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Review

Porcine reproductive and respiratory syndrome virus infection in the boar: a review

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Abstract

Porcine reproductive and respiratory syndrome (PRRS) is caused by PRRS virus, which, like other members of the *Arterividae* family, has the ability to infect macrophages and to persist in tissues for at least several months after the acute stage of infection subsides. As a consequence, PRRS has a complex epidemiologic profile and has been especially difficult to control under the usual conditions of commercial swine production. Although vaccines are commonly used, vaccination is only one of several approaches to be considered in designing a control strategy. At least equally important are procedures developed on the basis of a thorough understanding of the epidemiology of the disease. The objective of this review is to summarize current knowledge in relation to PRRS virus (PRRSV) infection in the boar. The information available related to this topic will be summarized and discussed, and the implications for the control of the condition highlighted. The main emphasis will be on questions about the pathogenesis of infection, including duration of viremia and the origin of PRRSV found in semen; the clinical signs associated with the disease, paying special attention to the effects on seminal quality; the epidemiology of the condition, with special emphasis on the duration of PRRSV shedding in semen and the implications that this may have on venereal transmission, as well as the role that other potential routes of shedding may have on the dissemination of PRRSV.

Keywords: PRRS; Boars; Reproductive tract

1. Introduction

Porcine reproductive and respiratory syndrome (PRRS) is one of the most economically devastating diseases affecting swine industry worldwide. The causative virus (PRRS virus, PRRSV) was first isolated in 1990 in The Netherlands [1] and shortly after in the USA [2].

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Primarily, on the basis of similar morphology, genomic organization, replication and transcription strategy, and protein composition, PRRSV is classified, with equine arteritis virus (EAV), lactate-dehydrogenase elevating virus (LDV) and simian hemorrhagic fever virus (SHFV), in the family *Arteriviridae* [3]. Members of this family also share the ability to replicate in macrophages and induce persistent infections in their natural host.

Although, in general, PRRS is clinically similar in North America and Europe, the respective strains differ in virulence [4], and in antigenic [5,6] and genetic [7] properties. These differences have led to the classification of strains into two subgroups: subgroup A, comprising most North American strains; and subgroup B, comprising most European strains [7].

The role of PRRSV in reproductive failure in the sow has been firmly established. Experimental infections have mirrored the clinical signs observed in natural outbreaks, including abortions, premature farrowings, mummified pigs, stillborn pigs, and elevated preweaning mortality [8–12]. Infection has also been associated with reduced conception rates, elevations in the percentage of regular, as well as irregular returns to estrus and sporadic early abortions [13]. Transplacental infection commonly follows exposure of gilts and sows during gestation [11,14–16], especially when initial exposure is late in gestation [11]. As a consequence, maternal infection can give rise to the birth of congenitally infected pigs and to an elevated number of mummified fetuses, the latter as a result of a high mortality rate among infected fetuses [8,9,11,14–18].

In the boar, clinical manifestations include anorexia, lethargy, and loss of libido [19,20], and, on some occasions, alterations in semen quality, including a decrease in sperm motility, an increase in morphoanomalies and cytoplasmic droplets, and a decrease in the percentage of spermatozoa with a normal acrosome. These alterations have been described in both natural and experimental infections [19,21,22]. However, the consequences of infection in relation to semen are still controversial since some researchers have found that the quality of semen remains within normal limits after infection with PRRSV [23,24].

The rapid spread and economic impact of PRRS have made it a frequent topic of research, especially in regard to its control. As with many other infectious diseases, the most effective means for control often depends on the use of vaccines as well as the implementation of improved management practices. Regarding the first option, there are currently several commercially available vaccines. These include live-modified-virus as well as inactivated-virus vaccines. However, the nature of the pig's immune response to PRRSV makes the development of an unquestionably safe as well as highly effective vaccine a formidable challenge. Consequently, in many affected herds, the development of strategies for control and perhaps eventual eradication of PRRS depends on a thorough knowledge of the epidemiology of the disease.

We know today that one of the main characteristics of PRRSV is its high transmissibility, which almost certainly contributed markedly to its quick spread around the world. Pigs are susceptible to infection by a number of routes, including oral, intranasal, intramuscular, intraperitoneal, and vaginal. They can also shed the virus in a number of ways, including saliva [25], nasal secretions [26], urine [26], feces [27], mammary gland secretions [28], and semen [24,29,30]. Moreover, the interval of shedding can also be extensive [25,29].

An important epidemiological feature of PRRSV, as well as other members of the *Arteriviridae* family, is their ability to induce persistent infections [31]. Notably, PRRSV