PRRS SUMMARY AND UPDATE

Introduction

Porcine reproductive and respiratory syndrome (PRRS) was first identified as a clinical entity in the United States in 1987, but there is serological evidence that it was in Canada in 1979, in the United States and Korea in 1985, and in Japan and Germany in 1988. We don’t know where the virus came from, but some have suggested a wildlife reservoir, possibly feral pigs. Interestingly, although the Lelystad strain (LV) and the U.S. strain (VR-2332) are different, they are similar enough to suggest that they had a common ancestor but evolved differently as they adapted to their own niches. If we accept that they had a common ancestor, then we are left with the possibility that source viruses still exist and contribute to the magnitude of the current strain variation. A large variation in viral genomic possibilities contributes to the development of new future strains and complicates eradication efforts.

Although PRRS is endemic in most countries, there are still herds in all countries that remain negative. The prevalence of positive herds is very difficult to estimate because the modified-live vaccines produce serum antibodies that are indistinguishable from those produced by the “wild” virus and because those vaccine viruses can infect naïve pigs. In addition, no statistically valid random samplings of the populations of interest have been undertaken. Our best estimate of prevalence is that, of those farms not using modified-live vaccine, 59 percent are positive to PRRS.

Characteristics

Infection

The PRRS virus is highly infectious: 10 or fewer particles are capable of establishing an infection when exposed by the intranasal route. Other routes (oral, vaginal, or eye) require higher doses, usually of the order of $10^{3}$ to $10^{5}$ particles. Breeding females can be infected by both undiluted semen (Yeager et al., 1993) and extended semen (Gradil et al., 1996) that carries the virus.

Shedding

Post-infection, pigs will shed the virus for extended periods in saliva (42 days), semen (43-92 days) and mammary secretions. The results with regard to fecal shedding are mixed: Wills et al. (1997) reported no infectious virus in feces up to 42 days post-infection. By contrast, Yoon et al. (1993) reported extensive fecal shedding over 35 days, while Rossow et al. (1994) found only intermittent shedding. Regardless of the origin, infected pigs shed the virus in copious amounts and quickly contaminate their surroundings.

Virus survival

If conditions are right (about 4°C, pH 7.5) the virus can survive from days to weeks. However, it is very susceptible to adverse conditions, especially drying, and will die within hours as conditions change from optimum. On the usual pig-associated fomites (plastic, steel, wood, straw, clothing, slurry, etc.) and at normal environmental temperatures (25-27°C), PRRS virus survives less than a day, but it can survive in water for up to 11 days. Thus, normal cleanup procedures with disinfection and drying will kill the virus.

Carriers

Persistence is a marked characteristic of PRRS viral infections, as some pigs harbor the virus for at least 157 days and possibly longer. In contrast, it is difficult to infect other pigs unless they are in very close contact—that is, the disease is not highly contagious. The low infectivity of the virus has ensured the survival of the virus in a herd. Viral survival depends on a proportion of the population being persistent with occasional shedding, while the rest is susceptible. Dee suggests that these subpopulations are important for the long-term survival of the infection in herds (Dee et al., 1993).

Hosts

Mallard ducks are susceptible to PRRS virus and can excrete it in their feces for up to 39 days post-experimental infection (Zimmerman et al., 1997). In addition, they
spread it among themselves and are capable of infecting pigs. Although mallards may not be significant vectors in the field, it does indicate that birds can carry the virus and infect pigs. But again, the significance of this in field outbreaks is still unknown. Rodents are not susceptible to the PRRS virus (Hooper et al., 1994).

Transmission

Researchers find it difficult to transmit the disease among pigs, which is surprising, given the widespread distribution of the disease. Experimentally, Torremorell et al. (1997) were able to infect only 2 of 8 pen-mates of acutely infected pigs, and Wills et al. (1997) found it much easier to infect pen-mates than pigs separated by 1 meter and less. There is some evidence from lactate dehydrogenase-elevating virus (LDV) in mice that suggests pigs may be infected via inoculation of skin lesions that occurs when the animals are mixed and fighting breaks out. But this theory has yet to be proven for swine.

The most commonly observed way for the disease to be transmitted between herds is by the use of contaminated semen or by the introduction of infected (carrier) animals. Transmission by air is possible, but it probably does not play as significant a role as it does in the transmission of other diseases, such as foot-and-mouth disease.

Le Potier et al. (1997) estimated which sources are most likely to infect herds in France:

- 56% infected pigs
- 21% indirect spread (trucks, boots, clothing, etc.)
- 20% infected semen
- 3% unidentified

These are likely to serve as our best estimates for other countries, too.

Once herds are infected, the virus tends to circulate forever and is very hard to eliminate. We don’t know why the virus is so persistent, but it is probably associated with carrier pigs (discussed above), the birth of susceptible neonates, and the introduction of naïve pigs (gilts and boars). Unfortunately, maternal antibodies do not provide adequate protection against PRRS, and any protection they do provide is short-lived. Consequently, when pigs are mixed at weaning, up to 100 percent become infected.

But exceptions to this rule do exist. Le Potier et al. (1997) reported that, for one farm, in-herd-bred replacement gilts did not seroconvert until post-mating.

Prevention and control

The best advice to any farmer trying to exclude PRRS is, Stop bringing infected pigs into the herd! As discussed above, infected pigs are the primary cause of most initial PRRS outbreaks.

Most modern herds bring female replacements into sow herds to replenish the breeding stock. If live, negative animals are introduced into a negative herd, they should be isolated for at least 40 days and blood-tested at arrival and 14 days post-arrival.

If the supply herd is negative and maintains that status, it is acceptable to enter the PRRS-negative replacements into PRRS-positive sow herds after the negative ones have been exposed to, and recovered from, an intentional infection of the sow-farm strain. This is known as acclimation. Acclimation serves to infect replacements in isolation at least 90 days before the replacements are introduced into the positive sow herd. The extended isolation allows time for recovery and development of immunity. PRRS infections result in a rather slow onset of immunity when compared to other diseases of the pig.

If the supply herd is positive, but maintains the same infectious PRRS virus strain as the receiving herd, it is acceptable to enter previously infected PRRS-positive replacements. The most risky situation is when the strain dominant on the supplier farm is not the same as the strain on the receiving farm. When producers experience difficulty attempting to match strains of supplier and receiving-sow farm, they often close their herds or production pyramids. Thus, genetic improvement is slowed as new genetic material enters only in the form of semen from a negative boar. Each herd manager should have his or her veterinarian discuss the herd’s status and arrive at the optimum procedures for introducing new genes.

Control

To be successful, any PRRS-sow-herd control strategy must decrease replication of the virus within a population of sows and boars. This will prevent infection of the neonates. Unfortunately, some herds seem to contain sub-populations of naïve animals as well as infectious animals. The virus replicates when the animals come into contact with each other, and the infection is maintained within the herd (Dee et al., 1993).

To eliminate these sub-populations:

1. Identify infectious and naïve animals.
2. Implement replacement acclimation procedures to reduce the population at risk. Replacements are often naïve on entry.
3. If need be, stabilize the breeding herd. In acute outbreaks, it may be necessary to physically spread the virus and either stop replacement entry for three or four months or infect a four-month supply of replacement animals. The outcome of stabilization assures that sow-to-pig infections are minimized.
4. Stabilize nursery and finishing herds after the sow herds are under control. Continuous-flow facilities may need to be depopulated and repopulated.

Vaccinations

Generally, modified-live vaccines are more efficacious than killed vaccines. However, modified-live vaccines are able to
replicate, spread through populations, and mutate to a more fit status. Consequently, they have compounded the problem.

Boehringer Ingelheim Vetmedica, Inc., sells Resp PRRS/Repro<sup>®</sup> (called INGELVAC<sup>®</sup> PRRS-MLV in Europe). In 1994 it was approved for use in 3- to 18-week-old pigs. In 1996 approval was given for administration to non-pregnant gilts and sows.

Schering-Plough’s product, Prime Pac<sup>®</sup>PRRS, was recently withdrawn from the market for patent infringement.

Intervet sells PRRomiSe<sup>®</sup> PRRS vaccine. PRRomiSe is a killed-virus product and an effective alternative to other PRRS vaccines. It has been sold by Bayer since 1997.

In deciding whether to use a modified-live vaccine you should know:

1. The vaccine viruses can cross the placenta and cause congenital infection.
2. They can be transmitted just like the “wild” virus.
3. They can persist for weeks or months.
4. They can be shed in boars’ semen and by infected breeding females.
5. Induced immunity is slow to develop.
6. They don’t always work.

References


—W. E. Morgan Morrow
—John Roberts

### CALENDAR OF EVENTS

#### November

2 Healthy Hog Seminar
Sampson Agri-Exposition Center
Clinton, N.C.

14 Carolina Swine Nutrition Conference
Sheraton Imperial Hotel
Research Triangle Park, N.C.

20 Wilson Regional Pork Conference
Wilson County Extension Center
Wilson, N.C.

29 Piedmont Regional Pork Conference
Person County Office Building
Roxboro, N.C.

#### December

6-7 National Swine Improvement Federation Annual Meeting
St. Louis, Mo.

#### January, 2002

8-9 North Carolina Pork Conference
New Bern Riverfront Convention Center
New Bern, N.C.
### 2001 CAROLINA SWINE NUTRITION CONFERENCE
Sheraton Imperial Hotel, Research Triangle Park, NC

**Tuesday, November 13**

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<tr>
<th>Time</th>
<th>Event</th>
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<tr>
<td>6:00 - 7:30 pm</td>
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**Wednesday, November 14**

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<tr>
<td>8:00 am</td>
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**Morning Session - Auditorium**  
*Dr. David Funderburke - Moderator*

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<tr>
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<th>Event</th>
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<tbody>
<tr>
<td>9:00 am</td>
<td>Welcome</td>
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<tr>
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<td>Larry Gunter</td>
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<tr>
<td>9:10 - 9:55 am</td>
<td>Effects of Nutrition on Immunity and Disease Resistance</td>
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<td>Dr. Kirk C. Klasing</td>
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<td>9:55 - 10:40 am</td>
<td>Amino Acid Requirements of the Piglet: Implications for Early Weaning</td>
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<td>Dr. Ronald O. Ball</td>
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<td>10:40 - 11:15 am</td>
<td>Coffee Break</td>
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<td>11:15 -</td>
<td>Dietary Influencers of Pork Quality and</td>
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<td>12:00 noon</td>
<td>Practical Solutions to Quality Problems</td>
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<td>Dr. Jeff Hansen</td>
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<td>12:00 noon - 1:00 pm</td>
<td>Luncheon</td>
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**Afternoon Session - Auditorium**  
*Dr. Jack Odle - Moderator*

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<tr>
<td>1:00 pm</td>
<td>Enzymes in Swine Nutrition</td>
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<td>Dr. J. Scott Radcliffe</td>
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<td>1:45 - 2:30 pm</td>
<td>Managing Water Quality and Supply to Weanling Pigs and Sows</td>
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<td>Dr. John Patience</td>
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<td>2:30 - 3:15 pm</td>
<td>Coffee Break</td>
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<td>3:15 pm - 4:00 pm</td>
<td>Sow and Young Pig Nutrition and Management: Challenging Present Practice</td>
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<td>Dr. R. Dean Boyd</td>
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<td>4:00 - 4:30 pm</td>
<td>Questions - Adjourn</td>
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