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Roongroje Thanawongnuwech

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Roongroje Thanawongnuwech
Emerging and Re-emerging Infectious Disease in Animals, Research Unit (CUEIDAs), Faculty of Veterinary Science, Chulalongkorn University, Bangkok 10330, Thailand

Porcine reproductive and respiratory syndrome virus (PRRSV) has been demonstrated serologically as early as 1979 in Canada, in the 1980's in other regions of North America, Europe and east Asia, in China in the mid-1990's and in Thailand in 1989 (Thanawongnuwech et al., 2004). PRRSV belongs to the family Arteriviridae, genus Arterivirus, generally divided into two major genotypes, European (EU or Type I) and North America (NA or Type II) and continues to be an economically significant swine disease particularly called porcine respiratory disease complex (PRDC) characterized by slow growth, decreased feed efficiency, anorexia, fever, cough and dyspnea in weaning to finishing pigs. Reproductive failures in sows and temporary infertility in boars are also prominent in naïve breeders. PRRSV antigenic and genetic heterogeneities as well as quasispecies evolution are documented (Goldberg et al., 2003; Schommer and Kleiboeker, 2006). In addition, co-existence of the two genotypes or more than one strains become potentially problematic since cross-protection among strains does not exist (Thanawongnuwech et al., 2004). Evidently, recombination between the Chinese modified live virus (MLV) vaccine and a local strain was demonstrated in the field when the MLV vaccine was heavily implemented during the PRRSV epidemic in China (Li et al., 2009). Additional information from the Chulalongkorn University-Veterinary Diagnostic Laboratory (CU-VDL) indicates increasing evidence of mixed infections among PRRSV strains within the same herd, causing difficulty for the control strategies either using vaccines or management strategies.

Recently, the NA PRRSV with a nucleotide deletion in the nsp2 coding region have been reported in USA, China, Japan, Denmark and Vietnam (Gao et al., 2004; Han et al., 2006; Li et al., 2007; Feng et al., 2008; Yoshii et al., 2008). Following the outbreaks of swine high fever (SHF) syndrome caused by highly pathogenic (HP)-PRRSV in China, many genetic variants of this virus have been isolated and recent data suggests that those variants were derived from the CH-1a strain in the south of China (An et al., 2010). A novel nucleotide deletion in nsp2 found in those Chinese isolates initially linked to the virulence of the virus may possibly attribute to a combination of HP-PRRS and other pathogens such as classical swine fever virus (CSFV), porcine circovirus (PCV-2) and probably other additional agents. The HP-PRRSV containing two discontinuous sequence deletions in the nonstructural protein (NSP) 2 gene, has initially occurred in 2007 and continued to be a problem in China, Vietnam (Wu et al., 2009) and the Philippines. Unavoidably, the HP-PRRSV eventually found in the back-yard pigs in NongKai province, Thailand in August 2010 caused high mortality rate in all age groups (Unpublished data). Evidently, PCV2 and classical swine fever virus were also demonstrated in those submitted tissues by PCR (CU-VDL). Based on epidemiologic evidence, the HP-PRRSV might gain its entry from the pig trade at the border area. The department of livestock development (DLD), Thailand has implemented stamping out strategy and active surveillance in those back-yard pigs and other pigs in the nearby provinces in order to stop the spreading out of the virus to other areas.

Although deletions in the NSP2 gene has previously been related to increased virulence of this particular HP-PRRSV strain, it has been further shown no virulence relation (Zhou et al., 2009). It should be noted that only an in vivo study is able to differentiate PRRSV virulence among strains. Indeed, genetic, antigenic and pathogenic variability existing among PRRSV strains have drawn great attention for diagnostics, control and prevention of this disease (Kim et al., 2007).

The HP-PRRSV affecting all stages of production manifests hyperthermia and severe respiratory depression, anorexia, red discoloration of ears and body. Piglets manifest cough and diarrhea, leg edema and paralysis. Pregnant sows manifest abortion and birth to weak born and stillborn piglets. Morbidity in nursery and growing pigs as well as pregnant animals may reach from 50 to 100% and

mortality from 20 to 90%. Surveillance and monitoring should routinely conduct to further assessing the situation and determine the magnitude of the problem in the affected countries.

Several risk factors predisposing PRRSV infection include variation in biosecurity levels, animals and animal movement, exposure from PRRSV-infected or vaccinated neighboring herds, infected semen, herd size, pig density and herd density in the areas (Mortensen et al., 2002; Christopher-Hennings et al., 2008). Spreading of PRRSV among farms normally causes by introducing infected pigs or semen, while spreading within farms is mainly due to the intra-herd movement of the carriers or infected pigs or when mixing pigs. PRRSV transmission occurs by various means including direct contact, vertical transmission from infected sows, infected semen and mechanical infection via infected needles or carrier insects, as well as aerosol transmission in some occasions (Pitkin et al., 2009[a]; Pitkin et al., 2009[b]). Virus-contaminated saliva, possibly due to prolonged recovery of virus from tonsils, also plays a major role in PRRSV transmission when mixing pigs at each production stage (Wills et al., 1997).

Interaction among pathogens has been demonstrated particularly when PRRSV acts as a major pathogen in the pathogenesis of PRDC and other immunomodulating effects (Thanawongnuwech et al., 2001). PRRSV predisposes pigs to *Streptococcus suis* and other opportunistic bacterial infections leading to secondary bacterial infection possibly caused by ineffective pulmonary clearance (Thanawongnuwech et al., 2000[a]). The severity of PRDC with the presence of PRRSV and *Mycoplasma hyopneumoniae* potentiates PRRSV-induced pneumonia or *M. hyopneumoniae*-induced pneumonia is probably due to the induction of proinflammatory cytokines by *M. hyopneumoniae* (Thanawongnuwech et al., 2004[b]). In order to improve PRRS clinical pictures decreasing other concurrent infections in the farm and create stress free environment must implement in the affected farms.

Interestingly, pigs infected with PRRSV or having PRRSV viremia during swine influenza or classical swine fever (CSF) vaccination showed decreased vaccine efficacy or vaccination failure by PRRSV (Suradhat et al., 2006; Kitikoon et al., 2009). Consistently, recent re-emerging of CSF outbreaks in Thailand mainly cause by CSF vaccine failure since CSF vaccination programs are usually scheduled after weaning when weanling pigs co-mingled and transmitted PRRSV to one another. Concurrent infections following PRRS outbreaks including Salmonellosis, Streptococcosis, Glasser's disease, greasy pig disease, Colibacillosis and Eperythrozoonosis are frequently observed particularly in the continuous flow or farrow-to-finish system. Controlling secondary infection would reduce the mortality rate due to septicemia or other complications.

Intervention strategies to prevent its spreading are the keys to control PRRSV. Continuous flow system commonly results in early infection after weaning and creates the virus source circulating in the system. Based on phylogenetic analyses, most frequent sources of infection in PRRS-positive farms is from the introduction of replacement animals carrying a new PRRSV strain rather than mutation of the already existing viruses (Pesente et al., 2006). Evidently, animal movement increases due to the demanding of the pig farmers during the high pork price. Several management techniques implemented to control the spreading of PRRSV consist of reducing both vertical and horizontal transmissions including sow herd stabilization, all in/all out, medicated early weaning, segregated early weaning, and nursery depopulation as well as vaccination with incomplete success. The effective means of disease prevention and control are rigorous bio-security along with sow herd stabilization by herd closure and closed herd. Eradication could be the ultimate tool for PRRSV control. Since current PRRS control strategies are not predictably successful, PRRS-associated losses will continue to be seen worldwide.

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