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Strategies to control PRRS: A summary of field and research experiences

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Abstract

Various methods for the control of PRRS virus have been published. The technology of nursery depopulation (ND) appears to effectively control the spread of virus between members of endemically infected populations. ND consists of a strategic adjustment in pig flow based on the presence of specific serologic patterns as detected by the indirect fluorescent antibody test. This pattern indicates a low seroprevalence of antibodies detected in the breeding herd and recently weaned piglets (< 10%), in contrast to a high (> 50%) seroprevalence in 8 to 10 week old piglets. ND has been carried out on swine farms in the US and results indicate improvements in nursery piglet growth rate and mortality levels. Three examples are provided in the following text. Recently a modified live virus vaccine (RespPRRS, NOBL Laboratories/Boehringer Ingleheim) has become commercially available. It is currently approved for use in piglets from 3 to 18 weeks of age; however, potential for the use in adult animals is currently under investigation. © 1997 Elsevier Science B.V.

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1. Introduction

Porcine reproductive and respiratory syndrome (PRRS) is a recently recognized disease of swine (Keffaber et al., 1992; Loula, 1991). The etiologic agent is a member of the viral group Arteriviridae (Wensvoort et al., 1991). Other members of this group include equine viral arteritis virus, lactate dehydrogenase elevating virus of mice and simian hemorrhagic fever virus (Meulenberg et al., 1995). Infected herds may experience severe reproductive losses and/or increased levels of post weaning pneumonia.

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with poor growth. The reproductive phase typically lasts for 2–3 months, however post weaning problems often become endemic. A reduction in growth rate (50–85%), an increase in the percentage of unmarketable (cull) pigs (10–30%) and elevated post weaning mortality (10–25%) can be seen (Polson et al., 1994). Diagnostic findings indicate a high level of pneumonia, with the isolation of PRRS virus in complex with a wide variety of other microbial agents. Bacterial isolates may include *Streptococcus suis*, *Haemophilus parasuis*, *Salmonella cholerae-suis*, *Actinobacillus pleuropneumoniae*, as well as *Mycoplasma hyopneumoniae* and *Pasteurella multocida*. Viral agents commonly involved with the respiratory disease complex include swine influenza virus (SIV) and porcine respiratory coronavirus (PRCV) (Halbur, 1995). Affected pigs rarely respond to high levels of medication and converting facilities to an all in/all out system has not been successful in solving the problem. The economic impact of the post weaning form of the disease can be severe. Losses have been estimated at $18.21 per head due to poor performance, a reduction in marketable pigs and elevated medication costs (Polson et al., 1994). The purpose of this paper is to describe the development and implementation of a cost effective control measure entitled nursery depopulation (ND). Field experiences with ND will be discussed, as well as some observations on the use of a commercially available PRRS vaccine.

2. Protocol development / pilot studies

The technology of nursery depopulation consists of interrupting the spread of virus between groups of pigs using calculated adjustments in pig flow. The following study describes the development and initial implementation of ND on 3 commercial swine operations (Dee and Joo, 1994).

3. Materials and methods

3.1. Farm history

Three farms were selected on the basis of the owners’ willingness to participate. Farm 1, with 500 sows, and farm 2, with 300 sows were commercial herds, and farm 3 with 275 sows was a seedstock herd. Farm 3 was producing pigs on several sites and using a system of modified medicated early weaning. On farms 1 and 2 the nursery and finishing buildings were on the same site as the breeding—farrowing building, but on farm 3 they were on different sites.

All three farms had experienced an outbreak of PRRS in late 1990, with similar reproductive problems resulting from the infection. Stillbirths and mummified fetuses increased from normal levels of 6–8% and 0.5–2%, respectively, up to 30–45% in total. During the outbreak, preweaning mortality averaged 25 to 50% in contrast to average values of 8–12% over the previous 5 years. While reproductive problems eventually subsided within two to three months, all three farms began to develop endemic nursery problems.
Farm 1 had an increase in nursery mortality from 2% up to 10% and post mortem findings indicated an increased level of pneumonia, pericarditis and polyserositis. *Haemophilus parasuis* and *Streptococcus suis* were the primary pathogens isolated. On farm 2, the nursery mortality increased from 2 to 15%. *Salmonella cholerae-suis* and *Streptococcus suis* were consistently isolated from piglets eight to nine weeks old. Farm 3 had an increase in mortality from 2 to 4% primarily due to *Streptococcus suis* septicemia. Since the farm was already using modified medicated early weaning, the nursery problems were not as severe; however, erratic outbreaks of streptococcal septicemia continued to occur. PRRS virus had been isolated from serum and tissue samples from all three farms.

**3.2. Initial serology**

**3.2.1. Farms 1 and 2**

In order to establish the prevalence of PRRS virus antibodies before the nursery was depopulated, 10 blood samples were collected from each of the following stages of production: the breeding herd, weaned pigs (18 to 22 days old), eight-to-nine-week-old nursery pigs and five-to-six-month-old finishing pigs. All the sera were tested for antibodies to PRRS virus by IFA test as previously described (Yoon et al., 1992).

**3.2.2. Farm 3**

Owing to the early weaning program, an offsite nursery and grower building was already established. The nursery facility contained four rooms, each holding 200 pigs or approximately two weeks of production. At the time of the testing, the room numbers and corresponding ages of the pigs were as follows: room 1 (two to three weeks), room 2 (four to five weeks), room 3 (six to seven weeks) and room 4 (eight to nine weeks). The grower building contained two rooms each with 200 pigs. The pigs were in this facility for four weeks and then moved to finishing barns on different sites. Ten pigs from each room were tested for antibody to the PRRS virus.

**3.3. Eradication protocol**

**3.3.1. Farms 1 and 2**

All the nursery pigs were either simultaneously moved to contract finishing facilities or sold as feeder pigs. The nurseries were completely emptied, cleaned three times with hot (> 95°C) water and disinfected with phenol and formaldehyde-based products. The slurry pits were pumped out after each cleaning. The facilities were empty for a total of 14 days, during which all the pigs were weaned to rented offsite nurseries. After the 14 day period had elapsed, conventional flow of pigs into cleaned facilities resumed.

**3.3.2. Farm 3**

Due to the early weaning age practiced on this farm (12 to 14 days old), offsite facilities capable of handling the young pigs could not be found. Therefore, only rooms which contained pigs of six to nine weeks old (rooms 3 and 4) were emptied. In addition, the entire grower building was emptied. The rooms were cleaned as previously
described for farms 1 and 2, but owing to the weaning schedule, rooms 3 and 4 could be left empty for only four and 11 days, respectively, after cleaning. The grower building remained empty for 14 days.

3.4. Follow-up serology

When the first group of pigs that were weaned into the cleaned facilities reached eight to nine weeks of age, 30 pigs were randomly selected for blood testing. This procedure was repeated monthly for three consecutive months and again three months later. If all the samples were negative for PRRS virus antibody, testing was terminated. Pig CHAMP data were examined before and after the repopulation and the percentage of mortality and average daily weight gain data were analyzed.

3.5. Results

The serological profiles prior to ND were similar on each farm. All samples collected from breeding animals were IFA negative. All the pigs tested within one week after weaning (25 to 29 days old) were negative, but 80 to 100% of the pigs tested at eight to nine weeks old had IFA titers ranging from 1:64–1:1024. The seroprevalence in finishing pigs ranged from 25 to 50% and titers ranged from 1:16–1:64. The results from farm 3 indicated that only nursery room 4 and the grower building contained IFA positive pigs: all the pigs tested in nursery rooms 1, 2 and 3 were IFA negative.

After the repopulation of the nurseries, no IFA positive animals were detected in the nurseries on any of the farms. Samples collected from the grower building on farm 3 were also negative. All samples analyzed throughout the six month testing period were negative as well. Improvements in nursery pig performance were recorded in all 3 farms. Growth rates improved from 0.14 to 0.36 kg in farm 1 and from 0.12 to 0.34 kg in farm 2. Mortality levels averaged 0.28 and 1.0%, respectively. Results from farm 3 indicated no change in daily growth rate (0.41 kg); however, mortality levels decreased from 4 to 2%.

3.6. Discussion

The clinical history and diagnostic data indicated that PRRS virus was present in the nursery population in all three farms. The initial serological data indicated that the virus was recirculating between the older, infected carrier pigs and the seronegative weaners which entered the nursery every week. By eliminating the carrier pigs, it was possible to break this cycle of infection, even when only half of the rooms in the nursery complex on farm 3 was depopulated. The results from farm 3 indicate that it may be possible to eliminate the virus without completely emptying the facility as long as the exposed pigs are identified. Improvements in performance were seen in farms 1 and 2, but no improvement in growth rate was seen in farm 3. However, ND reduced the mortality due to streptococcal septicemia from 4 to 2%.

This protocol demonstrates that it is possible to control PRRS. One disadvantage of this protocol is the need for an offsite facility that can handle the nursery pigs during the
clean-up period. In order to alleviate this problem, finishing facilities can be temporarily remodelled to accommodate young pigs by using hovers, straw bedding, heat lamps and lying boards to reduce the chilling effect of the concrete floors. Ideally, such a project should take place during warm weather, not only to improve the comfort of the piglets, but also to enhance the killing of the virus. If farrowing space is available, weaned pigs could remain in the crates or farrowing could be purposely skipped via strategic planning of matings to avoid having to wean offsite. Overbreeding later, together with early weaning could make up for the missed farrowings. This procedure would minimize the need for an offsite nursery but does require careful planning.

It has been demonstrated that fecal shedding can be one of the primary sources of the spread of the virus and special emphasis was therefore placed on the removal of manure and cleaning of the slurry pits (Yoon et al., 1993). All the nurseries contained shallow (40 to 60 cm) pull plug pits or flush gutters, so they were easy to clean.

Before initiating a nursery depopulation for the elimination of the PRRS virus, it is essential to obtain an accurate serological picture of the exposure of the animals to the virus. All three farms had a similar IFA profile and this may have enhanced the success of the programs. Samples collected from the breeding herd and from the pigs three to four weeks after weaning need to be IFA negative. A high prevalence of IFA negative sows was important for the success of this program, because it indicates the absence of active viral shedding or recent exposure in the sow herd. Samples from the finishing barn should be at the most 25 to 50% positive, with titers in the range of 1:16–1:64. Titers of 1:256 or higher may indicate recent exposure and could indicate the movement of the virus through the herd. If this is the case, the depopulation program should be postponed and the herd should be retested two to three months later.

Finally, it is not known for how long the nurseries will remain free of PRRS. This depopulation and cleaning protocol may need to be repeated, perhaps on an annual basis. However, the benefits of the program appear to outweigh the financial cost and short term inconvenience of offsite weaning.

4. Preparation for nursery depopulation

As previously described, it appears that it is important to control the transmission of PRRS virus in the breeding herd. Active shedding of the virus in the adult population leads to infected suckling piglets with subsequent movement of virus into the nursery. Critical to the control of breeding herd shedding is the management of the gilt pool. The introduction of seronegative, naive gilts to an infected breeding herd population has been demonstrated to result in recurrent episodes of reproductive failure (Dee and Joo, 1994). Similarly, introduction of viremic gilts may result in continuous exposure of the population to high levels of virus (Dee et al., 1995). It appears to be very important to understand the serostatus of the replacement herd as well as the breeding herd in order to adequately prepare gilts for introduction. It is recommended that animals which originate from a negative herd be housed in an offsite isolation facility for a 60 day period (Dee and Joo, 1995). With the advent of commercially available vaccines, it is now possible to immunize such stock prior to entry. Based on recent information, it
appears that the primary cell mediated immune response may be short lived following exposure to PRRS virus (Molitor, 1995). Therefore, 2 vaccinations are recommended, one on arrival and the other 30 days later. Acutely infected animals should also be isolated for a 60 day period to reduce the risk of introducing virus to the breeding herd.

The decision whether to vaccinate is farm specific. Published data has indicated that PRRS positive animals may enter negative herds if proper isolation and serologic monitoring is carried out (Dee et al., 1994). Sequential testing of specific animals has been recommended as a way to monitor the presence of circulating PRRS virus in an isolated population. Seronegative sentinel animals are also helpful to assess whether viral transmission is taking place. Following collection of evidence of antibody decay in monitored stock, as well as no evidence of seroconversion in sentinels, the animals in question may enter a herd.

It should be stressed that any animals which display persistent antibody titers \( \geq 1:256 \) during the 60 day isolation period should be culled. It has been described that carrier animals may demonstrate persistently high antibody titers over prolonged periods and do not display titer decay (Wills et al., 1995). The primary serologic test used in these protocols has been the indirect fluorescent antibody (IFA), which measures IgG. However a new IgM IFA test may be an applicable tool for monitoring active infection in similar situations. IgM titers are highly correlated with the presence of viremia and are short lived (Joe et al., 1995).

5. Field experiences with vaccine

The commercially available PRRS vaccine (RespPRRS-NOBL Laboratories/Boehringer Ingleheim) has been available for a short period of time. It is a modified live vaccine and approved for use in pigs from 3–18 weeks of age. The vaccine is not approved in breeding animals, however if it proves to be safe in pregnant sows, the use of vaccine may provide a consistent, protective immune response in the breeding herd. This could be critical to the success of controlling transmission of PRRS virus in adult populations. Until then, the use of the product in adult animals depends on farm specific data, discretionary use and valid veterinary–client–patient relationship.

To conclude, it must be emphasized that it is important to view vaccination as a part of a control program, not the final solution. Practitioners must be sure to collect adequate data and attempt to understand patterns of viral transmission, the risk of persistent infection and whether naive subpopulations of animals exist within infected farms, prior to implementing its use.

6. Conclusion

In our opinion, it has been encouraging to see practitioners attempt to solve PRRS problems using scientific means. Practitioners are using available diagnostic tools to acquire answers to many questions, including differences in exposure level to PRRS virus within populations, determining the PRRS serostatus of replacement animals and
whether stage specific viral transmission exists. However, in spite of all our progress PRRS is still a frustrating disease. Not everything has been successful and more answers are needed. Communication with research scientists and close working relationships with diagnosticians appears to be very important for the development of new measures of control, as well as the improvement of existing strategies.

References

Dee, S.A. and Joo, H.S., 1994. Prevention of the spread of PRRS virus in endemically infected pig herds by nursery depopulation. Vet. Rec., 135: 6–9.

Dee, S.A., Joo, H.S. and Pijoan, C., 1994. Controlling PRRS virus transmission: Handling infected seedstock. Comp. Cont. Ed., 16: 927–933.

Dee, S.A. and Joo, H.S., 1995. PRRS in the US: The re-education of the swine practitioner. Swine Health Product., 3: 81–84.

Dee, S.A., Joo, H.S. and Pijoan, C., 1995. Controlling the spread of PRRS virus in the breeding herd through management of the gilt pool. Swine Health Product., 3: 64–70.

Halbur, P., 1995. Strain variation of PRRS virus: Field and research experiences. Proc. Am. Assoc. of Swine Practitioners. pp. 391–394.

Joo, HS., Park, B.K., Dee, S.A. and Pijoan, C., 1995. An indirect fluorescent IgM antibody test for the detection of PRRS virus infection. Proc. 2nd Int. Symp. on PRRS.

Keffaber, K.K., Stevenson, G.W., Van Alstine, W.G., Kanitz, C.L., Harris, L., Gorcyca, D., Schlesinger, K., Schultz, R., Chladek, D.W. and Morrison, R.B., 1992. PRRS virus infection in nursery/grower pigs. Am. Assoc. Swine Pract. Newl., 30.

Loula, T.J., 1991. Mystery Swine Diseases. Agri. Pract., 12: 23.

Meulenberg, J.J.M., Bende, R., Ros, J., Petersen den Besten, A., Dreekhuyser, E., Wensvoort, G. and Moorman, R., 1995. Molecular characterization of Lelystad virus. Proc. 2nd Int. Symp. on PRRS, p. 3.

Molitor, T.W., 1995. Immune response to PRRS virus. Proc. Am. Assoc. of Swine Practitioners, pp. 395–396.

Polston, D.D., Gorcyca, D. and Chladek, D., 1994. An evaluation of the financial impact of PRRS in nursery pigs. Proc. 13th Int. Pig Veterinary Congress, p. 436.

Wensvoort, G., Terpstra, C., Pol, J.M.A., TerLaak, E.A. Bloemraad, M., Dekluyser, E.P., Kragten, C., Van Buiten, L., Den Besten, A., Wagenaar, F., Broekhuysen, J.M., Moouen, P., J.M., Zeistman, T., De Boer, E.A., Tobben, H.J., De Jung, M.F., VanVeld, P., Groenland, G.J.R., Van Gennip, J.A.M., Voets, M.T.H., Verheijden, H.H., and Braamskamp, J., 1991. Mystery swine disease in the Netherlands: The isolation of Lelystad virus. Vet. Quart., 13: 121–130.

Wills, R.W., Zimmerman, J.J., Yoon, K.J., Swenson, S.C., McGinley, M., Hill, H., Platt, K., Hennings, J.C. and Nelson, E., 1995. PRRS virus: A persistent infection. Proc. 2nd Int. Symp. on PRRS.

Yoon, I.J., Joo, H.S., Christianson, W.T., Kim, H.S., Collins, J.E., Morrison, R.B., and Dial, G.D., 1992. An indirect fluorescent antibody test for the detection of antibody of SIRS virus in swine sera. J. Vet. Diagn. Invest., 4: 144–147.

Yoon, I.J., Joo, H.S., Christianson, W.T., Morrison, R.B., and Dial, G.D., 1993. Persistent and contact infection in nursery pigs experimentally infected with PRRS virus. Swine Health Product., 1: 6–9.