

## Concurrent respiratory infections in 221 cases of PRRS virus pneumonia: 1992–1994

David H. Zeman, DVM, PhD, Diplomate ACVP

**Summary:** Porcine reproductive and respiratory syndrome virus (PRRSV) pneumonia was identified in 221 routinely submitted cases over a 35-month period at a north central United States veterinary diagnostic laboratory. Age distributions were fairly evenly represented among suckling, nursery, and grower-finisher production phases. Interstitial pneumonia and bronchopneumonia were frequently described histopathological lesions. Concurrent pulmonary bacterial infections were identified in 58% of the cases. Most commonly, these were *Pasteurella multocida*, *Streptococcus suis*, *Haemophilus parasuis*, and *Salmonella*. Concurrent swine influenza virus infection was detected in only four cases. There were no concurrent pulmonary pathogens in 39.8% of the study cases.

**Keywords:** swine, porcine reproductive and respiratory syndrome (PRRS), diagnosis

The porcine reproductive and respiratory syndrome virus (PRRSV) was first identified in the United States in late 1991.<sup>1,2</sup> Thus, only a relatively short time has passed since veterinary diagnostic labs began routinely testing for the virus in diagnostic submissions. For example, the South Dakota Veterinary Diagnostic Laboratory began regularly employing PRRSV diagnostic procedures in February 1992.<sup>3</sup> Now that several months of data have accumulated, it is possible to retrospectively evaluate large numbers of PRRS cases and share that information with practitioners and researchers. The purpose of this paper is to briefly describe 221 field cases of PRRSV pneumonia that were submitted for laboratory evaluation during an approximately 3-year period.

DZ: Animal Disease Research and Diagnostic Laboratory, South Dakota State University, Brookings, South Dakota, 57007

These cases represent the combined work of numerous people at the South Dakota Animal Disease Research and Diagnostic Laboratory, including pathologists D. Johnson, D. Nelson, M. Yaeger, R. Neiger, and D. Miskimins; bacteriology supervisor C. Gates; diagnostic virology supervisor P. Leslie-Steen; and research virologists D. Benfield and E. Nelson. All efforts of these and other lab personnel are gratefully acknowledged.

### Diagnostic notes are not peer reviewed

*Editor's Note:* This is the final "Diagnostic Notes" article in a series that has emphasized PRRS diagnostics. For more information regarding the subject, see the Diagnostic Notes in the January/February and March/April 1996 issues of Swine Health and Production.

### Materials and methods

The cases studied were routinely submitted to the South Dakota Animal Disease Research and Diagnostic Laboratory at Brookings, South Dakota between February 1, 1992 and December 31, 1994 (35 months). In this laboratory, a 'case' is defined as one to three animals or their tissues, submitted on the same day from the same farm with the same clinical syndrome. The selected time period represented the onset of routine diagnostic testing for PRRSV in this particular lab, and the cut-off date was randomly selected as the end of the calendar year 1994. All cases were submitted through referring veterinarians, and the vast majority originated from eastern South Dakota, southwest Minnesota, northwest Iowa, and northeast Nebraska.

Only laboratory-confirmed PRRSV pneumonia cases were selected: pigs with pneumonia or pneumonic lung tissues from field necropsies were the sources of the study lungs. Laboratory-confirmed PRRSV pneumonia was defined as:

- lung in which PRRSV was demonstrated by the fluorescent antibody test on fresh cryostat lung sections, or
- lung in which PRRSV was isolated on cell lines or alveolar macrophage culture systems.

Cases that only had serological indication of PRRSV infection or abortion case materials were not included in the study.

History, signalment, and clinical signs were taken from the submission form. This information was provided by the referring veterinarian or was provided by the owner when the owner accompanied the animals/tissues to the diagnostic lab. If the history did not clearly indicate the production phase (i.e., suckling, nursery, or grower-finisher), the following assumptions were made:

- pigs  $\leq 15$  lb or  $\leq 21$  days old were designated suckling pigs;
- pigs  $> 15$  lb but  $< 40$  lb or  $> 21$  days but  $< 60$  days old were designated nursery pigs; and
- pigs  $\geq 40$  lb or  $\geq 60$  days old were designated grower-finisher pigs.

Only 211 cases could be assigned to a production phase group, because there was no signalment information on 10 submission forms.

In this laboratory, cases are assigned on a daily basis to one of six veterinary pathologists. The pathologist studies the submission form, evaluates the tissues or performs the necropsy, and orders appropriate laboratory testing. Bacteriological and virological results are later correlated with histopathological findings and the case is summarized by the pathologist. Fresh tissues are kept for a period, permitting additional testing when necessary.

**Table 1**

## FA and VI detection of PRRSV in tested pigs

Virus detection result	Number of pigs
VI+, FA+	130
VI-, FA+	81
VI+, FA-	10
VI-, FA-	0

**Results**

During the 35-month study period, 221 cases of PRRSV pneumonia were identified. The primary owner's complaint was respiratory disease in 87.8% of the cases. Both respiratory and reproductive diseases concurrently were the primary owner's complaint in 8.1% of the cases. The remaining 4.1% (nine cases) had various other primary complaints, such as enteritis.

The pigs were fairly evenly distributed among production groups. The grower-finisher group was the most represented group at 38.4%. The suckling and nursery groups were evenly represented at 30.8%.

Porcine reproductive and respiratory syndrome virus was detected by fluorescent antibody (FA) testing of fresh lung in 95.4% of the 221 cases (Table 1). Virus isolation (VI) procedures yielded PRRSV in 63.3% of the total cases. The virus was detected by both FA and VI procedures in 130 (58.8%) of the total 221 cases.

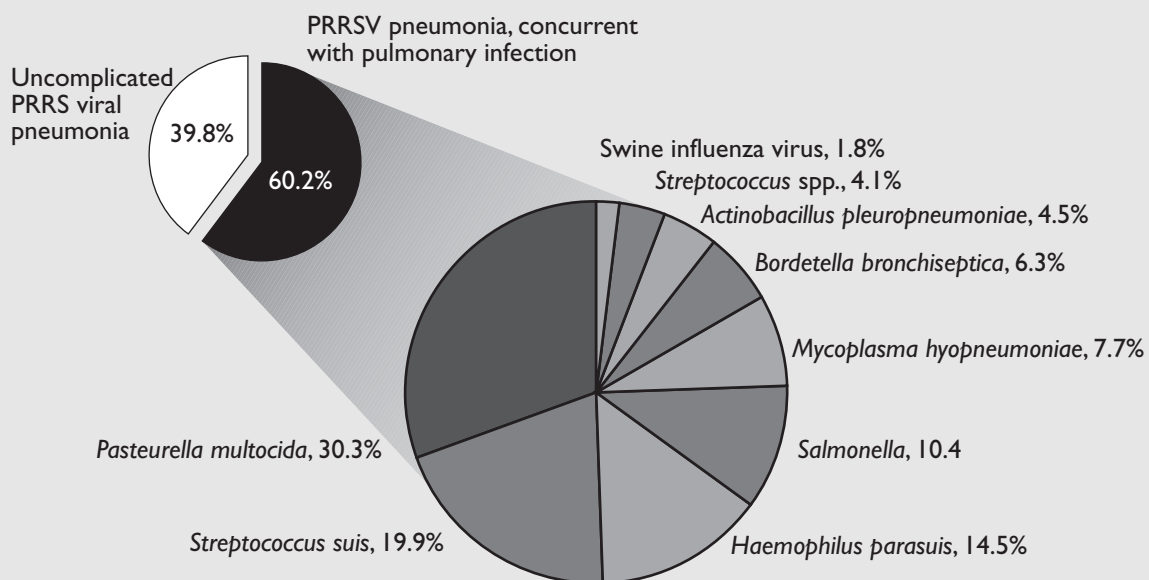
Concurrent pulmonary bacterial infections were identified in 58% of the total cases (Figure 1). In many cases, multiple bacterial infections were identified. Four cases had concurrent swine influenza virus infection, bringing the total concurrent pulmonary bacterial and/or viral infection rate up to 60.2%. Thus, 39.8% of the total cases represented uncomplicated PRRS viral pneumonia.

A variety of histopathological lesions were described. Interstitial pneumonia was most frequently noted, in 66.1% of the total cases. Bronchopneumonia was the next most frequent histological lesion, being noted in 42.1% of the cases. Also common were bronchiointerstitial pneumonia (14.0%) and pleuritis (12.2%). Most lungs had more than one histopathological lesion.

**Discussion**

Porcine reproductive and respiratory syndrome virus pneumonia is the most overwhelming cause of viral pneumonia in swine within our laboratory service region. Problems with PRRSV pneumonia in nursery or grower-finisher pigs are often discussed among practitioners and researchers.<sup>4</sup> However, in this study the distribution of cases was fairly even among production groups and there were an equal number of cases identified in suckling pigs compared to nursery pigs. This may be due in part to the fact that our lab has for some time promoted the submission of weak neonatal piglets, along with other samples, when investigating abortion storms.

The FA test applied to fresh lung tissue has been a rapid, reliable, and economical test for PRRSV detection. If the lung shows consolidation, we encourage the submission of both consolidated lung and nonconsolidated lung for laboratory examination (fresh and formalinized). We prefer to use the nonconsolidated lung for FA and VI testing, thus avoiding problems with bacterial contamination and inflammatory exudates. We then use the consolidated lung for bacterial cultures or other testing. The virus was isolated by culturing in 63.3% of the cases. Previous studies have shown that if tissues, including lung, are not kept properly cooled en route to the laboratory, the virus can easily die.<sup>5</sup> Sending serum samples along with the tissue samples could improve virus isolation success, as virus viability appears to be enhanced in serum. However, proper packaging and adequate coolant is still essential to overall diagnostic success.

**Figure 1**

Prevalence of concurrent pulmonary pathogens in tested pigs

Concurrent pulmonary infections, predominantly bacterial, were identified in 60.2% of the PRRS pneumonia cases. There is much interest in the role that these concurrent infections play in the morbidity and mortality of PRRSV-infected swine. Practitioners generally think that more bacterial infections are observed in PRRSV-infected herds. Research reports have been variable regarding secondary infections. Some researchers have shown enhanced bacterial disease when PRRSV-infected pigs were subsequently challenged with *Haemophilus parasuis*.<sup>6</sup> However, a recent large experiment using specific-pathogen-free pigs and the NEB-1 strain of PRRSV failed to demonstrate enhanced bacterial infections with *Streptococcus suis*, *H. parasuis*, *Pasteurella multocida*, or *Salmonella choleraesuis*.<sup>7</sup>

These apparent discrepancies regarding enhanced bacterial pneumonia in PRRSV-infected pigs could be explained by viral or bacterial strain differences, by environmental stress differences, or by failure to reproduce real-world scenarios experimentally. Conversely, it might also mean that the virus simply does not potentiate bacterial bronchopneumonia and that the detected co-infections are purely coincidental independent infections, and are not related to one another. In this study, 58% of our cases had concurrent bacterial pneumonia. Whether these concurrent infections are directly or coincidentally related, practitioners will have to address bacterial pneumonia in their therapeutic strategy for a high percentage of PRRS pneumonia cases. Future studies will likely continue to clarify the interaction between PRRSV and bacterial pathogens.

Concurrent pulmonary infections also seem to explain why both interstitial pneumonia and bronchopneumonia were common histopatho-

logical lesions in these cases. Bacterial co-infections would readily explain purulent exudation in airways and alveoli (bronchopneumonia), as well as the occasional purulent pleuritis or pericarditis. Significant interstitial pneumonia with minimal inflammatory exudation and relatively normal airway epithelium are still the hallmark histological findings with PRRSV pneumonia.

## References

1. Collins JE, Benfield DA, Christianson WT, et al: Isolation of swine infertility and respiratory syndrome virus (isolate ATCC VR-2332) in North America and experimental reproduction of the disease in gnotobiotic pigs. *J Vet Diagn Invest.* 1992; 4:117–126.
2. Benfield DA, Nelson E, Collins JE, et al: Characterization of swine infertility and respiratory syndrome (SIRS) virus (isolate ATCC VR-2332). *J Vet Diagn Invest.* 1992; 4:127–133.
3. Zeman D, Neiger R, Yaeger M, et al: Laboratory investigation of PRRS virus infection in three swine herds. *J Vet Diagn Invest.* 1993; 5:522–528.
4. Stevenson GW, Van Alstine WG, Kanitz CL: Characterization of infection with endemic porcine reproductive and respiratory syndrome virus in a swine herd. *JAVMA.* 1994; 204:1938–1942.
5. Van Alstine WG, Kanitz CL, Stevenson GW: Time and temperature survivability of PRRS virus in serum and tissues. *J Vet Diagn Invest.* 1993; 5:621–622.
6. Solano G, Segales J, Pijoan C: Interaction between porcine reproductive and respiratory syndrome virus (PRRSv) and *Haemophilus parasuis*. *Proc 2nd Intl Symp PRRS*, Copenhagen, Denmark. 1995: 21.
7. Cooper VL, Doster AR, Hesse NB, et al: Porcine reproductive and respiratory syndrome: NEB-1 PRRSV infection did not potentiate bacterial pathogens. *J Vet Diagn Invest.* 1993; 313–320.

