Seroprevalence of PRRS virus in the United States

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Summary—Outbreaks of porcine reproductive and respiratory syndrome (PRRS) in the United States were first reported in 1987, peaked in 1989-90, and stabilized in 1991-92. Recent data from many laboratories indicate that subclinical infections with PRRS virus may also be common. Limited serological studies have indicated that PRRS virus is widespread across the United States. The objective of this study was to determine the seroprevalence of PRRS virus in swine herds in the United States. For this purpose, we used sera collected in 1990 as part of the National Animal Health Monitoring System (NAHMS) survey of 412 randomly selected swine herds in 17 states. Blood samples from up to 10 sows and/or gilts were collected from each herd. The sera were tested for PRRS virus (VR-2332 strain) antibodies by an indirect-fluorescent antibody (IFA) test. Sera from 123 herds were also tested for antibodies to the Lelystad strain of PRRS virus. The prevalence of seropositive herds ranged from 0% in Oregon, Pennsylvania, and Tennessee to 82% in Michigan. The mean and median seroprevalence of herds within states was 33% and 29%, respectively. These data indicate that at the time of this serologic survey, PRRS virus infection was common in the United States.

Limited serologic studies have indicated that PRRS virus is widely disseminated among swine herds in the United States. In a survey done at the National Veterinary Services Laboratories, Ames, Iowa, the prevalence of antibody to PRRS virus in adult swine at the time of slaughter ranged from 4.6% in Kansas to 20% in Iowa. Herds were found to be seropositive for this virus as early as 1985 in Iowa and 1986 in Minnesota. However, the significance and impact of this disease on the United States swine population remains unclear because the seroprevalence of PRRS virus in all the hog-producing regions of the country has not been determined. The objective of this study was to determine the seroprevalence of PRRS virus in swine herds in the United States. This information can be used to estimate the impact of this disease on productivity and can also be used as a reference for past and future studies that analyze the epidemiology of PRRS virus.

Materials and methods

Serum samples
In 1990, the National Animal Health Monitoring System (NAHMS) conducted a survey in 17 states of the United States and selected 412 swine herds at random. Blood samples were collected from up to 10 sows and gilts from each herd (Table 1). The sampling distribution was as follows:
- approximately 58% of the herds had 10 samples each;
- 11% had 9 samples;
- 17% had 6-8 samples; and
- 14% had 1-5 samples.

Sera were frozen at -20°C until used. We tested a total of 3372 serum samples for antibodies against the VR-2332 strain of PRRS virus. Additionally, we tested sera from 64 herds that

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<th>Number of samples per herd</th>
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<tr>
<td>10</td>
<td>241 (58.5)</td>
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<td>8</td>
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<td>2</td>
<td>12 (2.9)</td>
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<tr>
<td>1</td>
<td>8 (1.9)</td>
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<tr>
<td><strong>Total</strong></td>
<td><strong>412 (100)</strong></td>
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were found negative and 59 herds that were found positive for antibodies to the VR-2332 strain of PRRS virus for antibodies to the Lelystad strain.

**Antigen**

We infected 3-day-old CL 2621 cell (Boehringer Ingelheim Animal Health, St. Joseph, Missouri) monolayers prepared in 96-well microtiter plates with 1000 TCID$_{50}$ of ATCC VR-2332 or the Lelystad strain of PRRS virus per well. After incubating at 37°C for 48-72 hours, we fixed the infected cells with cold absolute ethanol and stored the plates at -20°C until used.

**Serologic test**

We performed the IFA test as previously described.$^{1,7}$ We prepared four-fold dilutions of sera in phosphate-buffered saline (PBS; pH 7.2) with a starting dilution of 1:20, and transferred 100 μl of each dilution to virus-infected CL 2621 cells. After the plates were incubated for 45 minutes at 37°C, we washed them with PBS. We added 50 μl of an optimum dilution of a fluorescein-conjugated anti-porcine IgG (ICN Biomedicals, Costa Mesa, California) to each well. After incubating for 45 minutes, we washed the plates and observed them under a fluorescent microscope for specific cytoplasmic/perinuclear fluorescence.

**Analysis of data**

We considered a sample to be positive if we observed specific fluorescence at 1:20 dilution or above. We considered a herd to be positive if at least one animal was seropositive. We then calculated the seroprevalence of PRRS virus within the seropositive herds and per state. We used Chi-square analysis to test for a trend in the association between parity and likelihood of being seropositive in 74 herds for which parity was recorded and where at least one animal was seropositive. Using Chi-square analysis, we also compared the association between the number of samples collected per herd (1-5, 6-8, 9, 10) and the likelihood of detecting at least one seropositive animal.

**Results**

Of the 412 swine herds tested, 147 (36%) had at least one animal seropositive for the VR-2332 strain of PRRS virus. There was a significant association between seroprevalence and the number of samples collected per herd. We detected fewer seropositive herds than expected among those with 1-5 samples tested ($P<.05$; Fig 1). Of the 147 seropositive herds:

- 25% had a prevalence of $<14$%;
- 50% had a prevalence of $<30$%; and
- 75% had a prevalence $<43$% (Fig 2).

The prevalence of seropositive herds ranged from 0% in Oregon, Pennsylvania, and Tennessee to 82% in Michigan (Table 2). The mean and median seroprevalence of herds among states was 33% and 29%, respectively. There was no significant association between parity of a sow and the likelihood of her being found seropositive (Fig 3).

Of the 123 herds tested for antibodies to the Lelystad strain, 92 (74.8%) were negative (Table 3). When compared with the results obtained for VR-2332:
60 herds were negative for both strains; 32 herds were positive for VR-2332 but negative for Lelystad; 4 herds were positive only for antibodies against the Lelystad strain; and 27 herds were positive for both strains (Table 3).

Discussion

The data indicate that although the seroprevalence of PRRS varies considerably across the United States, overall, the virus has infected herds throughout the country. In the midwest, which is relatively dense with swine farms, the prevalence is moderately high, whereas evidence of PRRS virus was not detected in several states that have fewer swine herds.

We believe that the actual prevalence of PRRS-seropositive herds is higher than that reported in this study. This is due, in part, to sampling error. Because we did not sample the entire herd, the likelihood of detecting all seropositive herds is less than 100%. For example, if only five individuals were randomly selected from a herd that had a seroprevalence of 20%, there would be a 33% probability that we would have failed to detect PRRS virus in this herd. Since all animals in a herd were not tested, it is very likely that we inappropriately deemed some herds to be negative.

It appears that herds with a low seroprevalence among sows and gilts are extremely common. In this study, 50% of the seropositive herds had less than 30% seroprevalence. Loula reported similar findings in a survey of seven selected herds in Minnesota. Perhaps PRRS virus is not spreading within many herds and seroprevalence is declining as seropositive sows are replaced by seronegative gilts. Or perhaps antibody is not persisting in previously infected pigs. Stevenson, et al. described one such herd, in which the seroprevalence of seropositive sows was as low as 15%, with sows converting to a seronegative status within 10 weeks after infection. Because parity was not associated with the likelihood that the sows were seropositive, antibody decay to undetectable amounts after infection appears to be common.

We may also be underestimating the seroprevalence of PRRS because of the antigenic diversity among PRRS isolates. The likelihood of detecting PRRS-positive herds was increased when sera were tested for both ATCC VR-2332 and Lelystad strains of the virus. The estimated seroprevalence may have been further increased if we had used other PRRS virus isolates that are serologically distinct from the ATCC VR-2332 and Lelystad strains.

The relatively high seroprevalence detected in this study indicates that there was a higher incidence of infection of swine herds than is indicated by reports of clinical outbreaks. While clinical evaluation is subjective, the high seroprevalence indicates that subclinical infection with PRRS virus may be common in the swine industry.

Acknowledgements

This is the first reported use of sera collected from a random sample of swine herds in the United States. We acknowledge the assistance of Drs. Scott Hurd at NAHMS and Merwin Frey at National Veterinary Services Laboratories for giving us
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References


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**Expanding your risk limits**

To increase your tolerance for change and risk:

- Rearrange your office furniture. Take a new route to work.
- Take a course, class, or seminar that will help you learn a new skill.
- Upgrade your computer skills by learning how to use a new piece of software. If you’re not using a computer, sign up for a class and learn how to use one.
- Pick four things you’ve always wanted to try. Schedule one each quarter this year.
- Discuss with colleagues your assumptions about your job, co-workers, etc. Listen to what they say and challenge those assumptions.
- Improve your health. Diet, work out, stop smoking.
- Reward yourself when you try something new. But reward risk, not success. Success is its own reward.
- Try skiing, softball, basketball, or some other sport. The idea is to expand yourself and try new things.
- Pick out one thing you’d like to change in your life and go after it. Write it down, list the steps you need to take to accomplish it, set deadlines, and take the first step.