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Microbial ecology of swine farms and PRRS vaccine vaccination strategies

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ABSTRACT

The present study investigated the microbial ecology and vaccination strategies against porcine reproductive and respiratory syndrome (PRRS) in field condition. Four representative farms with a history of PRRS were included in this study. Over the almost 3-year period, the average detection rate was 68.9%, making PRRSV the first most frequently detected virus, followed by porcine circovirus type 2 (PCV2) (64.2%), pseudorabies virus (PRV) (11.03%) and classical swine fever virus (CSFV) (4.41%). *Streptococcus suis* (77.92%), *Haemophilus parasuis* (51.25%) and *Escherichia coli* (52.39%), *Pasteurella multocida* (26.77%) were isolated most frequently in association with PRRSV. Under the present microbial ecology, production performances of sows their offspring after mass vaccination with a PRRS attenuated vaccine were evaluated. In addition, large scale PRRS vaccines usage and efficacy were further performed. The results indicated that mass vaccination following our immunization program can improve health status and production performances of both sows (2 ml/i.m. booster after 4 weeks, and then immunized quarterly) and their offsprings (1 ml/i.m. on 14–18 days of age).

1. Introduction

Porcine reproductive and respiratory syndrome (PRRS) is currently considered to be the most significant and economically important infectious disease to afflict swine worldwide. A common biological property of these viruses is their primary replication in host alveolar macrophages (Wensvoort et al., 1991) and some cells of the monocyte lineage (Pol and Wagenaar, 1992; Voicu et al., 1994), from where they may influence the host immune response. PRRSV-induced damage to alveolar macrophage and local immunity may be important in the pathogenesis of PRRSV-induced increased susceptibility to other pathogens such as PCV2 (Chung et al., 2005), swine influenza virus (SIV) (Pol et al., 1997), CSFV (Albina et al., 2000), *Streptococcus suis* (Galina et al., 1994; Zhao et al., 2009), *Haemophilus parasuis* (Solano et al., 1997), *Salmonella choleraesuis* (Wills et al., 1997a), *Mycoplasma hyopneumoniae* (Thacker et al., 1998), *Actinobacillus pleuropneumoniae*, and *Pasteurella multocida* (Carvalho et al., 1997; Pol et al., 1997). The clinical expression of combined infections is more pronounced and complicated, which is not easy to be diagnosed. Meanwhile, this multifactorial disease complex increased infection pressure in the swine herds.

Additionally, PRRS virus can localize in various organ systems (Mengeling et al., 1995, 1996a,b; Rossow et al., 1995; Shibata et al., 1997) and produce persistent

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infections in the absence of viremia (Allende et al., 2000; Van Reeth, 1997; Wills et al., 1997b). Persistent infection of PRRSV plays an important role in PRRSV survival and transmission, and will likely pose a major obstacle in PRRS control programs (Done et al., 1996).

PRRSV is highly heterogenic and is divided into European and American subtypes based on genetic, antigenic and pathogenic differences (Meng, 2000). PRRSV has the ability to continuously undergo genetic change (Chang et al., 2002). For example, a highly pathogenic PRRS appeared in pigs in the central region of China and spread quickly to neighboring provinces in 2006 (Li et al., 2007; Tian et al., 2007; Tong et al., 2007; Zhou et al., 2008). This rapid evolution is driven by mutations, RNA recombination and geographical redistribution of PRRSV genotypes (Murtaugh et al., 2001, 2010), and results in emergence of new isolates with different levels of pathogencity and virulence expressed as a wide variety of clinical signs (Halbur et al., 1996; Mengeling et al., 1996a,b; Tian et al., 2007). Genetic diversity also makes the efficacy of current vaccines inability (Labarque et al., 2004). These factors above work together to complicate long-term control measures.

The key elements of a PRRS control and eradication programme are early disease detection and rapid laboratory confirmation to determine the microbial ecology within an infected production system control of the infection through different stamping out strategies to reduce infection pressure at various stages of production, and increase breeding herd immunity. Control options will depend on pig density, the degree of multi-site structure of farms, the movement of pigs. To date, different strategies aimed at PRRS control have been designed, for example: nursery depopulation (Dee and Joo, 1995), test and removal (Dee, 1998), mass vaccination with unidirectional pig flow (Dee and Philips, 1998), herd closure (Corzo et al., 2010) and control without vaccination or depopulation (McCaw and Henry, 1995). Under field conditions, the strategic combination of mass vaccination using PRRS MLV products and management of pig flow is beneficial for reducing persistence and duration of shedding, but not for elimination of the wild-type virus from the pigs, which is a successful approach to control PRRSV transmission in acutely infected swine herds (Dee and Philips, 1998; Gillispie and Carroll, 2003; Ridremont and Lebret, 2006; Rodriguez et al., 2006).

The aim of the present study was therefore to describe the carrying and infection patterns of batches of sows to various pathogens in PRRSV positive farms, and investigate on the effective vaccination strategies by clinical assay as well as large scale farm questionnaire.

2. Materials and methods

2.1. Farms selection and collected information

Four representative farms (S, Z, H and J), located in Shanghai, Zhejiang, Hubei and Jiangxi, respectively, were selected and distributed in the main swine production regions of China. Admission criteria used to select the farms were: all-in/all-out production, presence of two identical growing/finishing units, capacity to produce approximately 200 piglets within a time span of 1 month, farrow-to-finish herd or herds being part of a closed production system, a history of endemic PRRSV manifested as periodic mini-outbreaks of PRRS, and the farmers' knowledge of clinical signs and gross lesions. Dominant breeds in the farms were crosses between Landrace and Yorkshire (sows) with Duroc or Hampshire (boars). Information on pig production was gathered by interviews with farmers regarding sow scale, stocking density, reproductive performance, morbidity, mortality and hygienic controls, including a history of medications and vaccinations for pigs over the past year. During the lactation, nursery and finishing stage, litter mortality was calculated as the number of dead piglets/number in the litter. Farm mortality was the number of pig deaths/pig population before disease plus pigs born or introduced during the epizootic. Data on the size, productivity and mortality rate of each farm are given in Table 1.

2.2. Clinical examination and sampling

Pigs in farm (S, Z, H and J) were evaluated daily between 08:30 and 09:00 h by trained herd technicians for clinical signs, including appetite (abdominal fill), depression, chills, cough, tachypnea, dyspnea, vomiting, diarrhea, abortion, behavioral changes (clinical lameness), and central nervous system (CNS) scores.

During a study period from January 2008 to September 2010, tonsil, nasal, oro-pharyngeal, rectal swabs and blood samples for pathogens surveillance were taken from pigs. Serum was harvested by centrifugation for 10 min at 3000 rpm and stored at −20 °C until testing for the presence of antibodies. At necropsy, dead and sick pigs were examined for gross pathological findings, and organs and tissues including tissues including brain, lung, heart, liver, spleen, tonsil, thymus, intestine, lymph nodes (superficial inguinal, submandibular and mediastinal) and kidney were sampled for bacteriological and virological examinations.

Table 1
Reproductive performances of the four swine farms (S, Z, H, and J).

Swine farm	No. of sow	Abortion rate (%)	Mummified or dead piglets rate (%)	Mortality rate (%)			
				Lactation stage	Nursery stage	Fattening stage	Total
S	1200	12.56	15.34	14.23	5.67	3.76	23.66
Z	1500	10.29	14.45	12.03	4.52	3.79	20.34
H	1300	4.86	6.75	6.54	26.31	4.03	36.88
J	1000	7.31	8.16	14.23	10.53	2.89	27.65