The impact of endemic porcine reproductive and respiratory syndrome virus and other pathogens on reproductive performance in swine

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Summary

Objectives: To evaluate the impact of endemic infection with porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), transmissible gastroenteritis virus-porcine respiratory coronavirus (TGEV-PRCV), pseudorabies virus (PRV), *Mycoplasma hyopneumoniae,* and *Actinobacillus pleuropneumoniae* (APP) on the reproductive performance of sows.

Methods: Seventeen groups of 30 to 60 sows and gilts in seven herds were monitored over a 2-year period by serological testing for the pathogens listed above. Litter size, number of stillborn (including mummies), average weaning weight, preweaning deaths, and interfarrowing interval were recorded for the sows that were tested. Multiple linear regression analyses were performed to evaluate the association of the measures of reproductive performance with serological test results.

Results: Infection with PRRSV was consistently associated with poorer reproductive performance. Sows that had antibodies to PRRSV had, on average, 0.1 to 0.9 more stillborn piglets per litter than seronegative animals. The average interfarrowing interval was 3 to 10 days longer for PRRSV-seropositive sows. A consistent correlation between reproductive performance and serological results could not be found for SIV, TGEV-PRCV, PRV, *M hyopneumoniae,* or APP.

Implications: Even in herds where no clinical signs of the disease are present, reproductive performance may be substantially inferior in sows with high levels of antibody against PRRSV.

Keywords: swine, reproduction, endemic infection, porcine reproductive and respiratory syndrome virus

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Pathogens such as porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), transmissible gastroenteritis virus (TGEV), *Mycoplasma hyopneumoniae,* and *Actinobacillus pleuropneumoniae* (APP) are prevalent in a large percentage of swine herds in the United States.[1,2] Consequences of acute disease outbreaks due to these pathogens include abortions, stillbirths, reduced fertility of the sows, and increased mortality of the piglets.[3–6] However, herds may be infected with these pathogens without showing signs that differentiate or characteristically define the presence of a specific disease.[4,6,7] These endemic infections may be characterized by mild respiratory signs such as coughing or sneezing, or slightly reduced reproductive performance that does not elicit diagnosis and intervention by the producer.

Several studies have evaluated the financial impact of acute disease outbreaks in swine operations.[5,8–10] The economic consequences of endemic infection, on the other hand, are more difficult to measure. For decisions regarding herd health management, it is crucial to have information on the impact of infection on productivity. Interventions such as vaccination or elimination of a pathogen from a herd must be evaluated against the consequences of allowing the herd to remain endemically infected. The objective of this study was to estimate the impact of endemic infections with PRRSV, SIV, TGEV, pseudorabies virus (PRV), *M hyopneumoniae,* and APP on reproductive performance. These pathogens were selected for monitoring because of their potential impact on productivity,[4–6,11–14] and because a serological test that was economical and feasible for use under practice conditions was available for each organism. The impact of endemic infection with these pathogens on growth in finishing pigs has been evaluated previously.[15]

Materials and methods

Herds

This study was conducted in herds that met the following criteria for inclusion: herd size ≥100 sows, single-site management, use of computerized production records (PigChamp; Swine Data Management, Wheatland, Iowa), and a history of positive serological testing results for at least one of the monitored pathogens, as reported by the herd manager.

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Table 1: Characteristics of seven swine herds[1] serologically monitored in 1996 and 1997 for porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), transmissible gastroenteritis virus-porcine respiratory coronavirus (TGEV-PRCV), pseudorabies virus (PRV), *Mycoplasma hyopneumoniae (MH)*, and *Actinobacillus pleuropneumoniae* (APP)

	Herd						
	1	2	3	4	5	6	7
Years monitored	1996, 1997	1996	1996, 1997	1997	1997	1997	1997
Housing	Pasture	Confinement	Confinement	Confinement	Gestation outdoors	Confinement	Confinement
Number of sows	150	100	150	200	250	150	2000
Clinical disease history[2]	TGEV 1994	None	TGEV 1993	None	None	None	None
History of positive serological tests[2]	SIV	SIV, TGEV-PRCV, APP	PRRSV, SIV, TGEV-PRCV	SIV	PRRSV, SIV, MH	PRRSV, PRV	PRRSV, PRV
Vaccination of sows	None	SIV, TGEV	None	None	SIV, PRRSV[3]	PRV	PRV

[1] Sow herds with ≥100 sows, single-site management, use of computerized production records (PigChamp; Swine Data Management, Wheatland, Iowa), and a history of positive serological testing results for at least one of the monitored pathogens, as reported by the herd manager.

[2] Reported by herd manager.

[3] Sows were vaccinated beginning 4 months after the first test.

During the first year of the study, three university research herds were monitored, one of which (Herd 2) was depopulated at the end of the year. In that herd, only one group of 30 sows could be monitored before the farm was depopulated. In the second year, the two remaining and one new university research herd were monitored. Three commercial herds were also evaluated during 1997. A summary of herd characteristics and disease and vaccination history is provided in Table 1.

Sampling procedures

The study was conducted in 1996 and 1997, beginning in the spring of each year. Each herd was visited twice per year, with a 5- to 6-month interval between visits. In 1996, blood samples were collected from 30 sows in each herd, selected at random. This sample size provides a 95% probability of detecting seropositivity with a prevalence of at least 10%.[16] The sample size was increased to 60 sows per herd in 1997, because many sows were lost to follow-up during the first year. With a maintained sample size of 60, there is a 95% probability of detecting seropositivity if the prevalence is at least 5%. Whenever possible, sows sampled during the first herd visit were re-sampled on the second visit, with the remainder required to achieve the desired sample size selected at random. Data on reproductive performance of sows sampled for serological testing were obtained from computerized production records. For each sampling, the reproductive performance of the tested sows was recorded for the farrowing preceding and following the serological test. The recorded measures of production were the number of piglets born alive, the number of piglets born dead (including mummies), the number of piglets fostered on or off, the number of pigs weaned, the weaning weight of the litter, and the time between two consecutive farrowings (interfarrowing interval).

Serological testing

Endemic infections with the following pathogens were monitored by serological assays performed at the University of Illinois Veterinary Diagnostic Laboratory (Champaign, Illinois): PRRSV, HerdChek PRRS ELISA (Idexx, Westbrook, Maine), positive cutoff sample-to-positive (S:P) ratio 0.4; SIV, hemagglutination inhibition test for serotype H1N1, positive cutoff titer 40; TGEV-porcine respiratory coronavirus (PRCV), serum neutralization (Purdue strain TGEV), positive cutoff titer 64; and *M hyopneumoniae*, ELISA, positive cutoff S:P ratio 0.6.

The HerdChek Anti-PRV g1 assay (Idexx), was performed at Illinois Department of Agriculture Animal Disease Diagnostic Laboratory (Galesburg, Illinois), positive cutoff S:P ratio 0.7.

A complement fixation test for APP (serotypes 1, 3, 5, and 7) was performed at the Iowa State University Veterinary Diagnostic Laboratory (Ames, Iowa), positive cutoff titer 8.

Statistical methods

A difference in reproductive performance among seropositive and seronegative animals of the magnitude of the standard deviation can be detected with a sample size of 30 if the seroprevalence is 50% (power=0. 8 and α=0. 05).[16] With a lower or higher prevalence, only a difference among groups that is somewhat larger than the standard deviation can be detected with the same power, because, in this case, the sample size in one group becomes smaller. To evaluate the impact of endemic infection on sow productivity, multiple linear regression analyses were conducted (PROC REG, SAS Institute, Cary, North Carolina). This statistical method allows evaluation of the effect of a higher antibody titer

to a pathogen on reproductive performance, taking confounding factors into account. The calculated effect is corrected for the influence of other pathogens, as well as covariates such as parity of the sow or group tested. The outcomes were interfarrowing interval, number of liveborn piglets, number of stillborn piglets (including mummies), preweaning deaths, and weaning weights for the litters preceding and following the serological testing. The average weaning weight per pig was calculated as the weight of the litter at weaning divided by the number of pigs weaned. The number of preweaning deaths was calculated as the number of piglets born alive plus (or minus) the number of piglets fostered on (or off), minus the number of pigs weaned. All reproductive measures except interfarrowing interval were recorded for the farrowing before and the farrowing after the serological testing. The interfarrowing interval was calculated from the farrowing before to the farrowing after the serological testing. Sows that did not have records of two consecutive farrowings during the observation time were excluded from the analysis of interfarrowing intervals. The data for each herd were analyzed separately. In all of these models, serological test results for each of the monitored infections were used as the predictors. The proportion of variance accounted for uniquely by each predictor after all other variables in the model had been taken into account, the squared semipartial correlation coefficient (sr^2), was calculated. Serological test results were entered into the analyses as titer values or ELISA S:P ratios rather than as positive and negative values in order to utilize more precise quantitative information for the regression models. Parity was included as a covariate in all regression models because it has been reported to influence reproductive performance.[17] In Herd 5, vaccination status differed between the two groups of sows tested. In this herd, the product of vaccination status and serological titer was included as an interaction variable. Regression models were tested for satisfaction of the assumptions of normality, homogeneity of variance, the presence of influential observations, and independence of residuals.[18] The method of variable selection was initial forced entry of all predictors, with stepwise backward elimination. In order to control for confounding in the model, the *P*-to-remove criterion was set to 0.2. Thus, all variables with a *P*-value <0.2 were kept in the model. The level of significance was set to α=0.05. Predictions were that higher serological titers would be associated with fewer liveborn piglets, more stillborn piglets and mummies, more preweaning deaths, a lower average weaning weight, and a longer interfarrowing interval. In vaccinated animals, higher antibody levels might be associated with either a strong immune response to vaccination or an infection with the pathogen. Therefore, no predictions were made for vaccinated animals unless the serological test was capable of distinguishing field virus from vaccine virus.

Results

Reproductive performance of the study herds is summarized in Table 2. Serological test results for the monitored herds are presented in Table 3. Six of the seven herds were seropositive for PRRSV. In all herds tested, seroprevalence of both SIV and *M hyopneumoniae* was high. In five herds, more than 50% of sows tested positive for TGEV-PRCV. There was a low prevalence of APP in three herds. In two herds, there

Table 2: Mean (+SD) for measures of reproductive performance in seven swine herds before (Litter 1) and after (Litter 2) serological testing[1] for porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), transmissible gastroenteritis virus-porcine respiratory coronavirus (TGEV-PRCV), *Actinobacillus pleuropneumoniae* (APP), *Mycoplasma hyopneumoniae*, and pseudorabies virus (PRV). Herds had a history of positive serological testing results for at least one of the six pathogens.

	No. liveborn piglets		No. stillborn or mummified		No. preweaning deaths		Mean weaning weight (kg)		Interfarrowing interval (days)	
	Litter 1	Litter 2	Litter 1	Litter 2	Litter 1	Litter 2	Litter 1	Litter 2	Litter 1	Litter 2
Herd 1 (1996)	10.8 ± 2.7	10.2 ± 3.5	1.1 ± 1.4	2.0 ± 2.2	1.7 ± 1.8	3.2 ± 3.0	6.9 ± 1.2	6.8 ± 1.4	148.4 ± 3.6	151.0 ± 11.2
Herd 1 (1997)	10.7 ± 2.5	11.0 ± 2.9	1.1 ± 1.6	0.9 ± 1.4	2.0 ± 2.2	2.1 ± 2.2	6.8 ± 1.5	7.4 ± 1.4	152.7 ± 21.3	152.1 ± 13.7
Herd 2 (1996)	9.4 ± 2.8	9.4 ± 2.9	0.3 ± 0.6	0.2 ± 0.4	0.7 ± 1.0	0.9 ± 1.1	5.7 ± 0.9	5.0 ± 1.3	143.2 ± 9.1	149.1 ± 34.6
Herd 3 (1996)	10.4 ± 3.1	10.2 ± 3.4	0.5 ± 0.9	1.1 ± 2.2	1.5 ± 1.6	1.9 ± 1.5	5.3 ± 1.1	5.3 ± 1.2	138.3 ± 4.0	142.2 ± 42.5
Herd 3 (1997)	10.8 ± 2.8	10.7 ± 3.2	0.8 ± 1.3	0.9 ± 1.5	1.3 ± 1.2	1.6 ± 1.9	4.9 ± 1.2	4.8 ± 1.0	154.5 ± 43.2	155.4 ± 41.9
Herd 4 (1997)	9.3 ± 3.1	8.9 ± 2.8	1.8 ± 1.9	1.8 ± 2.0	0.7 ± 1.0	0.4 ± 0.6	5.2 ± 1.3	5.4 ± 1.3	152.1 ± 18.9	151.0 ± 17.3
Herd 5 (1997)	9.6 ± 2.7	9.2 ± 3.4	0.6 ± 1.0	0.5 ± 0.8	1.6 ± 1.8	1.6 ± 2.3	5.9 ± 1.1	5.9 ± 1.1	152.2 ± 25.1	158.3 ± 34.2
Herd 6 (1997)	10.0 ± 2.5	9.7 ± 2.7	0.7 ± 1.0	0.6 ± 1.0	1.0 ± 1.1	1.4 ± 1.9	6.7 ± 1.2	6.6 ± 1.0	148.4 ± 18.0	146.9 ± 16.9
Herd 7 (1997)	8.8 ± 2.9	9.3 ± 2.4	0.8 ± 1.3	0.7 ± 1.2	1.1 ± 1.4	1.5 ± 2.6	6.0 ± 1.1	6.1 ± 1.3	146.2 ± 14.6	157.3 ± 32.8

[1] Serological tests used were HerdChek PRRS ELISA (Idexx, Westbrook, Maine); hemagglutination inhibition test for SIV (H1N1); serum neutralization (Purdue strain) for TGEV-PRCV; ELISA for *M hyopneumoniae;* complement fixation test for APP (types 1, 3, 5, and 7); and PRV HerdChek ELISA (Idexx).

was a history of prior infection with PRV: no animals were sero-positive in one of these herds, and only a few were seropositive in the other.

The most consistent associations between measures of reproductive performance and serology results detected were for PRRSV, which was associated with lower measures of reproductive performance in five of the seven herds. In Herd 6, higher PRRSV ELISA S:P ratios were correlated with fewer piglets born alive (sr^2=0.06, *P*=.009). A sow with an S:P ratio of 0.4 (positive cutoff) had, on average, 0.34 fewer live-born piglets per litter than a sow with an S:P ratio of 0. An S:P ratio of 1.41 (75th percentile) in Herd 6 was associated with an average of 0.72 fewer piglets compared to a ratio of 0.56 (25th percentile). In contrast, in Herd 5, in which one group of sows was vaccinated, a higher PRRSV ELISA S:P ratio was associated with more piglets born alive (sr^2=0.09, *P*=.01). Furthermore, in Herds 3, 4, and 5, a higher PRRSV S:P ratio was associated with more stillborn and mummified piglets. In Herd 3 (1996), an S:P ratio of 0.4 (positive cutoff) was associated with an average of 0.52 more stillborn piglets and mummies compared to an S:P ratio of 0 (sr^2=0.12, *P*=.004). There was a difference of 1.24 stillborn piglets and mummies per litter between sows with an S:P ratio=0.08 (25th percentile) and those with an S:P ratio=1.03 (75th percentile). This effect was smaller in 1997, when seropositive sows had, on average, only 0.14 more stillborn piglets and mummies than seronegative sows (sr^2=0.04, *P*=.049). All sows in Herd 4 were seronegative for PRRSV. Nevertheless, sows with an S:P ratio of 0.04 (75th percentile) had an average of 0.90 more stillborn piglets and mummies per litter than sows with an S:P ratio of 0 (25th percentile) (sr^2=0.15, *P*=.004). In Herd 5, only the second group of sows was vaccinated against PRRSV. The effect of PRRSV S:P ratio on the number of stillborn pigs and mummies differed between the vaccinated and non-vaccinated groups. There was a marked increase in the number of stillborn piglets and mummies with a higher PRRSV S:P ratio in unvaccinated sows (sr^2=0.10, *P*=.003). In vaccinated sows, the number of stillborn piglets increased only slightly with higher S:P ratios.

In Herd 1 (1996), piglets of sows that were seropositive for PRRSV were more likely to die between birth and weaning than were piglets from seronegative sows (sr^2=0.02, *P*=.045). In Herd 5, a higher PRRSV S:P ratio was associated with a lower average weaning weight (sr^2=0.06, *P*=.018). In Herd 2, in contradiction to the predicted direction of the effect, higher PRRSV S:P ratios were associated with higher average weaning weight (sr^2=0.24, *P*=.016). In Herds 3, 4, and 5, a higher PRRSV S:P ratio was associated with a prolonged interfarrowing interval. In 1997, the interfarrowing interval for sows with a PRRSV S:P ratio of 0.4 (positive cutoff) was 4.6 days longer in Herd 5 (sr^2=0.09, *P*=.014) and 10.4 days longer in Herd 3 (sr^2=0.05, *P*=.036), compared to sows with an S:P ratio of 0. The difference in interfarrowing interval between sows with the 25th and 75th percentile PRRSV S:P ratios in 1997 was 10.9 days in Herd 3 and 24.7 days in Herd 5. In Herd 4, although all animals were negative for PRRSV, higher PRRSV S:P ratios were correlated with a longer interfarrowing interval (sr^2=0.15, *P*=.005). Sows with an S:P ratio of 0.04 (75th percentile) on average had an interfarrowing interval 2.6 days longer than sows with an S:P ratio of 0 (25th percentile).

A consistent negative association with reproductive performance across herds could not be determined for any of the other monitored pathogens. Endemic SIV infection was associated in Herd 6 (1997) with fewer piglets born alive (sr^2=0.12, *P*=.001). Sows seropositive for SIV in Herd 1 (1996)

Table 3: Seroprevalence (% positive) of sows in seven herds tested twice annually[1] at a 5–6-month interval in 1996 and 1997 for porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza virus (SIV), transmissible gastroenteritis virus-porcine respiratory coronavirus (TGEV-PRCV), pseudorabies virus (PRV), *Actinobacillus pleuropneumoniae* (APP), and *Mycoplasma hyopneumoniae* (MH). All herds had a history of seropositivity to at least one of these six pathogens.

Herd	Year	Test #	PRRSV	SIV	TGEV-PRCV	PRV	APP	MH
1	1996	1	16.7	30	66.7	NC[2]	0	ND[3]
		2	6.7	46.7	100	NC	0	ND
	1997	1	1.7	55	80	NC	0	53.3
		2	0	100	39.7	NC	0	60.3
2	1996	1	6.7	93.3[4]	56.7[4]	NC	20	ND
3	1996	1	50	86.7	36.7	NC	0	ND
		2	53.3	100	0	NC	0	ND
	1997	1	77.4	27.4	61.2	NC	0	80.6
		2	76.7	71.7	6.7	NC	0	66.7
4	1997	1	0	3.3	0	NC	1.7	78.3
		2	0	76.7	0	NC	0	80
5	1997	1	20.7	82.8	1.7	NC	0	65.5
		2	83.3[4]	100[4]	5	NC	0	90
6	1997	1	83.1	44.1	93.2	0	0	69.5
		2	80	56.7	88.3	0	0	81.7
7	1997	1	96.6	93.2	91.5	1.8	3.4	47.5
		2	89.5	100	59.6	2.6	7	82.5

[1] Not all herds were tested both years. In 1996, 30 sows were sampled per group, and in 1997, 60 sows were sampled per group. When possible, samples were collected from the same sows for both annual tests. Serological tests used were HerdChek PRRS ELISA (Idexx, Westbrook, Maine), positive cutoff sample:positive (S:P) ratio 0.4; hemagglutination inhibition test for SIV (H1N1), positive cutoff titer 40; serum neutralization (Purdue strain) for TGEV-PRCV, positive cutoff titer 64; ELISA for *M hyopneumoniae*, positive cutoff S:P ratio 0.6; complement fixation test for APP (types 1, 3, 5, and 7), positive cutoff titer 8; and PRV HerdChek ELISA (Idexx), positive cutoff S:P ratio 0.7.

[2] NC: Serological test not conducted because farm was not quarantined for PRV.

[3] ND: Serological test not conducted in 1996.

[4] Sows were vaccinated.

had, on average, fewer stillborn pigs (sr^2=0.05, *P*=.034) than seronegative sows. In Herd 3 (1997), higher SIV titers were associated with more preweaning deaths (sr^2=0.07, *P*=.011), while in Herd 1 (1996), higher SIV titers were associated with fewer preweaning deaths (sr^2=0.16, *P*=.001).

Higher titers for TGEV-PRCV were associated with poorer production in some herds, and with better production in others. In Herd 6 (1997), litters from sows with higher TGEV-PRCV titers had fewer liveborn piglets than seronegative sows (sr^2=0.03, *P*=.042). In Herd 1 (1997), higher TGEV-PRCV titers were correlated with higher preweaning mortality (sr^2=0.11, *P*=.002). In Herd 2, the sows were vaccinated for TGEV, but there were more preweaning deaths in litters from sows with higher TGEV-PRCV titers (sr^2=0.17, *P*=.009). In Herds 3 (1996) and 6 (1997), sows were not vaccinated against TGEV, and higher TGEV-PRCV titers were associated with fewer preweaning deaths (Herd 3: sr^2=0.11, *P*=.048; Herd 6: sr^2=0.07, *P*=.02). The average weaning weight was lower in litters from sows with higher TGEV-PRCV titers in Herd 1 (1997) (sr^2=0.21, *P*<.001), as well as in Herd 2 (1996) (sr^2=0.21, *P*=.022). The interfarrowing interval in litters from sows with higher TGEV-PRCV titers was longer in Herd 3 (1997) (sr^2=0.10, *P*=.005) and shorter in Herd 6 (1997) (sr^2=0.10, *P*=.018).

Actinobacillus pleuropneumoniae infection was associated with more preweaning deaths in Herd 2 (1996), which had the highest prevalence of this pathogen (sr^2=0.29, *P*=.002). The number of piglets born alive decreased with an increasing PRV S:P ratio (sr^2=0.03, *P*=.045) in Herd 6 (1997), one of the two herds where PRV was present.

Mycoplasma hyopneumoniae, although prevalent in all herds, had an impact on reproductive performance only in Herd 6 (1997). Preweaning deaths increased (sr^2=0.05, *P*=.022) and average weaning weight decreased (sr^2=0.05, *P*=.027) with an increasing S:P ratio for *M hyopneumoniae* in this herd.

Discussion

In this study, the impact of endemic infection on reproductive performance was measured in university herds and in a convenience sample of commercial herds. The data on pathogen prevalence are therefore not necessarily representative of Illinois swine herds.

Although there were no apparent clinical signs due to infection with PRRSV reported in any of these herds at the time of our study, serological evidence for infection with PRRSV was associated with poorer reproductive performance in five of the seven monitored herds. This virus is known to persist in the herd for a prolonged period of time.[7] The effect of endemic infection with PRRSV on the monitored herds was of a magnitude that might have a substantial economic impact.

The magnitude of the effect of PRRSV infection on reproductive performance differed among herds. This may have been due to variations in pathogenicity of the virus and the immune status of the herds. Co-infection with other pathogens might also have influenced the effect of PRRSV on reproductive performance.

The negative association of endemic PRRSV infection with reproductive performance has been identified previously.[14] The study of Baysinger et al[14] agreed with this study in identifying fewer liveborn pigs, higher pre-weaning mortality, and a longer interfarrowing interval in herds classified by producers as subclinically infected, compared to 'negative' herds, and differed in finding no association of endemic PRRSV with stillborn births and mummies. Direct comparisons are difficult because in the present study, the individual sow was the unit of analysis, whereas Baysinger et al[14] compared mean differences between herds, and did not require serological testing to confirm a herd as 'negative' for PRRSV infection.

There are several possible explanations for the presence of a significant correlation between higher PRRSV S:P ratios and more births of stillborn piglets and mummies and a longer interfarrowing interval in Herd 4, where no sows were diagnosed as serologically positive for PRRSV. First, it is possible that the positive cutoff value for the PRRSV serological test might be set too high to detect endemic infection. Second, the diagnostic test for PRRSV might have cross-reacted with another pathogen that had a negative effect on measures of reproductive performance. Finally, the significant association between PRRSV S:P ratios and reproductive performance might have occurred by chance.

In Herd 5, a different effect of PRRSV ELISA S:P ratio could be demonstrated for vaccinated and non-vaccinated sows. Even though the better reproductive performance in the vaccinated group might have been due to chance, this seems unlikely if one looks at the relationship between PRRSV S:P ratio and number of stillborn piglets and mummies per sow. In non-vaccinated sows, higher PRRSV S:P ratios were associated with a greater number of stillborn piglets and mummies. This relationship did not occur in vaccinated sows.

With the exception of PRRSV, associations of a specific endemic infection and poorer reproductive performance could be found only in some herds. Some effects occurred in the opposite direction to that predicted, especially for preweaning deaths and weaning weight. There were repeated significant associations of TGEV-PRCV with reproductive performance, but some results were contradictory. Several different factors might be responsible for the conflicting results. First, there was a large variation in reproductive performance among individual sows, due in part to differences in genetic potential and in part to random variation.[17] Second, an endemic infection with a pathogen might have a negative impact only under certain management conditions, and the effect of a single pathogen might also be dependent on general herd health status and the strain of the organism.[19] Third, the time when each herd was infected with the monitored pathogens was unknown. A negative association of antibody levels with reproductive performance may be expected if the pathogen persists in the animal, if the infection occurs during the observation period, or if previous infection has caused morphological damage to the reproductive system, thereby having a negative impact on reproductive performance beyond the period of infection and persistence of antibodies. Immunity of the sows from a prior infection might have had a positive impact on reproductive performance: high antibody levels of the sow might have reduced preweaning deaths and increased average weaning weight through the protective effect of maternal antibodies.

In any study of the association of multiple diagnostic test results with measures of re-

productive performance, some associations might be significant by chance. The proportion of variance accounted for by some predictors was small and may not be biologically important. With the sample size of sows used in most analyses (n = approximately 40), there was a statistical power of 0.80 to detect an sr^2 value of at least 0.14 as significant at $\alpha = 0.05$ in a one-tailed test.[16] Therefore, the association of any single variable with performance should not be overinterpreted. Repeated strong associations over time in a herd identifies a problem specific to that herd. It is valid to generalize the results to other herds than the ones that participated in our study only if an association has been identified in multiple herds. In this study, this was the case only for PRSSV, ie, only PRRSV infection showed a consistent, strong association with inferior reproductive performance across herds.

Implications

- Endemic PRRSV may have a significant negative impact on reproductive performance of sows.
- Measuring differences in performance between seropositive and seronegative sows within one herd may give an estimate of the effect of infection in that herd, and may also show the benefit of vaccinating sows.
- Decisions on herd health management may thus be based on economical analysis of costs and benefit of intervention.

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