CHAPTER TEN

Field Experiences with Different Methods of Controlling PRRS Virus

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by M McCaw

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Introduction

Optimization of herd health and production efficiency begins at birth. Set-backs to the piglet during lactation, particularly by PRRS virus in combination with secondary infections, will affect later stages of production and may predispose pigs to additional health problems. McREBEL™ management initially evolved from an attempt to control pre-weaning mortality and regain healthy growth during an outbreak of reproductive PRRS that had lasted for 18 months (McCaw, 1995). The underlying philosophy of this approach is that optimization of suckling piglet growth requires minimizing interventions and maximizing supportive care. McREBEL™ facilitates both and optimizes the growth of the suckling pig, as evidenced by increased weaning weights, decreased pre-weaning mortality and nursery mortality, and increased average nursery close-out weights vs traditional multiple cross-fostering management (McCaw, 2000a). Later applications of McREBEL™ and field research have shown its usefulness for meeting the needs of the sucking piglet, and, therefore, optimizing the survival and growth of healthy pigs (McCaw, 2000b). These effects are observed both in PRRS-affected and unaffected herds, although to different degrees, depending upon the herd’s general health status.

Control of Clinical PRRS

PRRS virus outbreaks were initially reported in North Carolina in the late 1980's (Dial et al., 1990). Clinical signs observed during the epidemic included sows off-feed, mid- to late-term abortions occurring with, and then followed by, increased numbers of stillbirths and mummified fetuses of decreasing size (Loula, 1991). Concurrent with the reproductive breaks was a marked increase in pre-weaning mortality among piglets born during the outbreak. Antibiotic treatments and supportive care of these piglets produced little or no response. Efforts that cross-fostered poor-doing, sick piglets on to “good-milking” sows to minimize competition from larger pigs produced particularly poor results (McCaw, 1995). Affected piglets and weaned pigs typically showed signs of secondary bacterial infections with Streptococcus suis, Haemophilus parasuis, E. coli, Pasteurella multocida type D, Bordetella bronchiseptica, Actinobacillus pleuropneumoniae, and/or other opportunistic pathogens (Done and Paton, 1995; Stevenson et al., 1993).

In 1991, it was demonstrated that PRRS was caused by a virus that grew in swine lung macrophages. Koch's postulates were fulfilled when it was shown that infecting susceptible pregnant sows with the virus caused fetal death and the weak-born piglet syndrome (Terpstra et al., 1991). Vertical transmission by in utero infection of fetuses was also proven in those initial studies, although it was not realized at the time how important in utero PRRS virus infection was to subsequent acute and chronic PRRS in nursery pigs and finishers (Benfield, 1997; Feng et al., 2001; Segalés and McCaw, 2002) Early empirical evidence came from the observation that it was necessary to stop virus circulation (horizontal transmission) in sow herds in order to improve nursery health. Keffaber et al. (1992) found nursery depopulation alone to be ineffective in the control PRRS virus-associated disease losses. Nursery depopulation for controlling PRRS-associated diseases was only successful in "stable" sow herds (Dee and Joo, 1994). The lesson to be learned was that stopping horizontal transmission between sows decreased the occurrence of infected sows transmitting the virus vertically to their unborn or suckling offspring. Afterwards, it became possible to successfully eliminate PRRS virus by nursery depopulation and subsequently sustain the PRRS virus-free status of the nursery.

An approach was needed to help swine producers control PRRS-associated losses in the farrowing house and nursery during the early phases of a PRRS outbreak. Obviously, nursery depopulation did nothing to improve PRRS-related morbidity and mortality in suckling piglets. In addition, on-going nursery pig disease losses needed to be controlled during the four months or more required for the sow herd to “stabilize” so that depopulation of the nursery could be successful (Dee and Joo, 1994). Aggressive antibiotic treatment of piglets and heroic efforts to cross-foster unhealthy, weak piglets onto nurse-sows for intensive care failed miserably to control the meningitis, arthritis, or scours, much less return piglets to normal growth rates and deliver healthy weaned pigs to the nursery (M McCaw, personal...
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Frequent handling and treatment of piglets contributed to the problem by spreading bacterial pathogens and PRRS virus to other litters via pathogen-contaminated personnel and equipment (McCaw, 2000a; Otake et al., 2002). Lastly, “normal” appearing, but viremic piglets in the early stages of bacterial infection carried disease problems to healthy litters following “exchange” fostering of visibly sick piglets to nurse sows. Field observations of ≥5-week-old, unhealthy piglets - unresponsive after multiple treatments and still suckling nurse sows in a herd chronically affected with reproductive PRRS - led to the creation of a new piglet management strategy (McREBEL™) designed to control the occurrence of secondary bacterial disease and mortality in both suckling and weaned pigs (McCaw, 1995).

The Principles behind McREBEL™

By 1994, key facts about PRRS virus had become known. These were incorporated into a method of managing PRRS virus-affected piglets during acute outbreaks of reproductive PRRS. These facts included the following:

1. Piglets could become infected with PRRS virus in utero (Terpstra et al., 1991).
2. PRRS virus infection alone did not kill piglets (Halbur et al., 1996).
3. PRRS-affected piglets were usually infected with at least one of a variety of secondary bacterial infections (Done and Paton, 1995; Stevenson et al., 1993). Preweaning mortality and many of the signs of disease during PRRS outbreaks resulted from secondary bacterial infections in PRRS virus-infected piglets.
4. Piglets born through caesarean section are born nearly bacteria free, as demonstrated through the creation of specific pathogen free (SPF) breeding stock (Twiehaus and Underhaul, 1970).

To these research and diagnostic findings was added the field observation that extensive fostering of piglets and aggressive use of antibiotics failed to control PRRS in suckling and weaned pigs. Therefore, a strategy was devised to control these losses by limiting the exposure of PRRS virus-infected susceptible piglets to pathogenic bacteria and making the litter the all-in/all-out (AIAO) production unit in the farrowing house. The procedure was named McREBEL™ to reflect the objectives, i.e., to protect piglets from the bacterial infections and disease that seemed to result in their poor growth and death: Management Changes to Reduce Exposure to Bacteria and Eliminate Losses. McREBEL™ was also designed to meet production objectives, such as the need to use all functional teats available in a farrowing room. This is achieved by allowing the fostering of piglets within the first 24 hours after birth to litters with open teats. Fostering is limited to moving the minimum number of piglets to fill unused teats, and expressly not for equalization of body-weights within litters. Limited fostering is allowed with the full knowledge that even this could compromise the few piglets that are moved during an active PRRS outbreak.

McREBEL™ Rules

The objective is to maximize the number of piglets remaining on their birth mother and, secondly, to maximize the number of piglets remaining on the colostrum mother. The litter is the AIAO unit.

1. Do not cross-foster piglets after 24 hours of age.
   a. Move the minimum number of pigs necessary to load functional teats.
   b. Do not cross-foster for the purpose of creating uniform size or single gender litters.
   c. Prioritize assignment of functional teats to larger and more vigorous piglets.
   d. Smallest piglets are given the lowest priority, usually leaving them in their birth litter as “extras” if there are more piglets than available functional teats.
2. Do not move piglets between farrowing rooms and follow strict AIAO production.
3. A piglet held back from weaning (cross-fostered to a younger litter) takes a teat away from a younger, potentially healthier pig. Therefore, remove very sick, moribund, or poor body condition pigs from the system.
   a. Eliminate piglets that do not improve after treatment.
   1. Extended antibiotic treatment periods may be needed to treat bacterial diseases of PRRS virus-infected piglets.
2. Change needles between litters or pens of treated pigs.

b. Eliminate very thin, starve-out, lame, swollen-jointed, light body weight, long-haired, chronically sick, and scouring piglets as they are found.

c. Eliminate piglets at weaning that are too light to survive in the nursery and have poor body condition

4. In the nursery, implement practices to maximize piglet survival and performance:

a. Size piglets into pens carefully.

b. Place the smallest piglets in a warm, non-drafty part of room.

c. Hand-feed the smallest piglets 4 to 6 times a day for 5 days.

d. Switch rations based upon weight of pigs in the pen, not the room.

e. Use heat lamps and/or plastic lying pads for small piglets.

f. Lower one water nipple per pen in designated small piglet pens and jam it open for 24 hours.

**McREBEL™ Farrowing Room Management**

The overall goal is to minimize mortality (pre-weaning mortality and nursery mortality combined) and maximize the average weight of the farrowing room and nursery for each farrowing or production group.

**Sow management**

Count functional teats on each sow at farrowing and place at least as many pigs on her as she has functional teats. "Overloading" sows with piglets, i.e., more piglets placed than estimated number of functional teats, is recommended when live born is very high. This ensures that all potentially functional teats are used, thus maximizing number of pigs raised. When in doubt, load sows with at least the average number of pigs weaned by her parity for the herd. Do not expect to wean any more quality piglets than there are functional teats in a farrowing room. To maximize the number of piglets weaned per room, maximize the number of functional teats in the room by proper gilt selection and sow culling practices. When more sows farrow than the number of crates available, keep sows with the best udders in the crates for full-length lactation. Culling decisions should include the sow's nursing ability and average number of pigs weaned.

At farrowing, the objective is to maximize the number of piglets remaining on their original mother and, secondly, to maximize the number of piglets remaining on the colostrum mother. Therefore, foster extra piglets to sows with extra functional teats in a way that optimizes the benefits of colostrum. Do not wait until "processing day" to foster piglets to open teats on sows. A piglet's ability to absorb colostral antibodies across the gut wall is reduced by half (half-life) approximately each three hours (Wagstrom et al., 2000). As a result, by 24 hours of age the piglet's ability to absorb colostral antibodies is minimal. Therefore, foster piglets 2 to 4 hours after birth if they are moving to a just-farrowed sow so that the foster mother provides the piglets colostrum. Alternatively, foster piglets at 6 to 12 hours of age if they are to be moved to a sow that farrowed the day before so that the piglets get colostrum from their birth mother. Mark these piglets to ensure they are processed the next day.

**Piglet management**

Ensure that crates are designed or adjusted to allow piglets free access to teats. Bottom bow bar, finger bar, or proctor type crates are best for this requirement. If you have adjustable straight-sided crates, make sure the crates are adjusted (or sows loaded to the properly adjusted crate) to the size of the sow's udder, i.e., the piglets are not forced to claw over the bar to reach the top teats.

Prioritize placement of piglets to functional teats by size. Give the highest priority to the largest pigs and the lowest to under-sized piglets. If more pigs are born than there are functional teats, make sure the under-sized pigs are the "extra" piglets in each litter. Do not create litters of under-sized piglets. This only prioritizes them over the medium and large pigs within farrowing rooms with too few functional teats to feed the number of piglets born live. Foster extra piglets based upon their ability to suck teats of the adopting sow, i.e., smaller pigs to young sows with small udders, larger pigs to older sows with large, heavy udders.

Do not foster piglets just to make litters of uniform piglet size. Size does not matter after the piglets have won and/or selected their teats at 36 hours of age. Cross-fostering after 36 hours of age will only displace another pig from its functional teat, cause fighting, and possibly carry new disease to the litter.
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There will be no more fighting if piglets are not fostered into or out of the litter. All pigs suckling a fully functional teat will thrive regardless of body size differences within the litter.

Look closely for piglets not suckling with littermates. Examine these pigs immediately for fever, scours, swollen joints, bad feet, gums damaged from needle teeth clipping, etc. and treat them. Never move smaller or sick piglets "back" to younger rooms. Moving the biggest and healthiest litters forward (early weaning) to open a sow for healthy "plateau" piglets within that room is acceptable.

If a sow dies or stops milking, keep the litter together as a group and in their original crate whenever possible. Wean the litter if the piglets are at least 14 days old. Otherwise, move a "just weaned" sow or "just farrowed" sow to the piglets' crate. Move a "just weaned" sow to the intact orphan litter if the piglets are 4 to 14 days old. Move a "just farrowed" and "zero weaned" sow (low number of live born) to the intact orphan litter if piglets are less than 4 days old.

Piglet body condition scoring may be used to monitor piglet health in the farrowing room:

1. **Good body condition** - At weaning, these pigs have sufficient body fat to make them appear round and smooth. Their shoulder blades, backbones, and pelvis bones cannot be seen.

2. **Fair body condition** - At weaning, the outline of the shoulder blade of these pigs can be seen, but not the backbone or pelvis. These pigs may have sucked a partially functional teat or been mildly affected by disease, such as lameness or diarrhea.

3. **Poor body condition** - In these pigs, shoulder blades, backbones, and pelvis bones are clearly visible at weaning. These pigs have been fostered frequently, sucked a poorly functional teat, or been affected by disease.

Expect 90% or more of the pigs to score “good” if sows and piglets are not sick and sows are milking normally.

It is better to euthanize unhealthy piglets than to try to save them. This would include small, weak, thin piglets (shoulder blades, backbone, and pelvis clearly visible), pigs that do not respond following 2 to 3 days of treatment for scours, thumping, swollen joints and lameness, and pigs in poor body condition at weaning.

**Summary**

The objective of McREBEL™ management is to optimize the health and growth of piglets and nursery pigs during and following PRRS outbreaks. McREBEL™ was developed in 1994 as a method to control PRRS-associated disease and mortality in suckling and nursery pigs. By that time, it had become apparent that extensive cross-fostering and antibiotic treatment did not control the high piglet morbidity and preweaning mortality seen during PRRS outbreaks. Furthermore, control of continued nursery pig losses could only be achieved by nursery depopulation months after the outbreak, i.e., when virus circulation stopped among the sows and the herd “stabilized.” Critical information known by that time included:

1. Piglets could be born alive and infected with PRRS virus.
2. Clinically-affected PRRS virus-infected piglets submitted to diagnostic laboratories died because of secondary bacterial disease.
3. Experimental infection with PRRS virus field isolates alone did not kill germ-free pigs.
4. Piglets were born nearly bacteria-free.

McREBEL™ evolved from these facts and the underlying premise that we needed to minimize the frequency and level of bacterial exposure to pigs in order to control clinical disease during PRRS outbreaks. Since conventional methods of caring for young pigs, such as antibiotic treatment and cross fostering, had consistently failed, the intention was to control clinical disease by minimizing piglet handling, treatment, and piglet fostering among litters. Ultimately, it was determined that groups of piglets raised using McREBEL™ management during PRRS outbreaks returned to normal levels of total mortality (preweaning mortality plus nursery mortality) and achieved nearly normal growth rates through the nursery. McREBEL™ may also offer economically significant production improvements in herds unaffected by PRRS and which foster piglets between litters frequently throughout lactation.
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Control in Large Systems
MA FitzSimmons and CS Daniels

Introduction

PRRS has been raising havoc with swine production for over a decade, but there is still little consensus among producers and veterinarians regarding control procedures in production systems. The confusion stems from the lack of concrete information in two areas: PRRS virus transmission and protective immunity.

Although the U.S. swine industry has three commercial vaccines at the time of this writing, no one has yet described how PRRS virus, either field virus or vaccine virus, elicits protective immunity. Furthermore, there is no practical way to evaluate cross-protection between the modified live virus (MLV) vaccines and heterologous field strains of PRRS virus. Vaccine has been used extensively in some swine production systems, but with mixed effects. Some systems report good results; others have blamed vaccines for creating even more severe disease problems. These highly dissimilar outcomes suggest one of two possibilities: 1) a lack of cross protection, or 2) no protection at all. If vaccines were cross protective, many herds should have improved dramatically when vaccinated. Since the problems the industry has had with PRRS have continued in the face of multiple vaccinations, it is easy to conclude that, in many cases, the vaccine has not created heterologous protection. That is, if the field virus were similar to the vaccine strains, the MLV products would have provided protection. In addition, there are many examples of farms returning to normal production without any vaccine use after severe outbreaks.

Therefore, our conclusion is that the long-term stability of PRRS virus-infected farms depends on consistent acclimatization. Acclimatization means preparing PRRS virus-naïve gilts for entry into the sow herd through the development of active immunity against field virus. For farms to maintain long-term stability, it is important that all PRRS virus-susceptible sows in the herd develop active immunity and that all replacements are immune prior to entry. The use of vaccine in these cases may create populations of sows that have not been exposed to the field strains within the herd. These unprotected populations can result in small re-breaks that show up as affected pigs in the nursery.

The following description details what may have happened over the past decade in the industry and helps explain why problems have escalated in the last five years.

A Clinician’s Perspective on Cyclic Outbreaks of PRRS

Since the initial breaks, it was observed that the rapid stabilization of the sow farm resulted from the rapid spread of the virus through an entirely susceptible population. If the original source of virus were incoming gilts, the continued introduction of these previously exposed, naturally protected replacements stopped the virus spread due to the absence of susceptible animals in the population. Ultimately, stabilization of the sow herd depended on one of three things occurring:

1. The entry of previously exposed, naturally protected gilts meant no susceptible population would redevelop.
2. The acclimatization (infection) of PRRSV-naïve replacements through exposure to infected nursery pigs maintained herd immunity.
3. The virus completely stopped circulating.

Over time, these stable farms eventually produced PRRS virus-negative pigs, these susceptible animals would then be moved into positive nursery/finishing systems, where they would become infected.

After a few years of unacceptable nursery/finishing performance, the industry implemented procedures to clean up the downstream pig flow, i.e., partial/complete depopulation (Dee, 1994, 1997a,b), all-in/all-out facilities (Dee, 1993), and/or unidirectional pig flow (Dee, 1998). The use of PRRS virus-positive replacement gilts eventually led to the near elimination of PRRS virus from the system. This situation resulted in naïve, completely

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susceptible gilts being produced for entering into the sow farm. The sow herd dynamics then began to change as the population started to differentiate into resistant and susceptible classes in regards to PRRS immunity. Some farms rolled through this phase and became PRRS virus negative. Other herds, after an uncertain length of time or number of naïve animal introductions, began to re-circulate virus and eventually classical, clinical PRRS reappeared. There were even re-breaks in cases where systems believed they were properly acclimating gilts by exposure to infected sows or nursery pigs. This was because the gilts they received were already positive, but to a heterologous strain of PRRS virus. These gilts were susceptible to the "resident" PRRS virus strain because the acclimatization process had failed, perhaps because the contact sows or nursery pigs to which they were exposed were no longer shedding virus. The use of MLV vaccines further confused the issue by creating antibodies that could not be differentiated from field virus. Therefore, seropositivity or seroconversion to PRRS virus could not be used to confirm exposure to field virus during acclimatization.

Some of these farms ended up in cycles: clinical outbreaks in the sow farm prompted procedures to acclimatize gilts, which lead to a clinically quiet phase, the production of negative pigs, and depopulation of nursery/finishing stages. Success in controlling PRRS virus circulation in the sow herd led to the production of negative “seeder” pigs, which resulted in the failure to transmit the endemic virus to resident gilts. The ultimate result was the introduction of PRRS virus-naïve replacement animals into the sow herd. Once in the breeding herd, these unprotected replacements eventually made contact with infected sows and became infected with the endemic PRRS virus. The result of the infection depended on her stage of gestation, but one possible outcome was weaning viremic pigs into the now-negative nursery. These cycles revolved around one main flaw, the failure to continually expose and protect incoming gilts to the resident PRRS virus. Several things led to this failure, including

1. The use of PRRS MLV vaccine in some acclimatization programs eliminated the system’s ability to determine by means of serology whether homologous field virus exposure had occurred.

2. The vaccine may have restricted the transmission of field virus to all intended replacements.

3. All in/all out gilt acclimatization required a consistent source of viremic sows or seeder pigs.

This last point is very important because it holds the key to successful long term stabilization of PRRS positive sow herds.

Methods of PRRS Control in Swine Units

Definitions

A few definitions are necessary before discussing an appropriate system for control of PRRS in large sow units. For simplicity, only gilt sources will be discussed, but the same concepts apply to boars. For clarity, "PRRS virus-naïve" is defined as an animal that has never been infected with either field strain or vaccine strain PRRS virus. In contrast, PRRS virus-negative is based on serum antibody status as determined by an ELISA or indirect fluorescent antibody (IFA) test. Within the specificity limits of the tests, PRRS virus-naïve animals will test negative, but infected or vaccinated animals may also test negative as antibody levels decline over time post infection.

Incoming animals can be put into two categories as derived by source: PRRS virus-naïve sow herds or positive sow herds. Naïve sow farms, by definition, produce PRRS virus-naïve replacement gilts. These gilts may be entering either PRRS virus-positive or naïve commercial farms. If entering naïve herds, these animals only need to be isolated; there is no need for off-site acclimatization (at least with regard to PRRS virus). If these animals are going to a PRRS virus-positive sow farm, they must be acclimatized to a PRRS virus homologous to the virus endemic on the farm before entering the sow herd. It is essential that these animals match the status of the sow herd before introduction.

The second category is animals sourcing from positive sow farms. Individual animals from these farms may be either naïve or PRRS virus-positive, depending on the production system and the success of the source sow farm’s PRRS stabilization protocol. The concept of making naïve pigs from previously infected sow herds has been debated extensively in the past five years, particularly in the context of gilt sales. There is no doubt that positive stable sow herds will make a lot of PRRS virus-naïve pigs. The problem with these systems is that they are always at risk of breaking down due to the unexpected introduction of naïve gilts or the circulation of another PRRS virus that is already present. Either
way, the result is PRRS virus-infected offspring moving downstream. If these pigs flow out to commercial finishing, there is no subsequent effect, except directly on that pig population. However, if these are replacement gilts, they could infect downstream sow herds and their production as well. Therefore, as a customer of that multiplication system you have to be concerned not only with the biosecurity of the source, but also their management of the PRRS stabilization process. It is the opinion of this author that, if you have a negative sow herd, you should only receive naïve replacements from naïve sow herds. If your gilt source was the origin of your resident PRRS virus but they have succeeded in making negative gilts, it would be acceptable to continue to purchase replacements from them, then acclimatize them to the resident virus via methods described later. However, if you have a positive sow herd and the gilts will originate from a herd other than the one that was the source of your virus, it would be preferable to only accept gilts from a truly negative source. It is important to understand that the same principles applied at the commercial level to create negative nursery/finisher populations are also applied at the source farm. It is important to understand that the same principles applied at the commercial level to create negative nursery/finisher populations are also applied at the source farm. It is important to understand that the same principles applied at the commercial level to create negative nursery/finisher populations are also applied at the source farm. It is important to understand that the same principles applied at the commercial level to create negative nursery/finisher populations are also applied at the source farm.

Receiving naïve gilts from naïve source farms

The ideal procedure is to receive naïve replacements from naïve source farms. This allows the option of acclimatizing the replacements to specific sow herds and their farm-specific strains of PRRS virus. It also decreases the chance of clinical breaks resulting from the introduction of a different PRRS virus into the herd. When naïve gilts are to be introduced to a positive sow farm, they must first develop immunity to the "resident" (endemic) strain of PRRS virus. This is the point at which differences in opinion arise. A procedure that has been widely publicized involves the use of a commercially available PRRS vaccine in combination with cull animal exposure in all-in/all-out off-site facilities to acclimatize incoming animals. The acceptance of this procedure has varied depending on past experience and perceived success. The system that will be described in this paper is quite different. This system is based on two simple premises:

1. Field virus infection results in effective long-term protection.
2. Successful PRRS acclimatization will decrease or eliminate the amount of virus circulating in the sow farm.

If acclimatization is managed as all-in/all-out and relies on exposure to cull sows to introduce virus to each group, unprotected gilts will eventually enter the sow farm because cull sows will not consistently shed and/or transmit the resident PRRS virus to naïve gilts. If gilts are vaccinated upon entry to the isolation/acclimatization site, it will be impossible to know if field virus exposure has occurred. Previous experiences suggest that commercial vaccines have not been completely cross protective (Benson et al., 2000). Without the ability to definitively determine if field virus infection has occurred, it is not possible to know whether successful PRRS acclimatization has occurred.

When introducing naïve gilts into positive farms the goal is exposure to a homologous field virus in an off-site acclimatization facility. For the purposes of this paper, the definition of acclimatization is infection of a susceptible animal with a specific disease organism. Natural infection and recovery is the surest way to elicit protection to PRRS virus and can be accomplished by one of three methods:

1. Exposure to viremic cull sows or nursery pigs.
2. Adding naïve pigs to a PRRS virus-infected, continuous pig flow.
3. Injection with live virus.

In the first method, the long-term risk is that shedding of PRRS virus in the sow farm eventually stops, thereby resulting in acclimatization failure. Continuous flow acclimatization is one way to address this eventuality. PRRS virus will continue to circulate in the population as long as susceptible gilts are continually introduced, but maintaining virus circulation is a function of population size, mixing procedures, and the interval between the introductions of susceptible gilts. Of these three, the introduction interval appears to be the most important factor. It is critical that the virus continues to circulate and successfully infects the next group. If too much time is left between introductions of gilts, the virus may stop circulating and populations of naïve gilts will again enter the sow herd. In practice, the clinical disease experienced during acclimatization is least severe in 10- to 14-week-old
A theoretical concern with continuous flow acclimatization is viral mutation. The virus goes through many replications, which may increase the chance for mutations that could create instability in the sow herd due to cross protection failure. In addition, it is impossible to control all the other infectious diseases circulating in the continuous flow population well enough to avoid increases in mortality and culling rates. Over time, some of these systems will become progressively worse and partial/complete depopulation may be necessary. If an acclimatization/isolation site is to be depopulated, it is important to maintain a source of virus to restart PRRS virus circulation. This can be accomplished by harvesting serum from viremic pigs and later injecting it into naïve gilts. An alternative to this procedure is to leave a few viremic pigs in the facility. Serologic testing is required to confirm exposure and infection when acclimatization is restarted.

The last method deals with intentionally exposing naïve gilts to live virus by injection. This is equivalent to using a live autogenous vaccine. This procedure may be necessary to allow sufficient "cool down" after infection if isolation/acclimatization is limited to 60 days. That is, in continuous flow systems, it may require three weeks for the virus to move through the population and expose every pig, which results in a delay of the "cool down" period. With injection, the "cool down" period starts immediately. Other advantages of this system include control of the dose and exact time of infection. If this system is used in conjunction with all-in/all-out pig flow, exposure to other pathogenic microorganisms can be reduced. This will lead to decreased mortality and culling. This procedure also requires less testing to confirm infection of the entire population because a few PRRS virus-positive gilts are sufficient to demonstrate that the inoculum was infectious at the time of injection. It should be acknowledged, that there are safety issues concerning this method that should be discussed by producers and veterinarians prior to its implementation. Use of this method has been challenged on ethical grounds, but it shares a striking similarity to other methods that are currently in use, e.g., intentional exposure of animals to materials contaminated with porcine parvovirus or transmissible gastroenteritis virus. Regardless of the method employed, the ultimate goal of acclimatization is to infect and consequently protect naïve replacement gilts against an endemic PRRS virus prior to entry into a positive sow farm. Acclimatization needs to consistently achieve the goal of infection/protection now and well into the future.

### Receiving gilts from positive farms

All replacements from PRRS virus-positive source farms should be viewed as positive gilts until proven otherwise. Some groups of gilts from positive farms are PRRS virus-naïve, but farms are known to re-break. What does this mean to the recipient sow farm? If the stability of the source farm is disrupted, virus may find its way downstream to your farm. This virus will not create significant clinical problems as long as it is sufficiently similar to the PRRS virus on your farm and susceptible sow populations have not developed due to failure to acclimatize naïve gilts. If the strain of PRRS virus the gilts introduce is sufficiently different, the result will be instability in the recipient herd.

“Naïve” gilts from positive sow farms need to be isolated and tested to prove they are actually PRRS virus-naïve. After their naïve status has been confirmed, they must be exposed to a homologous virus and prepared for entry into the sow herd. This program may consist of isolating weaned pigs in a nursery and, if they test negative ( naïve) at 10 to 12 weeks of age, acclimating them. The quality of diagnostic testing techniques available today determines the risk of receiving these animals. If the original gilt source sends positive and negative groups over time, as long as the virus is similar and continues to circulate in acclimatization there should not be a problem.

Acclimatization may not be needed if the source farm was the original-and-only source of your PRRS virus and it continues to allow PRRS virus to circulate through the finishing phase. Acclimatization may consist only of a "cool down" period of 60 to 90 days to decrease the risk of introducing a large level of virus into the sow farm. Actually, this system worked well until source farms started implementing techniques to create PRRS negative nursery/finishers – which then become your replacement gilts. Their reasoning was perfectly understandable when looking at nursery/finishing performance, but it created instability problems in the recipient commercial farms. This instability was due to a change in...
replacement gilt status from infected/protected to PRRS virus-naïve and the recipient farms' failure to adjust to the changing gilt status and take measures to assure acclimatization.

In summary, receiving gilts into positive farms creates two challenges. First, naïve gilts must be acclimatized to the "resident" PRRS virus. Second, if the gilts are PRRS virus-positive, the virus to which they were exposed must be the same as the virus endemic in the recipient herd to assure protection. Sufficient "cool down" time is important to allow the animals to respond immunologically and reduce the virus load. If the gilt source was not the original source of the endemic PRRS virus, additional problems may arise due to inadequate cross-protection. Receipt of seropositive gilts makes it impossible to measure if farm-specific PRRS virus exposure occurs.

Summary

Porcine reproductive and respiratory syndrome (PRRS) virus was identified over a decade ago. Even so, basic PRRS virus information, particularly in the area of immunity and transmission, is conspicuous by its absence. Controlling clinical cases in commercial production systems is a constant problem for producers and veterinarians. Although vaccines are available, the protection they confer is inconsistent. In this section, we discuss strategies for controlling PRRS, with an emphasis on methods for establishing and maintaining herd immunity. This overview is not intended to give answers to an individual farm or system PRRS problem. The intent is to stimulate thinking and to challenge popular paradigms. These practices may seem risky or radical to some, but not to the producers who have successfully used these techniques to protect their livelihood.

References


Control Using Oropharyngeal Scraping Inoculation
G Allison

Introduction

Natural exposure to infectious agents in a controlled fashion has long been used to infect swine and establish immunity. Typically, these methods involve exposure to acutely infected animals, and/or pathogen-contaminated tissues and manure. However, controlled PRRS virus infection in the field has proven to be a difficult or unreliable process. Exposure of susceptible animals to PRRS virus by contact with infected animals is successful in some instances and not in others. Exposure to contaminated materials is generally unsuccessful because PRRS virus is thermal and pH labile and, therefore, does not survive outside of the host for extended periods. The virus is unstable in environments containing low levels of detergents (Plagemann, 1996), it is easily activated in a dry environment (Blomraad et al., 1994), and will not survive on fomites commonly found in the barn (Pirtle and Beran 1996). Somewhat ironically, an inoculation of 10 or fewer PRRS virus particles by intranasal or intramuscular routes easily infects swine (Yoon et al., 1999). Thus, PRRS virus is frequently described as highly infectious but not overly contagious.

In this chapter, we describe a practical method for exposure of susceptible animals to PRRS virus. This method is appropriate in herds where the strategy for preventing clinical PRRS involves maintaining a PRRS virus-positive breeding herd. In brief, the method involves exposing naive replacement animals to a virus-contaminated inoculate collected from PRRS virus-infected grower pigs. As is true with all methods involving natural exposure, there is a distinct possibility of unknowingly transmitting not just PRRS virus, but also other pathogens. Whether this is a detriment or a benefit will depend on the herd and the circumstances, but should always be kept in mind and discussed with the herd owner. The advantages of oropharyngeal scraping inoculation are its reproducibility, simplicity, and the fact that animals establish immunity to PRRS virus strains circulating in the herd prior to their introduction.

Procedure

Animals to be sampled should be selected from populations with laboratory confirmation of PRRS virus infection. Oropharyngeal scraping samples are collected by using age-appropriate restraint and scraping the tonsil of the soft palate with a sterile, long-handled spoon. Typically, restraint involves snaring the animal and using an oral speculum to hold the mouth open. Collection spoons may be fashioned from standard stainless steel flatware to which an extension of appropriate length is welded to the handle. Sufficient pressure should be applied during scraping to obtain a quantity of saliva, mucus, and cellular debris in the bowl of the spoon. This generally requires 4 to 6 passes of the spoon. Pigs with excess feed in the oral cavity should not be sampled. In animals of 150 pounds (68 kilograms) or less, the entire procedure requires 15 seconds or less.

Once collected, the scrapings are removed from the spoons using a swab. The sample material can then be prepared, using simple laboratory techniques, for injection into susceptible animals. Prior to use, the material should be tested to confirm the presence of PRRS virus by PCR.

Summary

Oropharyngeal scraping inoculation has been utilized as a method to manage PRRS virus since May 1999 in a variety of clinical settings. The immunity that develops from PRRS virus infection protects the convalescent animal from subsequent challenge against the homologous viral strain. For that reason, this technique can be an important herd-specific component in the control of PRRS. As is shown in Tables 4.4.1 and 4.4.2, seroconversion of 100 percent of the inoculated animals is possible in the field. In both genders and in both mature and immature swine, the technique has reliably induced an antibody response that seems consistent with a stable population. Failure of the inoculation procedure, as was seen in Herd E, resulted from collecting oropharyngeal samples from swine subsequently found to be negative for PRRS virus infection. Accordingly, the inoculum should be tested for PRRS virus by PCR prior to use. Likewise, evaluation of animals for pre-existing infectious diseases, such as salmonellosis, is strongly advised as PRRS virus can act synergistically with concurrent infections. The advantages of this technique are its reproducibility, the development of immunity to herd-specific viral strains, and the ability to inoculate swine under...
controlled circumstances and allow for a "cool down" period prior to introduction into the herd.

References


Control with Modified-Live Virus (MLV) PRRS Vaccine
TG Gillespie

Introduction

Effectively controlling PRRS virus is one of the most challenging tasks facing veterinarians and swine producers today. Effective control of the PRRS virus can be complicated, and may involve many factors:

1. Number and type (identification by sequence) of viruses present in a farm or system.
2. Risk of new virus introductions.
3. Management strategies to combat PRRS.
4. Pig flow.

The objective of this paper is to focus on the role of MLV vaccine and immune management in the control of PRRS virus. Discussion will also include the use of MLV vaccine in an off-label use that requires a valid veterinarian-client-patient relationship (VCPR) and the use of all diagnostic tools available to understand virus activity prior to implementing the program and monitoring throughout.

Basics in PRRS Virus Control

Effective PRRS virus control requires a systematic approach that utilizes diagnostic investigations to determine where, and at what stage of production the virus is actively circulating in a farm or system. Once the pattern of viral circulation is understood, intervention strategies can be systematically employed in an effort to minimize PRRS viral circulation and its impact on the production system.

Among the tools implemented in the PRRS virus control formula is effective use of modified-live virus (MLV) PRRS vaccine for the control of the PRRS virus. There are two modified-live PRRS vaccines available in the U.S.: Ingelvac® PRRS MLV, and Ingelvac® PRRS ATP (Boehringer Ingelheim Vetmedica, Inc., St. Joseph, Missouri). Ingelvac® PRRS MLV is licensed for use in pigs 3 weeks of age or older and in non-pregnant gilts or sows 3 to 4 weeks prior to breeding in PRRS virus positive farms. Ingelvac® PRRS ATP is licensed for use in pigs 3 weeks of age or older, up to 18 weeks of age in PRRS virus positive farms.

Field Studies

A new technology being attempted in the swine industry is to utilize MLV PRRS vaccine in a population based approach for PRRS control. This protocol is often referred to as “mass vaccination” and implements modified-live vaccine to an entire population of growing pigs or a breeding herd population at a point in time. The objective of this approach is to establish a uniform or homogenous immune status within a population, by eliminating naïve susceptible subpopulations of pigs within that population.

The spread of field PRRS virus is enhanced when carrier pigs shed virus to subpopulations of naïve pigs that coexist within the same population (Dee et al., 1996). Published reports have also demonstrated that regularly introducing seronegative, naïve pigs into infected populations enhances the spread of virus and maintains the cycle of infection (Dee and Joo, 1994). The existence of susceptible subpopulations is a factor in the maintenance of persistent PRRS viral transmission from carrier animals in chronically infected populations. Vaccination has been used as a means to eliminate naïve subpopulations and to control the spread of field virus within infected herds (Dee and Philips, 1998).

The spread of field PRRS virus is enhanced when carrier pigs shed virus to subpopulations of naïve pigs that coexist within the same population (Dee et al., 1996). Published reports have also demonstrated that regularly introducing seronegative, naïve pigs into infected populations enhances the spread of virus and maintains the cycle of infection (Dee and Joo, 1994). The existence of susceptible subpopulations is a factor in the maintenance of persistent PRRS viral transmission from carrier animals in chronically infected populations. Vaccination has been used as a means to eliminate naïve subpopulations and to control the spread of field virus within infected herds (Dee and Philips, 1998).
1. Eliminating negative subpopulations through the process of mass vaccination will prevent the continued spread of field virus within the population.

2. Mass vaccination of the entire population will prevent the pig-to-pig spread of vaccine virus.

3. Establishing a period of herd closure and unidirectional pig flow for a controlled period of time, thereby preventing naïve pigs from being introduced into the vaccinated population, will produce a noninfectious population.

4. Once established, the noninfectious population will be incapable of transmitting field virus or vaccine virus, allowing both strains to be eliminated as vaccinated, infected pigs are marketed and naïve replacements are introduced over time.

The implications of this study were that the use of a protocol of mass vaccination and a period of herd closure eliminated naïve subpopulations and the spread of PRRS virus creating a noninfectious population. Mass vaccination and a 60-day period of closure and diverted pig flow enabled the entry of nonvaccinated PRRS negative pigs into the finishing phase of production to remain negative and led to the successful control and elimination of virus from the site.

This protocol has been repeated in other finishing populations with identical results (Philips et al., 2000; Wonderlich et al., 2002). Mass vaccination and herd closure has also been compared to a protocol that utilized herd closure alone for the control of PRRS virus in finishing populations of pigs (Philips and Dee, 2002). The results of the study suggested that mass vaccination with herd closure is an effective strategy for the control and elimination of PRRS virus from targeted finishing populations. This study clearly demonstrated that MLV vaccine could be successfully used in a PRRS virus control and elimination program. Finishing populations that employed mass vaccination and herd closure/unidirectional flow were successful in controlling and eliminating the PRRS virus (4 successes of 4 attempts). The results were consistent with the creation of a noninfectious population via mass vaccination. The finishing populations that employed only herd closure/unidirectional flow without the use of vaccine all failed to eliminate the PRRS virus (zero successes of four attempts). In the latter case, the results showed continued transmission of PRRS virus from infected pigs to naïve susceptible pigs, which resulted in the failure to control and eliminate the virus. Thus, site closure and unidirectional pig flow alone did not control or eliminate transmission of PRRS virus, again suggesting that immunization with an effective MLV PRRS vaccine was an essential component for achieving PRRS virus elimination.

Recent studies have investigated applying the mass vaccination and herd closure protocol to breeding herds (Flores and Dufresne, 2002; Gillespie et al., 2002; Philips et al., 2002; Turner and Dufresne, 2002). The strategy employed mass vaccination with a MLV PRRS vaccine and a period of herd closure with the goal of eliminating naïve subpopulations within the breeding herd and developing a protective immune response. The objective was to create a homogeneous immune status within the breeding herd population and minimize the spread of PRRS virus. The use of MLV vaccine in an off-label use requires a valid veterinarian-client-patient relationship (VCPR) with risk assessment activity, although ongoing research is being conducted to evaluate the use of MLV in all reproductive stages.

Systematic PRRS virus control in a farm or system begins with the breeding herd. Control of viral circulation in the breeding herd is fundamental to the process of reducing the prevalence of infected offspring and their impact on “down-stream” nursery and finishing populations. The goal of most farms or systems today is to “stabilize” the breeding herd as it regards PRRS virus status resulting in a reduction of PRRS virus prevalence in offspring and minimizing PRRS virus circulation in the nursery and finishing populations of pigs. PRRS stability in the breeding herd is defined as “the lack of evidence of detectable viral transmission; horizontally or vertically, from sow to offspring”. The mass vaccination protocol is implemented in an effort to achieve these objectives. Modified-live vaccine used in a mass vaccination protocol provides:

1. Consistent and controlled exposure of the breeding herd population.
2. Continued maintenance of a homogenous level of immunity.
3. Minimization of susceptible subpopulations.
4. Reduction of the risk of chronic replication/circulation of field virus.
The basic protocol employed in the mass vaccination strategies for breeding herds is:

1. Closure of the breeding herd for a minimum of 60 days.
2. Mass vaccination of the herd with MLV vaccine twice at 30-day intervals.
3. Implementation of a maintenance mass vaccination strategy that occurs on a quarterly interval or less, depending on the needs and level of challenge within the herd and specific herd risk factors.
4. Replacement gilts are routinely vaccinated with MLV PRRS vaccine in isolation/acclimatization beginning at least 42 days before introduction into the sow herd.

Results from studies that have utilized this protocol in breeding herds have demonstrated success in attaining PRRS stability and in the reduction in prevalence of field virus in “down-stream” nursery and finishing flows. These, in turn, result in improved health and production performance.

The reduction in prevalence of PRRS virus in growing pigs following stabilization of the breeding herd often allows control of the PRRS virus by pig flow strategies, such as all-in/all-out movement of pigs by facility or site. In situations where pig flow in the growing pig population is continuous by airspace, PRRS virus can continue to circulate in a chronic/endemic fashion. In these situations, it may be necessary to employ strategic vaccination in an effort to control viral circulation and minimize the impact of clinical disease. The use of MLV PRRS vaccine for the control of the PRRS virus in growing pigs for the respiratory form of the disease is an effective tool in these cases. Timing of vaccination is extremely important in order to optimize protective immunity offered by vaccination. It has been demonstrated that at least 4 weeks is required between vaccination and field virus exposure in order for the pig to generate a good protective immune response (Halbur and Roof, 1999). This suggests that a diagnostic investigation may be warranted in order to determine when pigs are being exposed to field virus so that optimum placement of vaccine can be accomplished. Diagnostic tools used in both monitoring and diagnostic investigations include the use of PCR tests on nursing piglets and early nursery pigs, as well as the ELISA on older nursery pigs and negative populations, such as sentinel replacement animals.

**Summary**

In summary, use of MLV PRRS vaccine can be successfully utilized to stabilize and control PRRS virus in conjunction with additional management techniques. A systemic approach is the most effective method in developing an immune management program. Points to bear in mind are the following:

1. Controlling PRRS virus is a challenging task and involves many factors.
2. Effective control requires a systematic approach that implements multiple PRRS virus management tools.
3. Modified-live PRRS vaccines can be effectively employed as an immune management tool in an effort to minimize field virus circulation in populations of pigs.
4. The first goal is to attain stability of the breeding population using strategic monitoring and immune management. Breeding herd stability will, in turn, influence the PRRS virus status of the nursery and finishing populations.
5. Strategic monitoring, including the use of PCR and ELISA, will indicate when stability has been achieved.
6. Immune management includes viral exposure internally (within the herd) and viral exposure externally (prevent new entry of virus into the herd).
7. Replacement gilt management is important in PRRS virus control programs. The animals must be properly prepared and stable/non-infectious prior to entry into the sow herd. In some cases, naïve replacements can be used in a monitoring program to detect virus activity (sentinels).

**References**


These approaches and techniques have not necessarily been rigorously tested under scientifically valid conditions and may not be appropriate for all herds. Therefore, readers are strongly encouraged to fully consider the merits, drawbacks, and implications of these methods prior to applying them in the field. These views are solely those of the author, and are not approved by the National Pork Board.


Control with Inactivated Virus PRRS Vaccine
E Thacker, B Thacker, W Wilson, and M Ackerman

Introduction

Porcine reproductive and respiratory syndrome (PRRS) virus continues to be one of the most important swine diseases in the world. Strategies for PRRS virus control and eradication vary among production systems and veterinary advisors, but there is growing sentiment that a PRRS virus-free herd is the “gold standard,” especially for herds that produce breeding stock. In the case of PRRS virus, the gold standard has not been easy to achieve.

Inactivated Virus PRRS Vaccines

Only one inactivated vaccine product (PRRomiSe®, Intervet Inc., Millsboro, DE) is currently licensed in the U.S. Several manufacturers produce autogenous inactivated vaccines, but little data is available regarding their use, efficacy, or immune response. Use of an inactivated PRRS virus vaccine in Europe was reported to significantly reduce the number of stillborn and mummies following experimental challenge (Charreyre et al., 1998; Reynaud et al., 1998). Current label directions for the licensed inactivated vaccine specify an initial 2.0 ml dose to breeding females 5 to 8 weeks after service and a second 2.0 ml dose 14 to 28 days later. The manufacturer recommends that this two-dose vaccination regimen be repeated at subsequent gestations. The vaccine has not been approved for use in pigs at other stages of production.

Studies with a Commercial Inactivated PRRS Vaccine

Iowa field study
A recent field study funded by the National Pork Board investigated the eradication of PRRS virus through intensive vaccination of sow herds and young pigs with PRRomiSe®. Three herds in Iowa that had recently experienced PRRS outbreaks participated in the project. Herd T contained 80 sows, farrowed 5 times per year, and all phases of production were contained on one site. Herd S contained 60 sows, farrowed 4 times per year, and all phases of production were housed on one site, with the exception of several small adjacent sites used for breeding stock. Herd K consisted of 150 sows and farrowed every 28 days (5 groups of sows, 3 week lactation). For this herd, one site contained the breeding herd and nursery pigs and another site contained the finishing pigs in one barn with 4 rooms.

The inactivated vaccine was administered according to the manufacturer’s recommendations with regard to dose and injection site. At the start of the study, all breeding animals were vaccinated twice at 3 to 4 week intervals. Thereafter, gilts were vaccinated twice prior to breeding, sows and boars once quarterly, and the pigs at weaning and again 3 to 5 weeks later.

All herds noted improvements in overall health status, especially related to respiratory disease. Herd K reported time-to-market decreased by 3 to 4 weeks. All producers were interested in continuing the vaccination program, including the pig vaccinations, although the expense of vaccinating the weaned pigs is probably prohibitive in the long term. In all herds, vaccination of sows appeared to increase serum antibody titers and most sows tested were seropositive following the initial intensive vaccination program. All herds were successful in producing PRRS virus-free pigs from the breeding herd. Herds S and T began producing negative pigs as soon as the intensive vaccination program had been instituted. Herd K, the herd with the most intensive pig flow, did not produce PRRS virus-free pigs until the third monthly weaning group after initiation of the program. Only Herd S was successful in maintaining a PRRS virus-free status through the finishing phase. Twice during the study, it appeared that the finishers in Herd K would stay negative, but the situation reversed to a pattern in which the pigs becoming infected within one month after entering the finisher.

Although there is no single definitive method for controlling or eliminating PRRS virus from herds or producing PRRS virus-free pigs, the results of this field study suggested that an inactivated PRRS vaccine could be beneficial in producing PRRS virus-free pigs from infected sow herds.

Study in a 600-sow multiplier herd
In a separate field study, PRRomiSe® vaccine was used to control PRRS in a 600-sow gilt multiplier herd with 3-site production (M. Ackerman, personal communication). In December of 1999, the entire
sow herd, which had previously been vaccinated with a modified-live virus (MLV) PRRS vaccine, was vaccinated once with the inactivated vaccine and all gilts in isolation were vaccinated twice, two weeks apart. Starting three weeks later, sows and gilts received a second dose of vaccine at 60 days of gestation. Vaccination of all sows and gilts at 60 days of gestation was continued thereafter. The practice in this herd has been to obtain gilts from a PRRS virus-free herd at 6 months of age. All incoming gilts are maintained in isolation/acclimatization facilities for 30 days prior to introduction into the sow herd. Gilts are vaccinated with the inactivated vaccine upon arrival and again 2 weeks later. Upon entry into the sow herd, nearly all gilts (over 96%) are seronegative for PRRS virus, although they have been vaccinated twice with the inactivated vaccine. Sow and gilts are given a booster dose at 60 days of gestation. At the beginning of the program, 40 percent of the sow herd was seropositive, the nursery pigs were seronegative, and the finishers were seropositive. Testing in May 2000 revealed seropositive nursery pigs, with antibody levels decreasing in a pattern suggestive of maternal antibodies. Nursery pigs were seronegative by 8 to 10 weeks of age and remained negative through the finisher. Since September of 2000, the sow herd has been tested monthly (36 to 45 animals per month) and the percentage of seropositive sows at each testing has ranged from 10.0 to 52.3 percent. As of April 2002, through natural attrition and continued vaccination with the inactivated vaccine, the herd is now 85 percent seronegative for PRRS virus. Older animals have been gradually replaced, as are all sows or gilts testing positive for serum antibodies. This regimen appears to be proceeding successfully with regard to eradicating PRRS virus from the herd based on serology and the production of PRRS virus-negative replacement gilts through the finisher.

Summary

PRRS virus vaccination is as a tool for decreasing the impact of PRRS virus on the breeding herd. The NAHMS 2000 survey reported that 53.5 percent of breeding females were vaccinated for PRRS virus, with MLV vaccine being the most commonly used (37.7%). MLV vaccines are typically thought to induce a more effective immune response compared to inactivated virus vaccines due to their ability to infect and replicate in cells.

Using an inactivated PRRS virus vaccine has several safety-related advantages, including no shedding of vaccine virus and no possibility of reversion to virulence. In the field, efficacy of either the inactivated or the MLV vaccine has been difficult to confirm. In the U.S., most PRRS virus vaccination is performed in the breeding herd and vaccination of sows with either inactivated virus or MLV is done repeatedly. Accordingly, these vaccines are mainly used to booster pre-existing immunity to wild type virus. Vaccine use in young pigs is low. The NAHMS 2000 study reported that only 6.4 percent of pigs in the survey were vaccinated for PRRS virus.

Studies evaluating the immune responses, as well as ability of the inactivated virus vaccine to produce PRRS virus-negative pigs from PRRS virus-positive sows, have been described. The immune response induced by the inactivated virus vaccine differs from that of the MLV product. Two of the studies reported here suggested that repeated vaccination with the MLV vaccine does not booster the immune response to PRRS virus. This lack of an anamnestic or recall response suggests that repeated chronic exposure to PRRS virus antigen through MLV vaccination may diminish the ability of lymphocytes to respond to the virus. However, the studies described here did not definitively demonstrate a complete lack of lymphocyte response to PRRS virus antigen. The efficacy of a vaccine cannot be determined by measurement of an immune response alone. Ultimately, challenge studies are required to determine the ability of a vaccine to induce protection against clinical disease. The results of the studies reported here highlight the need for more investigation of the immune responses induced by chronic exposure to wild-type virus and repeated vaccination. Increased understanding of the immune responses induced by repeated vaccination is required for not only PRRS virus but other pathogens, as well, in order to develop appropriate strategies for controlling disease in breeding herds where animals can be kept as long as 4 to 5 years.

References


These approaches and techniques have not necessarily been rigorously tested under scientifically valid conditions and may not be appropriate for all herds. Therefore, readers are strongly encouraged to fully consider the merits, drawbacks, and implications of these methods prior to applying them in the field. These views are solely those of the author, and are not approved by the National Pork Board.
Eradication Using Herd Closure
M Torremorell, S Henry, and WT Christianson

Introduction

PRRS virus infection can be costly, is difficult to control, and presents a major limitation to efficient production in intensive swine units. For these reasons, PRRS virus eradication strategies have recently received close attention from the U.S. swine industry. Various strategies have been described for PRRS eradication: total depopulation/repopulation, partial depopulation (Dee et al., 1993), Isovean® (Gramer et al., 1999) or segregated early weaning (Rajic et al., 2001), test and removal (Dee, 1998), mass vaccination with unidirectional pig flow (Dee and Philips, 1998) and herd closure are among the procedures that have been tried. In this section, we describe a procedure for PRRS eradication without depopulation based on herd closure. This procedure has also been described as PRRS elimination by roll over, flow-through, or normal attrition.

The success rate attributed to the herd closure program is estimated to be above 85 percent. The program should be implemented in fairly well isolated three site production systems where the sow herd can be left standing alone. Implementation of this program in continuous flow, farrow-to-finish farms, or in farms with poor and inconsistent gilt exposure programs may result in failure.

Control vs. Eradication

In PRRS virus control, the objective is to limit virus damage in the various stages of production. This is primarily achieved through management steps involving the gilt pool. Serologically positive or negative replacements are exposed to PRRS virus in the acclimatization or isolation unit and are allowed to recover from infection. These animals are then introduced into the breeding herd after they become immune, i.e., after when they are no longer viremic and a source of infection to herd mates. Therefore, replacement animals are introduced into the sow farm as PRRS virus seropositive animals.

In PRRS virus elimination, the objective is to remove the virus from infected herds. The virus must be eliminated from all stages of production in order to consider a production system negative and the system must demonstrate its negative status over time. With this objective, it is imperative to have negative gilts and boars available from the source farm(s) as replacements.

Herd Closure vs. Closed herd

Herd closure, or closing a farm, refers to a period of time during which replacement animals are not introduced. Closure applies to both internal replacements and replacements purchased from outside. An interruption of this type is an essential part of the PRRS virus eradication program described in this section. In a closed-herd system, replacements are produced internally and are introduced into the sow herd directly from the grower or finisher independently of their PRRS virus infection status. In general, “closed-herd systems” do not achieve PRRS virus elimination, although they do bring a measure of disease control because replacements usually have prior exposure to pathogens circulating in the herd.

Overview of the Process

Naïve, seronegative replacement animals are introduced into seropositive breeding herds when virus transmission has ceased. These negative animals replace the seropositive herd through normal attrition or by scheduled culling of the previously infected animals. This strategy results in a negative population of breeding animals over time.

To begin, a general evaluation of the farm or system determines whether PRRSV eradication is needed for the farm, whether it can be done, and if so, if it is economically practical.

Location - The farm should be sufficiently isolated from other farms so as to limit the risk of reinfection after the program is complete.

On-farm biosecurity - Strict on-farm biosecurity measures are needed to prevent the introduction of new PRRS viruses and to prevent movement of the virus within the farm or system.

Negative source of semen - Only semen from a routinely monitored PRRSV negative boar stud is used.
Replacement animals - Only naïve, seronegative replacements are used. Availability of an isolation area to hold and test the replacements is mandatory.

Transport - It is important to have a good understanding of how pigs are moved within the system and how the trucks are cleaned and disinfected. Special attention should be paid to cull and slaughter trucks.

Type of production system - Farrow-to-finish farms are at a disadvantage when using herd closure because of the risk of PRRS virus spreading from grower animals in proximity to the breeding herd.

The Principles Behind the Process

Evidence to support the herd closure strategies comes from field observations showing that, in closed populations, viral infections can be naturally eliminated over time (Freese and Joo, 1994; Harris et al., 1987; Torremorell et al., 2002). The principles supporting PRRS virus eradication by this method were recently summarized (Torremorell et al., 2000). These principles are summarized as follows:

Immunity. Pigs previously infected with, and recovered from, PRRS virus are immune to experimental, homologous challenge for an extended period of time. This suggests that homologous immunity is protective (Lager et al., 1997). In addition, preliminary data suggests that animals with a strong cellular immune response no longer harbor infectious virus (Meier et al., 2000). Importantly, this immunity may take up to six months to fully develop (Meier et al., 2000); thus, the need to close the herds for an extended period of time. The purpose of closure is to allow adequate time for such immunity to develop in all the adult animals.

Biosecurity/transmission. At the beginning of a PRRS virus elimination project, populations of pigs with differing immune status (immune vs. susceptible) coexist in the herd. During this time, it is essential to identify all possible sources of virus within the farm and to establish optimal biosecurity measures between the different populations to accommodate pig flow and prevent transmission. Biosecurity measures to prevent the possible introduction of new viral strains need to be recognized and strengthened.

Assessment and definition of the population. Defining the dynamics of the viral infection as it occurs within the farm is a prerequisite to implementing biosecurity and stopping transmission. It is important to identify the populations in which the virus circulates, the age at which pigs become infected, and the serologic and infection status of current replacements. From this information, it can be determined whether animal flow can be adequately managed to allow the needed segregation.

Replacement animal introduction. A consistent and dependable source of negative replacement animals and semen is required.

Sentinels as biologic indicators of infection. PRRS virus-naïve, seronegative sentinel animals can be used as biologic indicators to verify that virus is no longer circulating on the farm. Certainty in determining the time for safely beginning the introduction of negative replacements relies on the testing results from sentinels and the demonstration of continued freedom from PRRS virus infection. Different sentinel programs exist, but basically they can be grouped in two strategies: 1) sentinel animals can be mixed in an off-site location with weaned or cull sows and young gilts to assess seroconversion after proper contact; 2) vasectomized heat check boars can also be used as sentinels. In this case, by allowing the vasectomized boars to perform the daily heat detection activities, exposure to previously infected animals is assured. In some systems, the first groups of negative gilts may also be used as the sentinels.

Attrition effects on sow population. All previously exposed and/or infected animals need to be removed as the elimination process progresses. Whether removal of previously infected pigs is done by test-and-removal, accelerated culling based on age, or herd closure, the fact is that previously exposed animals are a risk factor for transmitting infection to susceptible animals.
Implementation of Elimination by Herd Closure

At this time, although elimination by herd closure is attractive, data is lacking to recommend this method over others. However, clinical experiences indicate that this method may be a safe, effective, cost efficient method to achieve successful PRRS virus elimination.

As discussed previously, candidate farm must stand alone, i.e., not house any growing animals other than nursing pigs. Three-site farms are good candidates. Farrow-to-finish farms face the problem of active virus infection in the growing pig population and the risk of re-introducing the virus into the negative breeding herd. A summary of the timeline involved in the implementation of the PRRSV elimination program by herd closure is provided in Table 1.

1. The first step in elimination by herd closure is to ensure that all reproductive animals undergo PRRS virus infection and recovery. This is achieved by managing the gilt pool and exposing replacement animals to PRRS virus in the isolation/quarantine area prior to introduction in the breeding herd. This step is critical since it will create a population of immune animals. In addition this step is considered preliminary to the eradication program and it is part of achieving control to PRRS virus infection.

2. The second step is to close the farm to the introduction of replacement animals. As a rule of thumb, the farm should be closed for a minimum of six months, although this will depend on the production flexibility of the farm and the clinical situation. Methods to decrease the period of closure are discussed in item c (below). In most cases, the period of closure will be longer. By closing the farm, naturally developing immunity eliminates virus infection from the herd.

To manage a period without animal introductions and, simultaneously, minimize the costs associated with the program, off-site breeding of negative replacements should be considered. If replacements are not available, breeding targets, parity structure, and overall production will be affected. In order to minimize the cost associated with the interruption of gilt introduction, three strategies can be implemented:

a. Use off-site breeding. This strategy will allow the farm to hit breeding targets in an off-site location. The pregnant gilts will be introduced into the sow farm at farrowing or late in gestation. The extra costs incurred are the lease of an off-site facility, added labor, and additional animal transport. The breeding project requires negative gilts, thus at the end of the closure period, production is resumed with negative replacement animals.

The economic and operational differences between this and total repopulation are several. Fewer gilts are needed for herd closure; essentially the same number of gilts that would be used in normal flow are required overall. Younger parity animals are not being culled (wasted) and normal replacement flow remains the same. An external site will have to be rented and, although breeding labor can be done by personnel already present on site one, there will be increased labor costs for the project. In contrast to depopulation, downtime costs are avoided and an extensive clean-up procedure on the sow farm is not required.

b. Add extra replacements to the sow herd prior to closure. This is recommended in systems where virus is actively circulating in the sow herd as, for instance, immediately following an outbreak. These replacements can be exposed and develop immunity to the virus while the sow farm is closed. This strategy may not work in situations where the sow farm is considered very stable and virus circulation is minimal, as the infection of replacement infection cannot be assured. This strategy mimics TGE elimination procedures (Harris et al., 1987) and requires active, on-going infection at the time the process is initiated.

c. Use replacements infected in the nursery. Farms in which replacements were infected as nursery-aged pigs have an additional option. A consistent nursery infection and recovery pattern, accompanied by freedom from clinical signs and verified by serologic monitoring, may reduce the closure time to 2 to 4 months. This can be achieved by changing the gilt introduction schedule to quarterly introductions. The decision to implement a shorter closure period depends on the number of viral strains in the herd, the sow farm stability, the acclimatization program, and the farm’s biosecurity.

3. Select a source of negative replacements. After initiation of an elimination project, all future replacements must be negative when
introduced into the sow farm. The semen source must also be negative.

4. **Introduce negative replacements into the sow farm.** The initial introduction of naïve, seronegative, animals into the sow farm represents the point of greatest risk in the process. The degree of risk at this stage is dependent upon the degree of assurance that virus circulation has ceased. Unfortunately, it is difficult to determine with certainty that virus circulation has stopped because laboratory techniques applied to large swine populations are not sufficiently sensitive to absolutely eliminate the possibility of virus transmission. One way to limit this risk is by using naïve, seronegative, sentinel animals prior to the introduction of negative replacements. Sentinel animals should be commingled with seropositive sows and gilts in a separate facility to determine whether virus is still being shed.

The replacement animals that entered immediately prior to the closure of the herd were, of course, PRRS positive and, therefore, the animals most likely to remain infected. Since they present the greatest risk of shedding virus, careful measures should be taken to segregate them from the naïve replacements for the longest time possible.

At farrowing, farrowing seropositive and seronegative gilts in the same rooms should be avoided. Additionally, cross-fostering of piglets between these two populations should also be avoided. This will be achieved by delaying breeding of the negative replacements for at least three weeks after the last positive gilt was bred.

5. **Eliminate PRRS virus from the growing pigs.** This is the last step in the process and requires depopulation of the nursery. This, in turn, will create an “empty bubble” that will be moved through the finisher leaving a population of PRRS negative pigs behind it. Vigorous cleaning and disinfection is mandatory. Virus elimination in the pig flow should not be attempted at the beginning of the eradication program; it should be performed towards the end, if not after, herd closure and when there are indications that the pig flow will remain negative.

6. **Eliminate exposed adult animals through normal attrition.** Removal of previously exposed animals is by the normal, or in some cases accelerated, culling of all seropositive females present at the time the farm was closed. This makes herd closure very attractive, since it allows for the preservation of the breeding herd, its economic value, and genetics. Overall, the program optimizes the value of herd age and productivity and minimizes the costs associated with premature removal of previously infected animals.

Throughout the process, routine serologic monitoring is required: sentinels at the beginning of the program and, after the introduction of naïve replacements, the negative breeding stock and the growing pig flow. Monitoring in the production flow is best performed at least on a monthly basis and with adequate statistical power to detect infection if present. Monitoring should be continued on an on-going basis. The purpose of monitoring is to confirm that the farm has become, and then remains, seronegative.

**Summary**

Although it is recognized that PRRS virus can be eradicated from farms, most of the PRRS elimination programs that have been described were implemented in farms associated with breeding stock companies. In general, these farms have good biosecurity programs and are located in low-density pig areas. Without any doubt, these circumstances have also contributed to the success of the programs. The question remains whether we can keep farms negative in large commercial production systems or in high-density pig areas. So far, it has been very difficult to keep farms negative that are part of larger commercial systems. In production systems, a system-wide approach versus a single-farm approach needs to be explored. Ultimately, the adoption of PRRS virus elimination programs on a large scale by swine producers will depend on successfully demonstrating the ability to keep farms negative after they are cleaned up.

**References**


These approaches and techniques have not necessarily been rigorously tested under scientifically valid conditions and may not be appropriate for all herds. Therefore, readers are strongly encouraged to fully consider the merits, drawbacks, and implications of these methods prior to applying them in the field. These views are solely those of the author, and are not approved by the National Pork Board.


