Novel Insights into Biology, Pathogenesis, Diagnosis, and Epidemiology

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Abstract

Porcine parvoviruses (PPVs) represent a diverse group of DNA viruses that significantly impact swine health, particularly reproductive performance. While PPV1 is a well-established cause of SMEDI syndrome (stillbirth, mummification, embryonic death, and infertility), the discovery of novel PPVs (nPPVs: PPV2–PPV8) has expanded the scope of viral epidemiology and pathogenesis in pigs. This article synthesizes current knowledge of both classical and novel PPVs, focusing on their molecular biology, taxonomy, evolution, clinical implications, diagnostic tools, and control strategies. Notably, nPPVs show considerable genetic diversity and potential for recombination, especially in high-density pig populations and through interaction with wild boars. Although the pathogenicity of nPPVs remains less defined, associations with respiratory and reproductive disorders have been suggested. Current diagnostic methods include serology, PCR and immunohistochemistry, while prevention relies heavily on inactivated vaccines and strict biosecurity. The emergence of genetically distinct PPVs and potential vaccine escape variants underscores the need for enhanced molecular surveillance and updated immunization strategies to mitigate the impact of these evolving pathogens on swine production.

Introduction

Pig husbandry is vital in India's agricultural sector, with a 9.055 million pig population (20th Livestock Census). Pigs convert agricultural by products and organic waste into premium pork, aiding in resource reuse. However, farmers face challenges like limited technical expertise, poor infrastructure and insufficient support services, such as veterinary care and market linkages, in areas where pig farming is prevalent. Pig illnesses pose a significant threat to the Indian agricultural sector, causing significant economic losses. This includes porcine parvovirus, classical swine fever, African swine fever, porcine reproductive and respiratory syndrome and swine influenza virus. To mitigate this, farmers must adopt biosecurity protocols, maintain sanitation standards, and seek veterinary support. Despite efforts, porcine infections remain a significant concern in Northeast India. This article contains a brief overview of porcine parvovirus infection which mainly cause respiratory and reproductive symptoms in pigs.

Molecular Biology and Classification

PPVs are small, non-enveloped viruses with a linear, single-stranded DNA genome of approximately 4–6 kb, flanked by palindromic hairpin structures essential for replication (Cotmore and Tattersall, 2006). The genome typically contains two main open reading frames (ORFs): ORF1 encodes non-structural proteins (NS1, NS2), crucial for replication (Zádori et al., 2005), while ORF2 encodes structural capsid proteins (VP1, VP2, sometimes VP3) (Simpson et al., 2002). The capsid is composed of 60 subunits, arranged in icosahedral symmetry, with VP2 being predominant (Chapman and Rossmann, 1993).

Taxonomically, PPV1 is classified as *Ungulate protoparvovirus* (Truyen and Streck, 2012). nPPVs are distributed among various genera and subfamilies: PPV2 and PPV3 in *Tetraparvovirus*, PPV4 and PPV6 in *Copiparvovirus*, and PPV7 in *Chaphamaparvovirus* (Tijssen et al., 2011). Notably, PPV4's genome is circular, unlike the linear genomes of other PPVs (Cheung et al., 2010).

Epidemiology and Prevalence

PPV1 is endemic worldwide and remains a major cause of reproductive failure, particularly in unvaccinated or partially immune herds (Mengeling et al., 2000). It is highly stable in the environment, resisting inactivation by many common disinfectants (Eterpi et al., 2009). Boars may play a role in transmission, as PPV has been detected in semen (Kim et al., 2003).

Serological surveys, such as those in Punjab, India, demonstrate a high prevalence (41.1%) of PPV antibodies in both sexes, consistent with global observations (Roic et al., 2012). nPPVs are globally distributed, with higher prevalence in fattener and finishing pigs, likely due to waning maternal antibodies and increased exposure (Xiao et al., 2013; Vargas-Bermudez et al., 2023). Wild boar populations serve as important reservoirs and sources of recombination (Cadar et al., 2011).

Pathogenesis and Clinical Impact

PPV1 (Classical)

PPV1 is the primary agent of SMEDI syndrome- stillbirth, mummification, embryonic death and infertility (Mengeling, 2006). The outcome depends on gestational timing: infection before day 35 leads to embryonic death; between days 35–70, mummification; after day 70, immunocompetent fetuses may survive and be born with anti-PPV antibodies (Bachmann et al., 1975). In adults, infection is typically subclinical, though transient lymphopenia can occur (Joo et al., 1976).

Macroscopically, the most frequent outcome of PPV (Porcine Parvovirus) infection is embryonic death, followed by the absorption of body fluids and soft tissues. Visible lesions vary and may include congestion, swelling (edema) and bleeding, often accompanied by the presence of sero-sanguineous fluid within the body cavities. After fetal death, skin discoloration from internal bleeding causes a dark appearance. As the tissue progressively loses moisture, the fetus eventually becomes mummified. The placenta may appear shrunken, with a brown to grey coloration and the volume of extra fetal fluid is typically reduced.

Microscopically, infected females may show localized clusters of mononuclear cells near the endometrial lining and deeper into the lamina propria. There is also significant perivascular accumulation of plasma cells and lymphocytes in the brain, spinal cord, and eye structures such as the choroid. In sows, the uterus exhibits heavy infiltration of mononuclear cells surrounding blood vessels in both the myometrium and endometrium.

In affected fetuses, tissue damage is generally widespread, with significant cellular necrosis seen in developing organs. Bleeding is commonly found beneath the skin and within muscle tissue. The lungs, kidneys, and skeletal muscles frequently show signs of necrosis and mineral deposition.

nPPVs (PPV2–PPV8)

The pathogenic role of nPPVs is less clear. PPV2 has been associated with porcine respiratory disease complex (PRDC), with high viral loads in affected

lungs and evidence of replication in alveolar macrophages and lymphocytes (Sozzi et al., 2014). Other nPPVs (PPV4, PPV6, PPV7) have been detected in aborted fetuses and reproductive tissues, suggesting a possible link to reproductive failure, though causality is unproven.

Diagnosis

- **Serology:** ELISA is effective for detecting PPV antibodies in serum.
- **Direct Detection:** PCR and quantitative PCR are gold standards for detecting viral DNA in tissues and fluids.
- **Immunohistochemistry (IHC):** IHC localizes PPV antigens in tissues, confirming infection and revealing tissue tropism.
- **Histopathology:** Characteristic lesions in fetuses and placental tissues support diagnosis, particularly when combined with direct detection.

Control and Prevention

- **Vaccination:** Inactivated vaccines (e.g., NADL-2 strain) are widely used and reduce clinical disease but may not prevent infection or shedding, especially with emerging variants.
- **Biosecurity:** PPVs are highly stable; effective disinfection requires aldehyde-based agents or high-concentration sodium hypochlorite.
- **Surveillance:** Ongoing monitoring for both classical and novel PPVs is critical, given the potential for vaccine escape and the uncertain role of nPPVs in disease.

Conclusion

PPV1 remains a major threat to swine reproduction globally, despite widespread vaccination. The emergence and spread of nPPVs present new challenges, with their roles in disease, evolution, and coinfection dynamics still being elucidated. Improved molecular surveillance, pathogenesis studies and updated vaccines targeting diverse circulating strains are needed to address these evolving threats.

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