Persistence of Porcine Reproductive and Respiratory Syndrome Virus in Intensive Farrow-to-Finish Pig Herds

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ABSTRACT

An epidemiological study of porcine reproductive and respiratory syndrome (PRRS) within pig herds was conducted in 8 intensive farrow-to-finish pig farms. Persistence of PRRS virus (PRRSV) in pig herds was demonstrated by regular postmortem examination on 2 farms for a period of 2 y. Virus isolation and serum neutralization (SN) tests were performed on the sera collected from 9 groups of pigs (10 pigs/group) of various ages on 8 pig farms. Except for 1 farm, isolation rates of PRRSV reached the highest level of 70 to 100% of pigs 6 to 8 wk of age, which coincided with the lowest levels of maternal immunity. In 1 pig herd, sows (39 in total) with SN titers of ≤ 1:2, 1:4–1:8, and ≥ 1:16 were designated as groups 1, 2, and 3, respectively. Sera were obtained from their progeny (3 pigs randomly selected from each litter) at various ages from 0 to 22 weeks. A positive correlation (r = 0.377, P < 0.001) between the SN titers of sows and those of their progeny (1-week-old piglets) was observed. Pigs at the age of 6 wk, only 7.9% of group 1 pigs compared to 72.4% of group 3 pigs were seropositive. A significant difference (P < 0.01) in the percentage of pigs with PRRSV viremia among the 3 groups was observed, with the lowest level found in group 3 pigs. The isolation rates of PRRSV from serum reached the maximum at the age of 9 wk for all 3 groups. The results indicated that passively acquired serum antibodies conferred a protective effect for piglets; however, loss of passive immunity at various ages of pigs produced susceptible pigs that resulted in PRRSV persistence in the pig herds. Pigs 6 to 9 weeks old were the major reservoir for PRRSV in farrow-to-finish pig herds.

RÉSUMÉ

Une étude épidémiologique sur le syndrome reproducteur et respiratoire porcin (SRRP) fut menée dans huit fermes de type naisseur-finisseur. La persistence du virus du SRRP dans les élevages fut démontrée au moyen d'examens post-mortem réguliers sur deux fermes pendant une période de deux ans. L'isolement viral et une épreuve de séroneutralisation (SN) ont été effectués sur des sérums prélevés de neuf groupes de 10 porcs d'âges variés sur les huit fermes. À l'exception d'une ferme, les taux d'isolement du virus SRRP atteignaient leur plus haut niveau (70 à 100%) chez les animaux de 6 à 8 semaines d'âge alors que le niveau d'immunité maternelle est à son plus bas. Dans un des troupeaux, les truies (n = 39) avec des titres de ≤ 1:2; 1:4-1:8, et ≥ 1:16 furent désignées comme appartenant au groupe 1, 2 et 3, respectivement. Des sérums furent prélevés de leur progénie (3 porcs choisis au hazard dans chaque portée) à des âges variés de 0 à 22 semaines. Une corrélation positive (r = 0,377, P < 0,001) fut notée entre le titre en SN des truies et ceux de leurs porcelets à une semaine d'âge. Chez les porcs âgées de 6 semaines, seulement 7,9 % de ceux du groupe 1 étaient séropositifs comparativement à 72,4 % pour ceux du groupe 3. Une différence significative (P < 0,01) du pourcentage de porcs virémiques fut notée entre les trois groupes, les animaux du groupe 3 ayant le plus bas pourcentage. Les taux d'isolement du virus SRRP à partir du sérum ont atteint leur maximum à l'âge de 9 semaines pour les trois groupes. Les résultats indiquent que l'acquisition passive d'anticorps sériques confèrent une protection aux porcelets. Toutefois, la perte cette immunité passive à des âges différents entraîne la présence persistante de porcs susceptibles à une infection par le virus du SRRP dans les élevages. Les porcs âgés de 6 à 9 semaines constituaient le réservoir principal du virus du SRRP dans les troupeaux de porcs de type naisseur-finisseur.

(Traduit par docteur Serge Messier)

INTRODUCTION

Porcine reproductive and respiratory syndrome (PRRS) was first reported in North America in 1987 (1,2,3) and in Europe in 1990 (4,5). The causative agent of the syndrome, PRRS virus (PRRSV), has been isolated and characterized (4,6). The PRRSV is a small, enveloped positive-stranded RNA virus with a genome of 15 kb (7,8). This virus is currently classified as a member of the *Arteriviridae*. The disease is characterized by severe reproductive failure including late-term abortions, early farrowings, stillbirths, and weakborn piglets in sows and increased mortality in neonates, nursery and growing pigs (1,4,9). Numerous studies described that the disease can become endemic in highly populated pig-producing regions and pig herds. The

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most significant long-term consequences of endemic infection are the periodic increases in mortality of nursery and grower pigs together with normal or sub-optimal reproductive performance of sows (3,10,11). The mortality rate in nursery piglets can reach 25% at times. Death of pigs is usually caused by bacterial infection of *Salmonella choleraesuis*, *Streptococcus suis*, or *Haemophilus parasuis* secondary to PRRSV infection (3). The mechanism by which the virus can persist in pig herds is not fully understood.

Intensive farrow-to-finish pig farms with over 2000 sows boomed in the 1960's in Taiwan. A continuous flow system was used on most of these farms. Outbreaks of PRRS were observed in several herds between July 1991 and June 1993 (12,13). A marked increase in mortality due to bacterial pneumonia secondary to PRRS was found in nursery and grower pigs. The disease caused significant economic damage to pig producers in Taiwan. The numbers of pigs marketed per sow per year decreased from a range of 16 to 18 in 1990 to only 14 to 16 in 1995 in many pig herds (unpublished data). No new disease other than PRRS has been reported in Taiwan since 1990. Endemic infection with PRRSV was suspected to be the major cause of the decline in pig production. In response to this suspicion, studies were done and the report described here confirms the persistence of PRRSV in several intensive farrow-to-finish pig herds and investigates the kinetics of serology in relation to PRRS viremia in pigs in the field.

MATERIALS AND METHODS

FARMS SELECTED

Eight intensive, farrow-to-finish pig herds (Table II) with 2000 to 2500 sows each were selected for this study. A continuous flow operation system was used in these farms. Pigs at the ages of 4, 8–9, and 18–19 wk were weaned, moved to growing houses, and moved to finishing houses, respectively. Open buildings covered on both sides with plastic curtain were used for grower and finisher pigs. All of the pig farms had infection with PRRSV demonstrated by serological and virological surveys. Farms E and F belonged to the same owner and were located with a distance of 150 m separating them. Farms G and H belonged to the same company but were in different locations at a distance of 55 km apart.

SURVEY OF PRRS VIRUS INFECTION BY POSTMORTEM EXAMINATION

Necropsies were done once a month for farms A and B from July 1994 to June 1996. Detailed microbiological and pathological examinations were done on dead pigs and the causes of death were determined. Special efforts were made to examine lung lesions. When gross lung lesions were suggestive of PRRSV infection, lung, bronchial lymph nodes and spleen were collected for virus isolation and immunochemical staining.

CROSS-SECTIONAL SURVEY OF PRRS VIRUS INFECTION IN VARIOUS AGES OF PIGS

Epidemiological studies within each herd were conducted by testing several groups of pigs, including sows and pigs at the ages of 2, 4, 6, 8, 10, 12, 16, and 22 wk. Each group consisted of 10 pigs and a total of 90 sera was collected from each of the 8 intensive farrow-to-finish pig herds. A 2nd blood sampling was done 1 y after the 1st one for farms F, G, and H. Virus isolation and serum neutralization (SN) tests were performed on all serum samples collected.

EFFECT OF MATERNAL IMMUNITY ON THE INFECTION OF PIGS BY PRRS VIRUS

This study was done on farm E. A periodic increase of mortality, sometimes reaching 15% in nursery pigs, had been observed on this farm for the previous 2 y. Infection of PRRSV on this farm was confirmed by virus isolation from sick pigs 2.5 y prior. The most common cause of death was bacterial pneumonia secondary to PRRSV infection.

As a routine operation, sows were cleaned and moved to the farrowing house 7 d before their due date for farrowing. Sera were collected from a batch of sows 5 d before farrowing. Virus isolation and SN tests were performed immediately after the collection of serum. The sows were divided into 3 groups according to their SN titers to PRRSV.

Group 1: Thirteen sows with SN titers of $\leq$ 1:2.

Group 2: Fourteen sows with SN titers between 1:4 and 1:8.

Group 3: Twelve sows with SN titers of $\geq$ 1:16.

Three pigs from each litter were randomly selected for bleeding at the age of 0, 1, 2, 4 , 6, 9, 12, 16, and 22 wk. Pigs at the age of 0, 4, 9, and 16 wk were at the stages of prior to the intake of colostrum, before weaning, before moving to the grower house, and before moving to the finisher house, respectively. Virus isolation and SN test were performed on all serum samples.

VIRUS ISOLATION

Isolation of PRRSV was done as described previously (12,13). Pulmonary alveolar macrophages (PAM) were obtained from 3- to 6-week-old specific pathogen free (SPF) pigs. PAM cultures were prepared in 24-well plates, with 1×10^6 cells/well in 1 mL of RPMI-1640 medium containing 10% fetal calf serum (FCS), 0.15% sodium bicarbonate and antibiotics. Tissues for the isolation of PRRSV were minced, diluted 1:10 in minimum essential medium (MEM) with 2% FCS, homogenized and centrifuged at 4500 X *g* at 4°C for 30 min. The supernatants were filtered through 0.4 μm membranes. The filtrates and a 1:10 dilution were used as inocula. PAM cultures were inoculated with 100 μL of filtrates or serum/well and incubated at 37°C in a humidified chamber containing 5% CO_2. Cultures were monitored daily for the appearance of cytopathic effect (CPE). When CPE was observed, PRRSV was identified by indirect immunofluorescent antibody (IIF) assay using monoclonal antibody SDOW17 (kindly provided by Dr. Benfield, South Dakota State University, Brooklings, South Dakota, USA) against the nucleocapsid protein of ATCC VR-2332 strain of PRRSV. A detailed IIF assay has been described previously (12,14).

SERUM NEUTRALIZATION TEST

The SN test was done as described by Morrison et al (15). Briefly, the test was performed in 96-well flat-bottom microtitration plates using MA-104 cells (16). The first isolated

TABLE I. Detection of PRRS virus in tissues of pigs at postmortem examination

Pig farms	No. of pigs necropsied[a]	No. of pigs with interstitial pneumonia	No. of pigs with PRRS virus detected[b]
A	255	82(32.2%)[c]	54(21.2%)[d]
B	257	101(39.3%)	75(29.2%)

[a] Postmortem examination was done once a month for a period of 2 y
[b] PRRS virus was detected by virus isolation, immunoperoxidase staining or both
[c] No. of pigs with interstitial pneumonia/No. of pigs necropsied
[d] No. of pigs with PRRS virus detected/No. of pigs necropsied

TABLE II. Results of virus isolation from serum collected from pigs of various ages at the same time in intensive farrow-to-finish pig herds with endemic infection of PRRS virus

Age of pigs (wk)	Percentages of pigs with PRRS viremia at various ages of pigs at each pig farm[a]										
	A	B	C	D	E	F-1[b]	F-2[b]	G-1	G-2	H-1	H-2
2	0	0	10	0	10	0	0	10	0	0	0
4	0	0	30	0	10	0	10	10	10	0	0
6	10	20	60	70	80	0	80	100	80	0	23
8	90	70	70	60	20	100	60	50	60	0	32
10	40	30	10	10	30	10	20	10	20	0	8
12	10	0	0	10	0	10	10	0	10	0	0
16	10	10	10	10	10	0	0	0	0	0	0
22	0	0	0	0	0	0	0	0	0	0	0
sows	0	0	0	0	0	0	0	0	0	0	0

[a] Except for farm H-2, sera for the isolation of PRRS virus were collected from 10 pigs for each group of age at each farm
[b] Sera were collected from the same pig farm with an interval of one year. Same denotation for G- 1, G-2 and H-1, H-2

strain (MD001) of PRRSV in Taiwan was used in the SN test. By testing a small number of representative sera collected from the experimental pig farms, comparable SN titers were obtained using MD001 isolate or the homologous PRRS virus strain in the preliminary studies. All serum samples were heated at 56°C for 40 min and were serially diluted 2-fold in MEM containing 3% FCS. Each dilution of serum was mixed with an equal volume of PRRSV containing 100 $TCID_{50}$ in 100 μL medium. After incubation of plates at 37°C for 1 h, 200 μL of each mixture was added to the microtitration plate with MA-104 cell monolayers in the well. The plates were incubated at 37°C in an atmosphere of 5% CO_2 for 4 d, and checked for CPE. Antibody titers were expressed as the highest dilution of sera in which no CPE was observed. An antibody titer ≥ 1:2 was considered positive. Each serum sample was run in triplicate. A negative serum pool from SPF pigs and a positive serum pool collected from experimentally infected pigs with a SN titer of 1:32 were included in each batch of sera tested.

IMMUNOPEROXIDASE STAINING FOR PRRS VIRUS DETECTION IN TISSUE

Avidin-biotin-based immunoperoxidase technique for the detection of PRRSV antigens in tissues was performed as described previously (13,17). Frozen or dewaxed paraffin sections of lungs, bronchial lymph nodes and spleens were used in this study. Endogenous peroxidase was removed by immersion of the sections in 0.3% hydrogen peroxide at room temperature for 1 h, followed by 2 washes in phosphate buffered saline (PBS). Slides were then digested with 0.05% protease (protease XIV, Sigma Chemical Co., St, Louis, Missouri, USA) at 37°C for 15 min followed by washing in PBS. Blocking of nonspecific staining was done with a 5% solution of normal goat serum. After washing in PBS, primary monoclonal antibody SDOW17 diluted 1:1000 in PBS was added and incubated at 37°C for 1 h. The slides were washed in PBS and a biotinylated goat anti-mouse antibody (Dako Corp, Carpinteria, California, USA) was added and incubated at room temperature for 30 min followed by washing. The sections were incubated with peroxidase-conjugated streptavidin (Zymed Laboratories, South San Francisco, California, USA) for 40 min. After washing in PBS, 3,3′-diaminobenzidine tetrahydrochloride (Vector Laboratories, Burlingame, California, USA) was added and incubated for 5 min. The sections were then washed and stained with hematoxylin. Dark brown cytoplasmic deposits observed in macrophages indicated a positive reaction.

STATISTICAL ANALYSIS

Correlation coefficients were calculated between the SN titers of one-week-old piglets and those of their dams, and between the isolation of PRRSV from sera and the SN titers of pigs. The differences of the percentages of pigs with PRRS viremia and the rates of seropositive pigs among the 3 groups at each time point were determined by chi-square tests. A P value < 0.05 was considered statistically significant.

RESULTS

VIRUS DETECTION BY POSTMORTEM EXAMINATION

Of the 255 pigs necropsied during the period of 2 y from farm A, 82 (32.2%; Table I) had interstitial pneumonia characterized by the infiltration of alveolar septa with mononuclear cells especially macrophages, a few neutrophils and necrotic cell debris. Hyperplasia of type 2 pneumocytes and the accumulation of macrophages, degenerated cells and proteinaceous exudate were frequently observed in alveolar spaces. Among the 82 lungs suspected of infection with PRRSV, 54 were confirmed by virus isolation, immunoperoxidase staining or both. Similar results were found at farm B but with a higher percentage of pigs with suspected and confirmed infection with PRRSV. Of the 69 bacterial cultures isolated secondary to PRRSV infection in pigs, 27 (39.1%), 14 (20.3%), and 7 (10.1%) were *Salmonella choleraesuis*, hemolytic *Escherichia coli*, and *Streptococcus suis*, respectively.

CROSS-SECTIONAL SURVEY OF PRRS VIRUS INFECTION IN VARIOUS AGES OF PIGS

Pigs from all farms except H-1 had PRRS virus isolated from serum

(Table II). With the exception of farm C, PRRSV isolation rates were 0% or only 10% for pigs before the age of 4 wk. Virus isolation rates reached the highest levels ranging from 70 to 100% of pigs when pigs were 6 or 8 wk old. The number of pigs with PRRS viremia decreased thereafter. At the age of 12 and 16 wk, only 10% of pigs at most on any farm had PRRS viremia. No virus could be isolated from the sera collected from 22-week-old pigs or sows.

No PRRSV could be isolated from the first batch of serum samples collected from farm H. However, antibodies against PRRSV existed in the pigs (Table III). An increased sample size was determined for the collection of serum samples 1 y later and the isolation rates (Table II) of PRRSV from the sera were 5/22 (23%), 7/22 (32%), and 1/12 (8%) for pigs at the ages of 6, 8, and 10 wk, respectively. Sera were also obtained from 10 sick pigs, 6 and 8 weeks of age for the isolation of PRRSV. Five of the 10 pigs had PRRS viremia.

Similar serological profiles were observed for pig herds from farms A to G. An example of the distribution of SN titers in pigs of various ages is shown in Table IV. Most of the sows had high SN titers to PRRSV. Consequently, high antibody titers were observed for 2-week-old piglets due to passive immunity. As the age of pigs increased, antibody titers decreased and were lowest in pigs 6 or 8 wk old. Thereafter, antibody titers increased due to natural infections with PRRSV. Similar serological profiles, but with much lower SN titers than other tested herds, were observed for the serum samples collected from the 1st and 2nd bleedings at farm H (Table III).

EFFECT OF MATERNAL IMMUNITY ON THE INFECTION OF PIGS BY PRRS VIRUS

All piglets were seronegative to PRRSV before the intake of colostrum (week 0, Figure 1). Except for 1 pig in group 1 and another in group 2, all one-week-old pigs were seropositive. Although SN titers of group 1 sows were ≤ 1:2, 97.4% of their progeny had SN titers equal to or higher than 1:2. There was a positive correlation ($r = 0.377$, $P < 0.001$) between the SN titers of one-week-old piglets and those of their dams. Levels of

TABLE III. Results of a one-time survey of serum neutralization titers to PRRS virus in pigs of various ages at H-1 farm

Age of pigs (wk)	Distribution of SN titers to PRRS virus in pigs of various ages[a]							
	< 1:2	1:2	1:4	1:8	1:16	1:32	1:64	≥ 1:128
2	5[b]	2	3					
4	6	2	2					
6	4	4	1	1				
8	2	1	1					
10	7	1	2					
12	6	2	2					
16	7	2	1					
22	7	3						
sows	2	3	3	2				

[a] Sera were collected from 10 pigs for each group of age
[b] Numbers of pigs

TABLE IV. Results of a one-time survey of serum neutralization titers to PRRS virus in pigs of various ages at G-1 farm

Age of pigs (wk)	Distribution of SN titers to PRRS virus in pigs of various ages[a]							
	< 1:2	1:2	1:4	1:8	1:16	1:32	1:64	≥ 1:128
2		4[b]	1	1		2	1	1
4	1	1	3	4		1		
6	8		1	1				
8	4	2	1	3				
10		2	3		3			2
12	1		2	1	1	4		1
16	1	1	5		1	1		1
22	4	2			3			1
sows	1	1			1	2	3	2

[a] Sera were collected from 10 pigs for each group of age
[b] Numbers of pigs

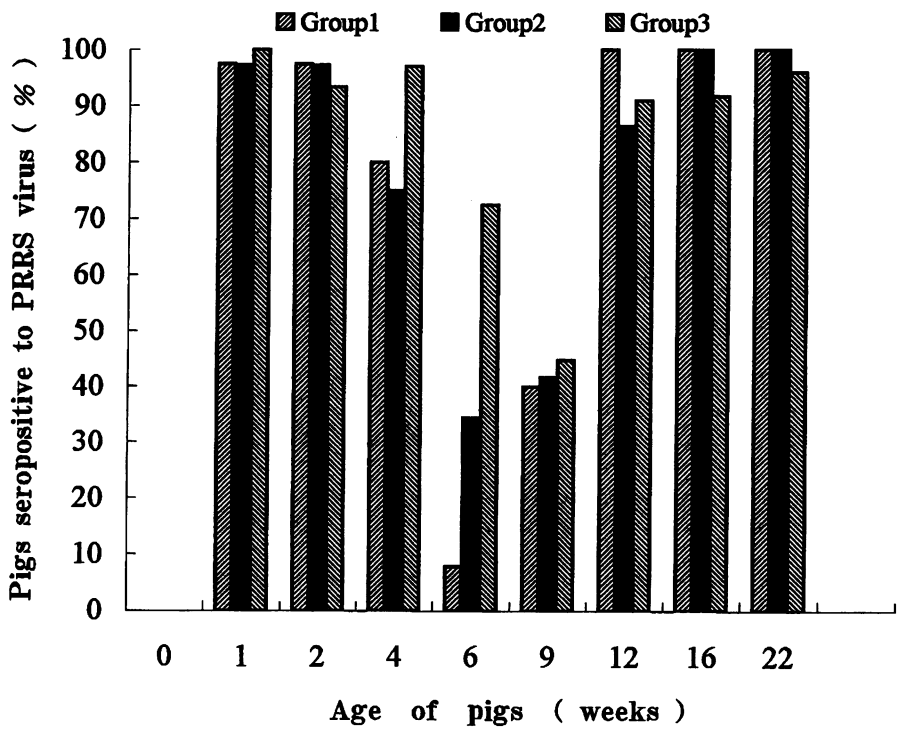

Figure 1. Percentages of pigs with positive serum neutralization titers for PRRS virus from the age of O (prior to the intake of colostrum) to 22 wk in a PRRS endemic farm. Pigs were divided into 3 groups according to the serum neutralization titers of their dams (Group 1: progeny of sows with SN titers ≤ 1:2; Group 2: progeny of sows with SN titers 1:4-1:8; Group 3: progeny of sows with SN titers ≥ 1:16). Sera of 3 pigs from each litter were collected.

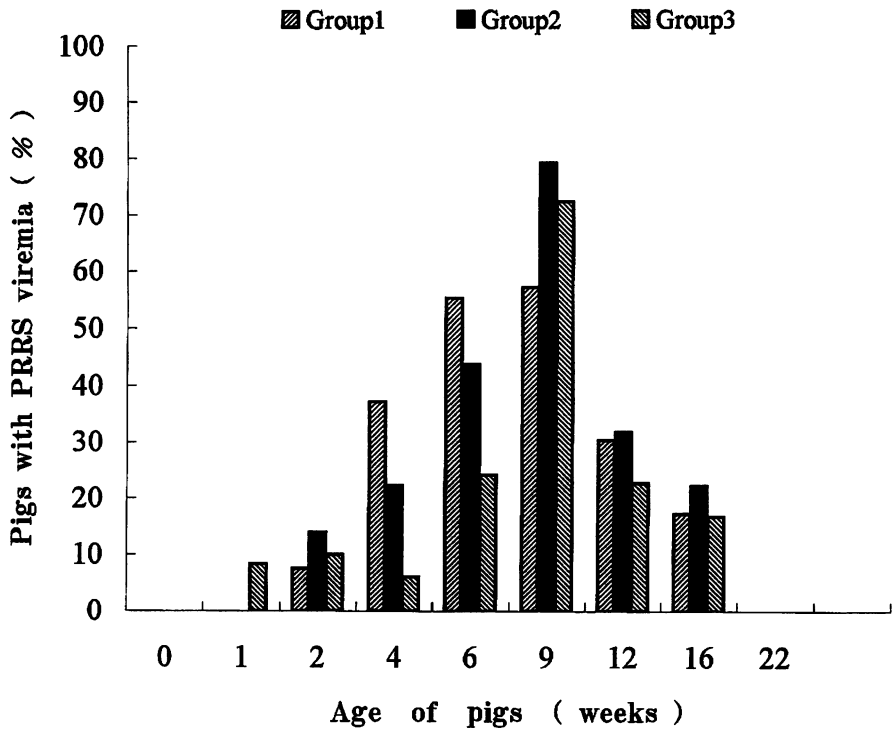

Figure 2. Percentages of pigs with PRRS viremia from the ages of O (prior to the intake of colostrum) to 22 wk in a PRRS endemic farm. (See also the legend of Figure 1.)

maternal antibodies remained relatively high but gradually declined before the age of 4 wk. The percentage of pigs with positive SN titers dropped from 80.0% at 4 wk of age to 7.9% at 6 wk of age for group 1 piglets. Similar kinetics of serology were observed for group 2 pigs but the lowest percentage of seropositive pigs was 34.4% in pigs 6 wk old. Thereafter, PRRSV antibody titers in pigs continually increased and all pigs were seropositive at the ages of 12 and 16 wk for groups 1 and 2 pigs. For pigs in group 3, 97.0% of pigs remained seropositive at the age of 4 wk but the seropositive rate decreased as pigs grew older and dropped to its lowest level of 44.8% when pigs were 9 wk old. An obvious increase of seropositive pigs was observed from the age of 9 wk to 12 wk and remained high throughout the experimental period. Significant differences in the rates of seropositive pigs to PRRSV among the 3 groups were found at the age of 4 ($P < 0.05$) and 6 ($P < 0.01$) wk.

No PRRSV was isolated from the pigs prior to the intake of colostrum (week 0) and at the age of 22 wk (Figure 2). At the age of 1 wk, PRRSV was isolated only from the sera of 3 (8.3%) of 36 pigs in group 3. Two of them had SN titers $\geq$ 1:128 and were born to the same sow. Another one with a SN titer of 1:64 belonged to another litter. The percentages of pigs with PRRS viremia continually increased and reached maximal levels of 57.1%, 79.2%, and 72.4% for groups 1, 2, and 3, respectively, for pigs at the age of 9 wk. Significant differences were found in the isolation rates of PRRSV from sera among the 3 groups of pigs at the ages of 4 ($P < 0.01$) and 6 ($P < .05$) wk. A negative correlation ($r = -0.386$, $P < 0.001$) was observed between the isolation of PRRSV from sera and the SN titers of pigs.

DISCUSSION

Persistence of PRRSV in intensive farrow-to-finish pig herds was demonstrated by a two-year-period of postmortem examinations at farms A and B. Surveys of PRRSV infection (Table II) with an interval of one year conducted at farms F, G, and H, further confirmed that many intensive, farrow-to-finish pig farms in Taiwan suffer from endemic infection with PRRSV. Similar results had been reported by other researchers. More than 2.5 y after an initial outbreak of PRRS, virus was isolated from sick pigs in nursery houses (11,18). Studies on the isolation of PRRSV from serum of pigs of various ages demonstrated that virus circulated in 3- to 12-week-old pigs. Researchers in the Netherlands also reported (9) that 1 y after the original disease outbreaks, antibodies against PRRSV were detected in 3–5 month-old replacement gilts, indicating the persistence of virus in pig herds.

Results of virus isolation tests on sera collected from cross-sectional (Table II) and sequential sampling (Figure 2) were consistent. PRRSV could be isolated from sera of 1- to 16-week old pigs with highest isolation rates in 6- to 9-week-old pigs. A similar study was conducted by Stevenson et al (11) in a 500 sow farrow-to-finish pig herd. They concluded that the pigs in the nursery and growing houses were the major reservoir for PRRSV. Based on the information mentioned above, an epidemiological survey of the infection of PRRSV in selected herds was conducted in Taiwan (paper submitted). A total of 10 or 20 of 6- to 9-week-old pigs were bled in each of 38 herd. Virus isolation using swine pulmonary alveolar macrophages was performed within 24 h after the collection of serum. Of 688 sera from 38 swine herds tested, 428 sera (62.2%) had PRRSV isolated and 36 farms (94.7%) had at least two or more pigs that were PRRSV positive. This further supports the conclusion that most pig herds in Taiwan are endemically infected with PRRSV.

Results of this study provide evidence to support the profound protective effect of maternal immunity against PRRSV infection. A positive correlation between the SN titers of sows and those of their 1 wk old progeny was demonstrated. Significant differences in the rates of seropositive pigs among the 3 groups at the age of 6 wk was observed. Only 7.9% of group 1 pigs (born to the sows with SN titers $\leq$ 1:2) compared to 72.4% of group 3 pigs (born to the sows with SN titers $\geq$ 1:16) were seropositive. In the cross-sectional survey of PRRSV infection done in the 8 pig herds, SN titers of pigs reached the lowest levels at the age of

6 or 8 wk, which coincided with the highest isolation rates of PRRSV from the sera. A negative correlation between the isolation of PRRSV from serum and the SN titers of pigs was observed. Finally, a significant difference in the percentage of pigs with PRRS viremia among the 3 groups was observed, with lowest level found in group 3 pigs at the age of 4 and 6 wk. The above information indicates that pigs with higher maternal immunity stay seropositive longer and thus the age of pigs with the highest rates of PRRS viremia was higher.

Keeping sows with consistent and high antibody titers to PRRSV might be beneficial to the intensive farrow-to-finish pig herds. High maternal immunity can provide stronger and longer lasting protection for piglets against infection with PRRSV. More consistent levels of maternal antibody could result in the loss of passive immunity of pigs at the same time and shorten the duration of pigs with PRRS viremia on a herd basis. A decrease in concentration of PRRSV circulating in the pig herds can be expected. Furthermore, outbreaks of reproductive failure caused by PRRSV with irregular intervals of 2 to 4 y occurred in some pig herds (10, unpublished observation). Keeping sows with high antibody titers against PRRSV can minimize the loss due to reproductive failure in pregnant sows. A vaccine suitable for use in sows might be available in the near future (19).

Results from this study and others (9,20) reveal that multiple factors contribute to the persistence of PRRSV in intensive farrow-to-finish pig herds. Maternal immunity confers a protective effect for piglets; however, loss of passive immunity at various ages of pigs based on the antibody levels transferred from the dams provided a constant stream of susceptible pigs for PRRSV allowing the viral burden to build up in pig herds. The source of infection for susceptible pigs to continue the chain of infection is another key factor for the persistence of PRRSV in pig herds. As described above, pigs at the age of 6 to 9 wk were the major reservoir for PRRSV and thus play a key role in the dissemination of virus within swine herds. PRRSV could not be isolated from the serum of sows, however, with the exception of farm H, about 70% of sows in the tested herds were seropositive. An indication of active infection or heavy contamination existed in the sow population. Persistent infection of PRRSV in individual pigs for extended periods of time has been reported (21,22). Re-excretion of PRRSV in pigs by exogenous corticosteroid treatment 15 wk after the initial seroconversion to the virus has also been demonstrated (20). Shedding of virus from sows under certain circumstances such as pregnancy, farrowing, or regrouping, might happen and could result in vertical or horizontal transmission of PRRSV to susceptible pigs. Seronegative replacement gilts and sows susceptible to PRRSV could also act as the source of infection by in utero transmission or by shedding the virus to infect the piglets after birth (9,10,20).

In farm E, pigs with PRRS viremia at the ages of 1 and 2 wk (Figure 1) presumably provided the source of infection for susceptible pigs when the maternal immunity waned. No PRRSV and serum neutralization antibody were detected in the serum collected from the pigs prior to the intake of colostrum. An indication of in utero infection of PRRSV did not occur during the experimental period. Consequently, the source of PRRSV infection in pigs 1- to 2-week-old could be the environment after the birth of pigs. The identification of the origin of PRRSV and the transmission routes could provide control strategies to stop initiation of the infection in the pig herds. Among the 14 piglets mentioned above, 11 (78.6%) had SN titers of $\geq$ 1:16. Coexistence of PRRSV and antibodies in pigs has been reported by researchers (21). Antibody-dependent-enhancement of PRRSV replication has been demonstrated in vitro (23) and in vivo (24). The detailed mechanism by which PRRSV can infect piglets in the presence of high SN titers is not clear and merits further study.

Farms G and H belong to the same company and are located 55 km apart. The buildings, management system and feeds were similar for both farms. Replacement gilts were imported monthly from the same multiplier herd in which endemic infection of PRRSV has been confirmed. The average monthly mortalities of pigs after weaning (including nursery, grower and finisher pigs) were 3.16% and 1.42% for farms G and H, respectively from July 1994 to June 1996. Farm G had endemic infection of PRRSV similar to other pig herds tested. However, no virus could be isolated from the first batch of serum samples collected from farm H (Table II). Much lower isolation rates of PRRSV than those of other tested herds were also observed for the 2nd batch of serum samples. Furthermore, antibody against PRRSV existed in the pigs of farm H but with much lower SN titers than those of other tested pig populations (Tables III & IV). The above information indicated that a relatively lower concentration of PRRSV was circulating in pigs of farm H than in other pigs in the other herds and with only minimal damage to the pig production. The reasons for the difference in the epidemiology of PRRS and the productive performance in farms G and H are not clear. An isolated environment with good hygiene status and proper density, temperature, humidity, and ventilation for pigs were considered to be major factors contributing to the difference. A detailed investigation is ongoing in both farms. The findings of the differences between farms G and H might be the key to solving the PRRS problem for the intensive farrow-to-finish pig herds. More studies are needed to develop effective prevention and control strategies to reduce the economic losses caused by PRRS in pig production.

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