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ETIOLOGY, EPIDEMIOLOGY AND PATHOGENESIS IN PORCINE CIRCOVIRUS TYPE 3 INFECTION IN IN NEWBORN, WEANED, OR FATTENING PIGLETS – a literature review

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Abstract

In order to treat, combat, and prevent any disease, an essential step is to identify the causative agent. PCV-3 is a porcine circovirus that was identified and characterized relatively recently, in 2016. It has since been identified in numerous countries around the world, in pig herds, in both sick and healthy animals, as well as in wild pigs and even other animal species.

Key words: etiology, epidemiology, pathogenesis, PCV-3, pigs

Research as of 2025 confirms Porcine Circovirus 3 (PCV3) is globally widespread, present in domestic pigs and wild boar, and likely circulated since the 1990^{s}, but its exact disease-causing mechanisms are still being uncovered, though it's linked to reproductive failure, inflammation, and neurological issues, often with co-infections complicating diagnosis

Studies show it causes immune responses and lesions in organs, but gaps remain in understanding how it interacts with other pathogens and its role in subclinical infections, highlighting the need for more research.

In Romania, research on PCV3 is almost non-existent, but this virus certainly causes economic losses in pig herds. Future trends will most likely include studies on the epidemiology of the virus in the country, the clinical signs produced by infection in different categories of pigs, the lesions produced in pigs with or without clinical signs, but also aspects related to the pathogenesis of the disease.

Etiology

To date, four porcine circoviruses have been identified: PCV 1, PCV 2, PCV 3, PCV 4, all belonging to the *Circoviridae* family, genus Circovirus. They are approximately 15-25 nm in size, with a single-stranded DNA genome of approximately 1760 nucleotides, with icosahedral symmetry (Rosario K. *et al*, 2017; Saporiti V., 2021).

PCV 1 was identified in 1974 as a contaminant of cell cultures. PCV 2 was identified in 1997 and associated with a complex of multifactorial diseases in pigs (sometimes referred to as post-weaning multisystemic wasting syndrome) that mainly affects young animals and can manifest in various ways, including weight loss, respiratory disorders, enteritis, enlarged lymph nodes, jaundice (Harding J.C., 2007; Tan C.Y. *et al*, 2020; Saporiti V., 2021).

PCV 3 was identified in 2016 in the US and is considered to be involved, among other things, in dermatonephrotic syndrome in pigs (Tan C.Y. *et al*, 2020; Saporiti V., 2021).

PCV 4 was recently identified (in 2019) and associated with the same syndrome as PCV3, but also with respiratory and enteric symptoms (Tan C.Y. *et al*, 2020; Saporiti V., 2021).

The PCV 3 virus shows a variable percentage of genetic similarity with other viruses of the same genus, sometimes less than 50%. It has three ORF (open reading frame) regions that can be identified in the replicative, double-stranded form. ORF 1 is the most genetically conserved sequence and encodes the initiation of replication, ORF 2 encodes the capsid protein and is the most immunogenic sequence, ORF 3 encodes a non-structural protein (in PCV1 and PCV2 it induces host cell apoptosis) whose function is not yet fully understood in PCV3 (Saporiti V., 2021; Chen D. *et al*, 2023).

The pathogenicity of the virus is not fully understood. It has been identified in almost all types of tissues collected and tested from pigs showing a variety of clinical signs. However, the

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presence of the virus has not yet been sufficient to demonstrate its action as the sole causative agent of these manifestations; rather, PCV3 is considered to be a co-factor, alongside other causes that may be related to the individual or the environment. In addition, PCV3 has been identified in tissues collected from sick pigs, along with other pathogens (PCV2), atypical porcine pestivirus, porcine reproductive and respiratory syndrome virus (PRRSV), porcine papovavirus (PPV), classical swine fever virus (CSF), porcine kobuvirus (PKV), porcine pseudorabies virus (PRV), porcine sapelovirus (PSV), Streptococcus spp., Mycoplasma spp., Pasteurella spp., Leptospira spp.), which further complicates the establishment of causality of the observed lesions or clinical signs (Phan T.G. *et al*, 2017; Chen D. *et al*, 2017; Kedkovid R. *et al*, 2018; Kim S.C. *et al*, 2018; Zhao D. *et al*, 2018; Dal Santo A.C. *et al*, 2020; Saporiti V., 2021).

Epidemiological aspects

After the initial identification of the virus in the US, it has also been implicated in swine diseases in Thailand, Malaysia, Spain, Colombia, Namibia, and Vietnam (Nguyen V.G. *et al*, 2018; Klaumann F. *et al*, 2018; Tan C.*Y. et al,* 2020; Vargaz-Bermudez D.S. *et al*, 2021; Saporiti V., 2021; Molini U. *et al* 2024;), and Romania (Gubceac E. *et al*, 2023).

PCV3 was first identified by NGS (next generation sequencing) in an outbreak where pigs showed symptoms associated with porcine dermato-nephrotic syndrome (PDNS), increased mortality among sows, and low fertility rates. Young piglets, approximately 3-9 weeks old, suffered from various pathologies such as digestive (accompanied or not by rectal prolapse), respiratory, neurological (including congenital tremor), cardiovascular, multisystemic, reproductive, or even associated with PDNS (renal lesions, erythema, and skin necrosis) (Palinski R. *et al*, 2016; Saporiti V., 2021; Chen D. *et al* 2023).

Several retrospective studies have shown that PCV3 has been circulating in the global swine population for at least the last 20 years, before being identified in 2016 (Klaumann F. *et al*, 2018; Saporiti V., 2021).

Over time, the virus has been identified in all tissues tested: lungs, lymph nodes, kidneys, spleen, tonsils, heart, liver, central nervous system, placenta, dead and even mummified fetuses, as well as in serum (Li, X. *et al* 2018). The highest concentrations of the virus were found in lymph nodes and lungs in weaned piglets (Tan C.Y. *et al*, 2020).

PCV3 has also been identified in wild pigs, in varying proportions, between 23-42%, without necessarily being associated with any clinical manifestations, suggesting their possible role as a virus reservoir in nature (Klaumann F. *et al*, 2019; Tan C.Y. *et al*, 2020; Saporiti V., 2021). The frequency of detection was higher in adults (over 24 months) than in sub-adults (between 12 and 24 months) or young animals (less than 12 months), and some wild boars were positive throughout the study (Klaumann F. *et al*, 2019).

PCV3 has also been identified in other animal species that could play a role as natural reservoirs and possible sources of infection, such as dogs, cats, mice, cattle, various species of wild goats and roe deer, mouflon, as well as ticks and mosquitoes (Saporiti V., 2021).

The clear involvement of the virus in the pathologies with which it has been associated was only established in 2019 by Jiang and colleagues, who succeeded in experimentally infecting animals with PCV3 and obtaining clinical signs in experimentally infected animals similar to those observed in naturally infected animals and correlating with the lesions observed at necropsy (Jiang S. *et al*, 2019). Alongside this study, two others succeeded in illustrating lesions associated with experimental infection with PCV3, but not clinical signs (Mora-Diaz J. *et al*, 2020; Temeeyasen G. *et al*, 2021).

Klaumann and colleagues found that persistent PCV3 infection occurs in adult pigs, but rarely. Persistent PCV3 infection can occur in piglets between lactation and the growth/fattening period (Klaumann F. *et al*, 2019).

A study conducted in Korea (Kim, 2018) showed that PCV3 can be detected in lung and heart samples from stillborn fetuses (gestational age 60–100 days) as well as in samples from suckling piglets (3–7 days).

Klaumann and colleagues found that PCV3 infection can be intermittent (most likely latent), or it may occur only once in the lifetime of most pigs, and PCV3-specific antibodies may provide pigs with long-term protection against reinfection (Klaumann F. *et al*, 2019).

Pathogenesis

PCV3 infection causes a systemic inflammatory syndrome that affects a large number of organs and tissue types. Studies have shown that the virus replicates in the heart, liver, lungs, kidneys, intestines, CNS, and lymph nodes, but to a lesser extent than PCV2 or PCV4. The presence of the virus has been detected in samples collected from the mouth or rectum, but it was quantitatively lower than in the case of PCV2 or PCV4 (Saporiti V., 2021; Zheng J. *et al*, 2025). However, another study showed that PVC3 is excreted through nasal secretions between days 3-28 PI, sometimes with a

break between days 7/10 - 10/14 PI (Temeeyasen G. *et al*, 2021). The same study also demonstrates that the virus is excreted in faeces in the highest concentration in the first week after infection.

Furthermore, an experimental study has shown that, unlike its predecessor PCV2, PCV3 does not cause immunosuppression but actually stimulates the synthesis of antibodies and cytokines (Zheng, 2025). The same study also showed that antibody titers increase until days 21-28 post-infection, but then decrease towards day 35 PI. Last but not least, RT-PCR established that PCV3 infection causes increased cytokine concentrations in various organs, as follows:

- IFNα in the kidneys, heart, spleen, and CNS;
- IL-2 in the heart, spleen, and CNS;
- IL-6 in the heart;
- IL-10 in the spleen and kidneys (Saporiti V., 2021; Zheng J. *et al*, 2025).

Several studies have shown that PCV 3 can cause subclinical infections with persistent viremia, which means that there may be many animals in a herd that shed the virus but cannot be identified based on clinical signs for treatment or culling (Klauman F. *et al*, 2019; Saporiti V., 2021).

In the experimental study conducted by Temeeyasen and colleagues, the viral load in pigs infected with PCV3 was very high, with over 108 genomic copies per ml−1, and sustained for up to 6 weeks post-infection. The virus was also identified by qPCR in all tissues tested, where lesions indicative of multisystemic inflammation were also observed (Temeeyasen G. *et al*, 2021).

The same study identifies an early and sustained synthesis of IgG-type anti-PCV3 antibodies in experimentally infected pigs. The authors suggest that IgM titers may appear later in the course of the disease, playing a role in viral neutralization processes, following a mechanism similar to that observed with PCV2. They also illustrate that viremia levels may be associated with IgG levels and that pigs with high viremia titers have high IgG titers and vice versa (Temeeyasen G. *et al*, 2021). However, the study conducted by Mora-Diaz and colleagues identified viremia at 28 dPCV and a high IgM antibody titer at 7-14 dPCV (Mora-Diaz J. *et al*, 2020).

In the study conducted by Jiang, 2019, the highest amount of viral DNA was correlated with a low titer of antibodies against PCV3 in serum. The authors suggest that the simultaneous presence of PCV3 and its antibodies suggests that the virus may have evolved a strategy for maintaining itself in nature, as has been observed for the encephalomyocarditis virus and foot-and-mouth disease virus (Billinis C. *et al*, 1999; Jiang S. *et al*, 2019).

Temeeyasen mentions that multisystemic inflammatory lesions characterized by perivasculitis of the kidney, spleen, liver, heart, and serous tunic of the small intestine were observed in association with PCV3 replication within the lesions, as confirmed by in situ hybridization, although the lesions were less intense than those observed in natural infection (Temeeyasen G *et al*, 2021). In some of the tissues detected positively by ISH, the virus was not detected in inflammatory foci, which may indicate viral elimination by cytotoxic mechanisms. Other authors (Arruda B. *et al*, 2019) have described viral replication on endothelial cells, which may also be associated with viral detection by ISH in non-inflammatory foci.

The results demonstrated that PCV3 did not have a significant regulatory effect on all T lymphocyte populations under the study conditions. The detection of prolonged viremia and detection in multiple tissues at 42 dPCV suggested that the action of T lymphocytes on the virus might be reduced (Temeeyasen G. *et al*, 2021).

PCV3 infection causes multisystemic inflammatory lesions and lymphocytic dysplasia accompanied by necrosis, which can cause immunosuppression in piglets and increase their susceptibility to infections with other primary and secondary pathogens that can affect growth performance, but which can also increase morbidity and mortality (Jiang, 2019).

The pathogenic mechanisms of PCV3 are not yet fully understood, and the ideas formulated by many authors are based at least in part on the mechanisms observed in other porcine circoviruses. For example, the apoptotic activity of the ORF3 protein has been described both in vitro and in vivo for PCV1 and PCV2 (Liu X. *et al*, 2016; Karuppannan A.K. *et al*, 2011), while its function in PCV3 is still unknown. The pathological differences observed between circoviruses could also derive from possible structural differences, but also from the role of the non-structural ORF3 protein (Temeeyasen G. *et al*, 2021).

It is also possible that, in terms of the types of tissues in which viral replication occurs, PCV3 does not show tropism for the gastrointestinal tract. The intermittent presence of the virus in feces could be caused by viral transudate as a result of viremia in infected pigs following repeated exposure through horizontal transmission (Temeeyasn G. *et al*, 2021).

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