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Review article

Pathogenesis and control of the Chinese highly pathogenic porcine reproductive and respiratory syndrome virus

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ABSTRACT

Porcine reproductive and respiratory syndrome virus (PRRSV) has remained a major threat to the worldwide swine industry ever since its first discovery in the early 1990s. Under the selective pressures in the field, this positive-stranded RNA virus undergoes rapid genetic evolution that eventually leads to emergence in 2006 of the devastating Chinese highly pathogenic PRRSV (HP-PRRSV). The atypical nature of HP-PRRSV has caused colossal economic losses to the swine producers in China and the surrounding countries. In this review, we summarize the recent advances in our understanding of the pathogenesis, evolution and ongoing field practices on the control of this troubling virus in China.

Contents

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1. Introduction

Porcine reproductive and respiratory syndrome (PRRS), a disease that is typically manifested by reproductive failures (e.g. late-term abortion, stillbirth and mummification) in sows and respiratory distress (e.g. interstitial pneumonia) in growing pigs, remains a major threat to the worldwide swine industry; it emerged almost simultaneously in North America and Europe in the late 1980s and then spread quickly to other parts of the world within a few years. The etiological agent of PRRS was identified to be porcine reproductive and respiratory syndrome virus (PRRSV) in 1991 in the Netherlands and 1992 in the United States (Collins et al., 1992; Wensvoort et al., 1991); it is a positive-stranded RNA virus that is classified within the family *Arteriviridae* of the order *Nidovirales* (Kuhn et al., 2016). Surprisingly, the PRRSV isolates apparently had undergone divergent evolution on the two continents; they display remarkable genetic differences (about 40%) at the nucleotide level, despite some shared molecular features (e.g., similar genomic organization, virion structure, and replication strategies, etc.) (Allende et al., 1999; Nelsen et al., 1999). Accordingly, PRRSV is classified into two genotypes, namely the European PRRSV (type 1) and North American PRRSV (type 2). The Lelystad virus (LV) strain represents the European type 1 whereas the strain VR-2332 serves as the representative of the North American type 2.

The error-prone nature of PRRSV RNA polymerase and its frequent recombination as well as the selective pressures in the field have spurred the rapid viral evolution (Murtaugh et al., 2010). Exemplified by type 2 PRRSV strains, the continuous evolution has spawned many virulent strains that account for several major epidemics of PRRS in the past 30 years (Shi et al., 2010a; 2010b). Following the original outbreak in the late 1980s, an “acute PRRS” broke out in 1996 in Iowa and other states of the United States and was associated with an increased abortion rate and sow mortality (Bush et al., 1999; Halbur and Bush, 1997). The viral isolates (e.g. ATP, JA-142, etc.) were found heterogeneous and genetically distinct from the then attenuated live vaccine virus, belonging to the now classified lineage 8 and 9 of type 2 PRRSV (Shi et al., 2010a). Five years later, the highly virulent PRRSV MN184 strains emerged suddenly in Minnesota, USA. The most prominent feature is the three-discontinuous deletions with a total size of 131 amino acids in the nsp2-coding region, leading to the shortest PRRSV genome at the time (Han et al., 2006).

Emergence of the Chinese highly pathogenic PRRSV (HP-PRRSV) represents another milestone along the course of PRRSV evolution. Different from the previous outbreaks, this time the devastating virus descended on the continent Asia (Tian et al., 2007). The epidemic caused death of millions of pigs in the initial bout of outbreak in 2006 (Zhou and Yang, 2010) and has since been plaguing the Chinese swine industry. The most recent evolving episode is the appearance of the virulent PRRSV NADC30 strains in the United states (Brockmeier et al., 2012), and the NADC30-like strains in China (Li et al., 2016a; Zhang et al., 2016a; Zhao et al., 2015a; Zhou et al., 2015). Strikingly, they all carry the genetic marker of PRRSV MN184 strains, namely the exact discontinuous deletions in the nsp2-coding region. Moreover, the NADC30-like strains are now starting to recombine with the Chinese HP-PRRSV in the field (Li et al., 2016b; Zhao et al., 2015a), adding a further layer of extreme complexity to the PRRS control. In this review, we focus on the recent advances in the pathogenesis and evolution of HP-PRRSV as well as the current practices on the disease control.

2. Epidemiological and clinical characteristics of HP-PRSV infection

2.1. Emergence of HP-PRRSV in China

On the Chinese mainland, the year 1995 saw the first case report on PRRS that took place in an intensive pig farm in North China (Guo et al., 1996). The isolated strain CH-1a is of North American lineage but shows a distinct relationship with VR-2332. In contrast, a later PRRSV strain named BJ-4 isolated from an aborted fetus in Beijing around 1996 was rather close to the North American prototype (Yang et al., 1997). In the subsequent years, PRRSV spread from the North to other parts of China and became highly prevalent in the majority of pig farms. Evolution spurred emergence of novel variants. Around 2004, the first natural deletion variant was characterized in China (Gao et al., 2004). The isolated strains HB-1(sh)/2002 and HB-2(sh)/2002 originated from a swine farm undergoing an acute PRRS outbreak (Gao et al., 2004). In particular, HB-2(sh)/2002 is evolutionarily close to CH-1a but carries a 12-aa deletion in the nsp2-coding region in combination with 1-aa deletion in the gene coding for glycoprotein GP3 (Gao et al., 2004). During the 10-year period from 1995 to 2005, there were numerous case reports on emergence of PRRSV variants in the field, and consequently, PRRSV remained as a major pathogen of swine.

The emergence of HP-PRRSV was sudden and a mystery. Beginning in the summer of 2006, a swine “high fever disease (SHFD)”of unknown etiology stroke the swine farms in Jiangxi province; it affected pigs of all ages and the infection was highly contagious. Very soon, the disease spread to most of the southeast and central provinces. As of Spring 2007, the epidemic had preceded to the swine farms in Southwest and North China. Within less than one year, the disease resulted in the death of millions of pigs and devastated many swine farms in China. Consequently, the overall pork production dropped dramatically and the market saw a huge spike of the pork prices.

In June 2007, the etiological agent was identified to be a novel variant of type 2 PRRSV (Tian et al., 2007). When tested in piglets,