**Porcine Reproductive and Respiratory Syndrome (PRRS): A Comprehensive Review of Recent Developments in Diagnosis and Control Strategies**

**ABSTRACT:**

Porcine reproductive and respiratory syndrome (PRRS) is one of the most important diseases of pigs. It is caused by the PRRS virus (PRRSV) of the genus *Betaarterivirus,* belonging to the family *Arteriviridae* and order *Nidovirales*. The virus is transmitted through direct contact, such as the oronasal and genital routes, and also indirectly via contact with fomites contaminated with the infectious virus. The disease is characterized by respiratory symptoms in pigs of all age groups and reproductive failure in gestating gilts and sows. The reproductive disease is characterized by abortion, fetal mummification, stillbirth, and birth of weak piglets. Whereas the respiratory syndrome is characterized by fever, inappetence, lethargy, and dyspnea etc. The infection leads to significant economic losses in pig production in pig producing countries. Early diagnosis of the disease along with adopting proper biosecurity measures remains critical in the prevention and control of the disease. This review aims to provide a comprehensive picture of recent developments in the diagnosis of Porcine Reproductive and Respiratory Syndrome and its control strategies. We will explore innovations in PRRSV detection methods, including advanced molecular testing techniques. We will also look at the latest approaches in disease prevention and control, including vaccination strategies, biosecurity programs, and farm management, with a focus on integrated solutions aimed at mitigating the devastating impact of PRRS on pig production.

*Keywords: Porcine reproductive and respiratory syndrome, PRRS, diagnosis, control, infectious disease, pig*

1. **INTRODUCTION:**

Porcine reproductive and respiratory syndrome (PRRS) was first reported to cause a disease outbreak in pigs during the late 1980s in the United States of America (USA); later the disease invaded Canada. The disease was characterized by reproductive ailments (mummification, stillbirth, and abortion), respiratory distress, decreased weight gain, and increased mortality [1]. Initially the disease was named ‘mystery swine disease (MSD)’, ‘swine infertility and respiratory syndrome’, and ‘blue-ear pig disease’ as the exact etiology was unknown [2]. During the 1990s the first European outbreak of the disease was reported from Germany [3], from where the disease spread rapidly throughout Europe. In the year 1991, studies showed the virus was isolated in porcine alveolar macrophages and determined that it is caused by an unknown RNA virus [4]. The viruses used in these studies were isolated at the central veterinary institute at Lelystad in the Netherlands [5]. The isolated strain was named after the institute as the Lelystad strain. The present name of the disease, ‘porcine reproductive and respiratory syndrome (PRRS),’ was proposed by the European scholars in the same year. Later, the virus strain VR-2332 was isolated from tissue homogenates of diseased pigs of the Minnesota region in continuous cell line CL-2621 [6]. Though the disease was first reported in the late 1980s, retrospective serological studies have shown that antibodies against PRRSV were present in sera collected during 1979 from Canadian herds [7].

PRRS is caused by the PRRS virus (PRRSV) of the genus *Betaarterivirus* under the family *Arteriviridae* and order *Nidovirales* [8,9]. Morphologically the virus is spherical and enveloped with a core measuring 25-35 nm and a diameter of 45-80 nm [10,11]. PRRSV has a buoyant density of 1.19 in CsCl and 1.14 in sucrose [12]. The genome of the virus is positive-sense single-stranded RNA in nature [13]. There are at least 10 open reading frames (ORFs) in a genome size of approximately 15 kb [14]. *Arteriviridae* has the smallest genome size under order *Nidovirales*. There are two genotypes of the virus: European type (PRRSV-1) and North American type (PRRSV-2). The prototypes of the European and North American genotypes are the Lelystad and VR-2332 viruses, respectively [15,20]. There are significant genetic differences between the European and American genotypes of PRRSV, with respect to only 55-70% nucleotide identity [16,17]. Therefore, it is believed that the virus has divergent evolution in two continents, originating from a distant common ancestor [17]. In Asia the disease was first reported in Japan in the year 1988, but in retrospective studies it has been seen that the antibody was present since 1985 [18].

The disease is endemic in most of the pig-producing countries except Australia, New Zealand, five European countries (Norway, Sweden, Finland, Switzerland, and Hungary), three South American countries (Argentina, Brazil, and Chile), and Cuba [19,20]. PRRSV is transmitted via various body fluids such as feces, urine, saliva, milk, and semen [21]. In addition, transmission can also occur directly from the sick pigs when they come in contact with a susceptible population [22] and via aerosol [23]. PRRSV infects pigs belonging to all age groups, manifested by respiratory issues and reproductive disorder mainly in gestating gilts and sows [24]. PRRSV infection also leads to sickness with a delayed immune response resulting in extended viremia, which facilitates virus transmission [25]. Rarely PRRSV infection persists longer than 200 days [26]. PRRS causes significant economic losses to the swine industry. In the US only, the disease is estimated to cause losses of $600 million annually [27].

1. **CLINICAL SIGNS AND SYMPTOMS:**

The severity and clinical signs of PRRS depend on the production stage at which the pig is infected, the strain of virus involved, the immune status of the animal, and co-infections. In some cases, the disease is subclinical without any clinical signs. Pigs of all age groups exhibit clinical signs of respiratory distress. The respiratory form of the disease is characterized by a significant increase in body temperature of pigs up to 41-42°C, anorexia, lethargy, rough hair coat, hyperaemia, dyspnea, coughing, sneezing, and bluish discoloration of the ear, snout, and vulva [20]. The course of the disease is 1-3 weeks; death of pigs usually occurs within 5-7 days, and then the mortality is gradually reduced. The morbidity is usually 50-100%, whereas mortality ranges between 20-100%. Mortality rate is usually high in younger pigs and decreases with age. PRRSV can easily replicate and cause pathology in both the endometrium and placenta, resulting in transplacental infection of the fetus and causing reproductive disease [28,29,30]. Reproductive abnormalities associated with the virus are most likely due to the damage it causes to the placenta and endometrium [31]. Pregnant sows often abort but seldom die from the disease. There is infertility, fetal mummification, and stillbirths. Abortions induced by PRRSV may persist within a herd for 10 weeks to 6 months [32]. The respiratory syndrome is characterized by sneezing, coughing, dyspnea, ocular secretion, conjunctivitis, etc. In some cases, constipation or diarrhea, as well as neural signs, are seen. In the long term the diseased pig becomes pale and emaciated with rough hair coats [33]. On the farm level the disease is characterized by increased piglet mortality, a decrease in semen quality of boars, and an increase in various reproductive disorders such as abortion, mummification, stillbirth, weak birth, etc. [34].

At necropsy there is severe pulmonary edema and consolidation. Edema of lymph nodes is also observed. In some case there is pulmonary interstitial hyperplasia and congestion, bleeding in the larynx and trachea and mucosal congestion, hydropsia, and ulcers in the gastrointestinal system [33]. Microscopically, there is moderate to severe multifocal interstitial pneumonia characterized by a mixed population of mononuclear cells infiltrating the alveolar septum, hypertrophy and hyperplasia of pneumocytes, and an apparent mixed necrotic and inflammatory alveolar exudate.

**3. DIAGNOSIS:**

**3.1 Virus isolation:**

The samples for isolation of the virus are collected from recently infected animals. After collection, the samples are quickly sent to the laboratory under refrigeration. During necropsy, samples are usually collected from different organs such as lungs, spleens, lymph nodes, serum, and plasma. Samples for detection of the virus include whole blood, serum, buffy coat, lung, lymph nodes, spleen, and tonsil of affected animals [35]. Isolation of the virus from samples collected from infected animals confirms the disease [36]. PRRS virus grows well on primary porcine alveolar macrophages (PAM), continuous cell line CL 2621, and MA 104 [37]. Cytopathic effect (CPE) characterized by clumping, rounding, and lysis of the cell in PAM within 1-4 days [38].

**3.2 Serological tests:**

Four serological tests are commonly used for diagnosis of PRRS: IPMA, IFA, ELISA and SNT [39]. IPMA was the first serological test available for diagnosis of the disease and has been used extensively in Europe [5]. As a variant to IPMA, the indirect fluorescent antibody (IFA) test was developed [40] and has been used extensively in North America. Both IPMA and IFA have high sensitivity and specificity and can detect PRRSV antibodies seven to fourteen days after infection. An enzyme-linked immunosorbent assay (ELISA) was developed for the detection of PRRSV antibody using infected alveolar macrophage cell culture supernatant as antigen [41]. The ELISA test is very sensitive in detecting antibodies and does so as early as 9 days after infection; however, the use of ELISA is limited since antibodies are thought to persist for only five to six months [42]. Serum neutralization test (SNT) may be employed for antibody detection against the virus, but the sensitivity of SNT is less than that of IPMA, IFA, or ELISA [43].

##### **3.3 Molecular tests:**

Various molecular tests for detection of viral RNA are available, such as polymerase chain reaction (PCR) [44], quantitative real-time PCR (qPCR) [45], digital PCR (dPCR) [46], loop-mediated isothermal amplification (LAMP) [47], recombinase polymerase amplification (RPA) [48], and metagenomic next-generation sequencing (mNGS) [49], etc.

**3.4 Differential diagnosis:**

The reproductive signs associated with PRRS need to be carefully differentiated from those caused by other infectious diseases with similar clinical presentations. These include leptospirosis, porcine parvovirus infection, porcine enterovirus infection, haemagglutinating encephalomyelitis, *Toxoplasma gondii*, Aujeszky’s disease (pseudorabies), African swine fever (ASF), and classical swine fever (CSF) [50]. Accurate diagnosis is crucial, as these diseases vary significantly in epidemiology, control measures, and public health implications. In addition to laboratory testing, the diagnosis of the disease shall be based on the clinical picture and postmortem examinations.

## For the respiratory and post-weaning form of PRRS, differential diagnosis should consider diseases such as myocarditis, swine influenza, enzootic pneumonia (primarily caused by *Mycoplasma hyopneumoniae*), proliferative and necrotizing pneumonia, swine respiratory coronavirus, porcine circovirus-associated disease, Nipah virus infection, and infections caused by *Haemophilus parasuis* [51]. A combination of clinical evaluation, serology, PCR, and histopathology is often required to establish a definitive diagnosis and distinguish PRRS from these other respiratory pathogens.

1. **PREVENTION AND CONTROL:**

The control and eradication of PRRS primarily depend on early detection of the disease, rapid laboratory confirmation, surveillance, quarantine, biosecurity measures, stamping out of affected or in-contact animals, vaccination, etc. Continuous surveillance, farmer education, and coordination between veterinary authorities and stakeholders are also vital to ensure effective control and eradication of the disease in pig populations. Establishment of age-separated pig farms is recommended to control the disease [52].

**4.1 Surveillance:**

Routine serological testing is crucial to keep the herd free from the disease [53]. Clinical examination of the pigs, along with collection of samples from a statistically representative number of animals, and review of production records for signs of reproductive failure, such as abortions, stillbirths, weak-born piglets, and elevated pre-weaning mortality, is important for prevention and control of the disease. In areas with feral pig populations, sero-surveillance becomes especially important for detecting asymptomatic carriers and tracking transmission between wild and domestic pigs.

**4.2 Quarantine and movement controls:**

All farms with confirmed or suspected PRRS infections should be immediately placed under strict quarantine. In village or free-range systems, pigs should be confined within secure enclosures to prevent further spread. The movement of pigs into or out of infected farms or localities must be prohibited.

**4.3 Biosecurity:**

Strict biosecurity protocols such as procuring new stocks from disease-free herds, limiting visitors, installing perimeter fencing, and proper disposal of carcasses are important [54]. Vehicles used for moving infected or exposed pigs, feed, farm machinery, etc., must undergo thorough cleaning and disinfection before being reused. The utensils and equipment should be thoroughly cleaned after each use, and sharing of these items should be avoided among farms. Proper disposal of carcasses, waste management, and disinfection of farm premises are important to limit infection to susceptible populations. Farm workers should wear dedicated work clothing and avoid contact with other pig populations to prevent indirect disease transmission. These biosecurity measures are essential to break the transmission cycle and contain the disease.

**4.4 Zoning:**

If PRRS is endemic in only certain regions of a country, then it is essential to establish clearly defined infected and disease-free zones. Strict regulations should then be enforced to control the movement of pigs, pig products, and related goods between these zones. This zoning approach helps in containing the disease within affected areas, protects uninfected regions, and facilitates more targeted surveillance, control, and eventual eradication efforts.

**4.5 Stamping out:**

This strategy is most appropriate during the early stages of an outbreak, particularly when the affected area is small and the number of pigs to be culled is limited. However, employing this strategy depends on the concerned country. Stumping out strategy has a huge economic impact. After stumping out of infected and in-contact pigs, the carcass should be properly disposed of following proper guidelines.

**4.6 Cleaning and disinfection:**

PRRS virus (PRRSV) is very sensitive to common disinfectants, routine cleaning and disinfection is generally sufficient for decontaminating farms, vehicles, and equipment. It has been seen that lipid solvents such as detergents, quaternary ammonium compounds, chlorine-based disinfectants, as well as phenolic and organic acid formulations, are highly effective in inactivating PRRSV. However, before disinfection cleaning to remove organic matter should always take place. The presence of dirt or manure can reduce the efficacy of chemical agents.

**4.7 Vaccination:**

While vaccination cannot completely prevent PRRSV infection, it remains one of the most effective tools for managing and mitigating the impact of the disease [55]. For a vaccine to be effective, it must closely match the circulating antigenic strain. Field experience has shown that vaccination with a homologous strain (one closely related to the field virus) provides significantly better protection than vaccination with a heterologous strain. Both, modified-live virus (MLV) and inactivated (killed) vaccines are licensed for both the respiratory and reproductive forms of PRRS. These vaccines are typically administered to sows and gilts three to six weeks before breeding, and to piglets as early as three weeks of age. The efficacy of the inactivated vaccine is generally lower than that of MLV vaccines. A common strategy includes vaccinating seronegative replacement breeding stock 60 to 90 days prior to introduction into the herd, allowing sufficient time for the development of protective immunity. Diverse genetic variety of the virus makes the vaccine often ineffective [50]. Development of DNA vaccines and recombinant vector vaccines are recommended for better efficiency [56].

**4.8 Sentinel and restocking:**

To prevent re-infection with PRRSV, restocking of farms should only be carried out after a minimum of 14 days following thorough cleaning and disinfection. This allows sufficient time for any residual virus to be inactivated. Once the farm is repopulated, serological testing should be conducted on the new animals at approximately six weeks and again at two months post-introduction to monitor for any signs of viral circulation or re-emergence. However poor biosecurity measures pose significant risk of new outbreak of the disease. Therefore, restocking shall be done with animal from reliable sources, and shall be accompanied with strict biosecurity measures.

**4.9 Public awareness:**

Community engagement is vital for the success of any control program. Educating pig farmers about the biosecurity measures, timely reporting, dangers of swill feeding are important for control of the disease.

1. **CONCLUSION AND FUTURE RESEARCH:**

PRRS is one of the most important disease of pigs causing significant economic losses to the farmers. The disease is characterized by respiratory syndromes in pigs of all ages and reproductive syndrome in sows. Different diagnostic tests are available for diagnosis of the disease such as virus isolation, serological tests and molecular tests for detection of viral nucleic acids. The control of the disease is focused on continuous surveillance, maintaining proper biosecurity, stumping out and vaccination etc. Both inactivated and modified live virus (MLV) vaccines are available for the disease. The inactivated vaccines are generally less effective than the MLV vaccines but the MLV vaccination may lead to shedding of the vaccine virus by the animal to the environment.

Further study into immune evasion mechanisms of the virus, host genetic resistance and mechanism of persistence of the virus in the host body is required for formulating effective control measures. Molecular epidemiology may be undertaken to track evolution, recombination and emergence of virulent variants. Development of rapid and sensitive diagnostic tools, surveillance system, new generation vaccines, effective against diverse strains of PRRSV, also DIVA vaccines for distinguishing the vaccinated and infected animal is important for combating the disease.

**Disclaimer (Artificial intelligence**)

We hereby declare that no generative AI technologies such as Large Language Models been used during the writing or editing of the manuscript.

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