IMPACT OF THE IMPLEMENTATION OF THE MCREBEL MANAGEMENT PROGRAM IN A COMMERCIAL BREED-TO-WEAN UNIT FOLLOWING AN INFECTION WITH PORCINE REPRODUCTIVE AND RESPIRATORY SYNDROME

BY

JENNY REBEKAH MORRIS

THESIS

Submitted in partial fulfillment of the requirements
for the degree of Master of Science in Animal Sciences
in the Graduate College of the
University of Illinois at Urbana-Champaign, 2018

Urbana, Illinois

Advisor:

Professor Mike Ellis

Abstract

The objective of this study was to determine the effects of the implementation of the McRebel management program on pre-weaning mortality and timing of increase in the percentage of litters testing negative for the Porcine Reproductive and Respiratory Syndrome (PRRS) virus under commercial conditions. The study was carried out at a breed-to-wean facility that had recently tested positive for PRRS 1-7-4 wild type strain. The study used a randomized complete block design (blocking factor was farrowing date) with 2 treatments: 1) McRebel program (involved no cross-fostering with the implementation of additional biosecurity measures to eliminate cross-contamination); 2) Control (cross-fostering according to standard commercial procedures for PRRS negative farms and no additional biosecurity procedures). A total of 4,238 litters housed in 109 rooms forming 54 complete replicates were allotted onto the trial over nine weeks. Room was the experimental unit and a replicate consisted of 2 rooms; treatments were randomly allotted to room. Litter performance measurements were collected from the sow cards that recorded barn, room, crate, parity, sow identification, number piglets born alive and number of piglets weaned. Fluid sampling for diagnostic testing were collected from litters at processing for the McRebel treatment beginning at week 6 of the study; blood sampling for diagnostic testing were collected from litters at weaning for both treatments, beginning at week 7 of the study. The study was carried out for 10 weeks. A total of 18 fluid samples and 240 blood samples for diagnostic testing were collected throughout the study. Litter performance and piglet pre-weaning mortality data for each room were tested for normality and analyzed using the PROC MIXED procedure of SAS. Diagnostic data were analyzed using the Chi-square test using the PROC FREQ procedure of SAS. Pre-weaning mortality was greater ($P < 0.05$) for the

McRebel than the Control treatment in several weeks of the study and for the overall study period (16.87 and 13.13%, respectively). The number of litters testing negative for PRRS did not follow the expected trend. The first fluid samples collected in week 6 of the study were all negative for PRRS for both treatments. Consequently, collection of blood samples at weaning started at week 7 of the study. At this time (week 7 of the study), the percentage of litters testing negative for PRRS was greater ($P < 0.05$) for the McRebel than the Control treatment. However, for the remainder of the study period (weeks 8-10) there was no difference ($P > 0.05$) between the 2 treatments for the percentage of litters testing negative for PRRS. Overall, the results of this study suggest that implementing McRebel procedures does increase pre-weaning mortality by 3.74%. The pigs on this study on both treatments became PRRS negative much earlier than expected after the initial infection, therefore, there is a need to repeat the study. In addition, future studies should initiate testing for PRRS much earlier than in the current experiment.

Key words: PRRS, pre-weaning mortality, McRebel

Table of Contents

Chapter I: Literature Review

Porcine Reproductive and Respiratory Syndrome (PRRS) is a virus that is widespread in the US swine industry which affects all stages of production with an estimated impact of $560 million annually (Rowland et al., 1999). One of the main goals of a swine producer is to alleviate or prevent the impact of PRRS. Employing management strategies that reduce the spread of PRRS within a herd is one means to achieve this goal. Properly managing a breeding herd which has PRRS is critical to minimize the impact of the virus on production. When a herd has been infected with PRRS, the McRebel program (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses from PRRS) is normally implemented. This program is intended to eliminate the circulation of virus between pigs within the herd. The program includes the cessation of cross-fostering (McCaw, 1995); however, cross-fostering is a management approach that is used to decrease piglet mortality (Neat et al., 1991). Virus transmission is particularly high immediately after a PRRS outbreak (Albina, 1997) when all pigs are initially exposed to the virus. At this time, the implementation of McRebel may have limited impact on the transfer of the virus between pigs and continuation with cross-fostering could reduce piglet mortality. This chapter will review the literature relating to the McRebel program and the component parts.

Porcine Reproductive and Respiratory Syndrome

Origination and Clinical Signs: Porcine Reproductive and Respiratory Syndrome appeared in the United States in 1987 and in Europe in 1988. The origin of the virus is unknown, however, it has similar properties, including persistent viremia and infection, to equine arteritis virus and simian hemorrhagic fever virus (Plagemann and Moennig, 1992). The structure of PRRS is a single-stranded RNA virus in the *Arterivirus* genus. The virus is encased in an envelope which

gives it the ability to live without a host and not be affected by temperature, pH, or some cleaning agents (Cho et al., 2006). The virus can survive without a host for up to four months (Benfield et al., 1992). The virus has two different genotypes: European (Lelystad) and North American, however, disease severity is affected by isolate type (Cho et al., 2006). Halbur et al. (1995) reported that different isolates of PRRS generate different symptoms, rectal temperatures, and histological lung lesions in affected pigs.

There are two classifications of contagious diseases: endemic and epidemic. Epidemic diseases cause an outbreak with high infection rates and eventually an immunity is established, whereas an endemic disease is common and consistently present in a herd. PRRS has characteristics of both of these contagious disease characteristics. Specifically, PRRS is classified as epidemic in terms of reproductive symptoms and as endemic in terms of respiratory symptoms (Blaha, 2000).

The clinical symptoms of PRRS are variable and differ depending on prior exposure and virulence. In fact, some herds that are infected with PRRS do not present any clinical symptoms (Meredith, 1994) and production remains within a normal range (Zimmerman et al., 1997). Clinical symptoms for PRRS can be categorized by illness, secondary pathogens, or effects on reproduction. Clinical symptoms of the illness include fever, blue ears, lack of appetite, lethargy, and respiratory distress. Secondary clinical symptoms that can occur include an increase in respiratory diseases such as *Mycoplasma* and pneumonia, and mortality. Secondary pathogens are commonly seen in PRRS positive pigs due to a weakened immune system. Clinical symptoms of reproductive problems include increased abortions, still-births, mummified piglets, and premature farrowing (Meredith, 1994).

Pathogenesis: The PRRS virus replicates on alveolar macrophages on the lungs and tonsils. PRRS virus intercepts and prevents the response of neutralizing antibodies and interferons. A neutralizing antibody uses a mechanism to defend a cell from infection by neutralizing harmful biological effects of disease. Interferons are signal proteins that alert nearby cells when infection is present. Since PRRS decreases both of these signals, in some cases PRRS is not recognized in the body for three to four months. PRRS is able to invade the cell and secondary pathogens have an increased opportunity to infect the cell. Pathogenesis can also affect the fetus. PRRS can cross the placenta and infect fetuses after ninety days into gestation (Lunney et al., 2010).

Persistence is a term that is used to describe PRRS pathogenesis, however, the PRRS pathogenesis mechanism remains unknown (Rowland et al., 1999). Persistence refers to the virus levels in the animal being low for long periods and then decreasing with time (Cho et al., 2006). Lunney et al. (2010) reported that the virus has been infectious up to ninety-eight days post-infection, and that a pig is able to eliminate the virus by day 200 post-infection. A study by Horter et al. (2002) reported that of 60 pigs that were inoculated with PRRS virus 100% were PRRS positive until 63 days after inoculation and 90% were PRRS positive at 105 days after inoculation which confirms that PRRS remains infectious for an extended period of time. This is in agreement with a study by Pijoan et al. (1994) that reported that the virus can persist in a herd for 6 months to 2 years depending on the replacement rate used.

There are two immune response mechanisms that are exhibited by an animal: innate and adaptive. Innate immune response is the first defense mechanism utilized by the body. Adaptive immune response is an antigen-specific defensive response (Salak-Johnson and McGlone, 2007). There are two adaptive immune responses to a virus. First is humoral immunity which utilizes B-lymphoctyes. A humoral immunity occurs when the virus has not yet entered the cells. An

antibody will be produced from a humoral response which neutralizes disease. The second is cell-mediated immunity which utilizes T-lymphocytes (Bandrick et al., 2011). Cell-mediated immunity occurs once the virus has crossed the cell barrier and is within the cell. The response involves phagocytosis and, when necessary, apoptosis. Phagocytosis involves destruction of the virus, and apoptosis involves destruction of an entire cell which has been infected by a virus. The objective of these two immune response mechanisms is to remove the disease from the host by any means possible. Both responses are important in PRRS infection (Mateau et al., 2008). Piglets can receive humoral immunity from any sow, but only cell-mediated immunity from the birth sow. Cross-fostered piglets will be limited in cell-mediated immunity if moved within the first 24 hours after birth, which is why most piglet movement occurs after this time.

Virus Transmission: PRRS transmission can occur either by direct animal to animal contact, aerosol, or indirect contact (Albina, 1997; Wagstrom et al., 2001). Direct contact occurs when an infected animal makes contact with a susceptible animal. A study by Wagstrom et al. (2001) reported that sows inoculated with either the PRRS virus or a modified live virus can shed the virus through milk secretions. Transmission can occur from infected semen because the virus can stay viable for up to thirty-five days according to Albina (1997). Wills et al. (1997) reported that PRRS was not detectable in the feces after day 55 post-inoculation and up to day 124 post-inoculation which is when the trial ended.

Aerosol transmission can occur when animals are in close proximity with one another, however, there is disagreement on the importance of PRRS aerosol transmission (Albina, 1997). A study by Le Poitier et al. (1995) found that 45% of farms within 500 meters from a PRRS outbreak also became infected, whereas only 2% of farms became infected that were between 500 meters and up to 2 kilometers from the PRRS outbreak. In contrast, Otake et al. (2002)

conducted a field study where pigs in a barn were inoculated with PRRS and two trailers with sentinel pigs were placed outside of the barn near the exhaust fans. One trailer was placed 1 meter from exhaust fans and the other trailer was placed 30 meters from exhaust fans on the other side of the building. The trailers remained in position for 72 hours. The sentinel pigs in both trailers did not test positive for PRRS.

Indirect contact can come from objects that carry infection (fomites), such as boots, vehicles, and wildlife. Studies indicate that fomites can be decreased by keeping farm-specific boots on site, implementing foot baths between rooms, changing gloves between litters, and double-bagging items coming into the farm (Cho et al., 2006). Transportation vehicles can also indirectly transmit PRRS, however, transportation vehicles are crucial in the swine industry. Dee et al. (2004) conducted a study to determine the appropriate method to decrease the transmission of PRRS by transportation vehicles. There were four treatments relating to the cleaning of the trailer between loads: 1) removing wood shavings from the trailer, 2) removing wood shavings, washing, and disinfecting the trailer, 3) as treatment 2 with the addition of freezing and thawing the trailer, 4) removing wood shavings, washing, disinfecting, and drying the trailer. A total of ten swabs for PRRS testing were taken on the trailer before and after treatment implementation. The fourth treatment was the only treatment to significantly reduce the number of PRRS positive swabs.

Virus Circulation: PRRS virus circulation is decreased when there is low pig density, decreased animal movement, mild weather, and when new animals are quarantined before introduction into a farm. Once the virus enters a farm a high sero-prevalence will occur within three months. The virus circulates throughout the herd on average for sixteen months. Circulation occurs for an extended period of time because the initial infection may not reach all pigs within the herd at the

same time. Therefore, not all pigs will seroconvert at the same time. Negative pigs may convert at a later time prolonging the presence of the virus. Another issue is shedding time. Some pigs can shed the virus for up to three months, therefore, introducing new animals into the herd too soon can prolong the presence of the virus in the herd (Albina, 1997).

Economic Loss: PRRS is one of the most costly diseases that producers have to deal with. Estimates suggest that the annual cost of PRRS to the US swine industry is $560 million (Rowland et al., 1999). PRRS affects all parts of the swine production process, both breed to wean and wean to market. The increased costs of PRRS in both parts are associated with increased labor, veterinary costs, and the costs of the additional biosecurity measures that are applied. At breed-to-wean farms, there is a decrease in the number of pigs weaned and an increase in sow mortality and culling (DiPietre and Mulberry, 2017). An analysis carried out by Pejsak and Markowska-Daniel (1997) found that production costs increased because the prevention and treatment expenses associated with PRRS were 60% greater than before the herd became PRRS positive. An additional analysis by Neumann et al. (2005) estimated that the cost for PRRS affecting a breed to wean facility would be $74.16 per litter.

At wean to market farms, there is a decrease in average daily feed intake (ADFI), average daily gain (ADG), and feed efficiency. A decrease in ADFI results in a greater proportion of the feed consumed being used to meet maintenance requirements instead of for growth. A decrease in growth rate lengthens the time for pigs to reach market weight and decreases the overall annual output from a facility. Decreased feed efficiency can have a major impact on costs since feed is the most expensive cost in production (Gabler et al., 2013). Mortality also increases when PRRS is present. DiPietre and Mulberry (2017) suggested that wean-to-market mortality

can increase from 5 to 15% resulting in a decrease in the weight of pork produced, however, the greatest impact on cost was increased time to market weight.

A study by Holtkamp et al. (2012) analyzed the net present value of individual herds to evaluate the cost of PRRS. Net present value is the difference between the present costs of production (labor, feed, etc.) and the present value of the pigs produced. Net present value was estimated by the number of months that were required to reach the break-even point for a herd infected with PRRS that implemented an elimination program. The elimination program treatments that were compared involved complete depopulation and repopulation, and herd closure. When herds had high costs associated with implementing an elimination program, herd closure required 6 months and complete depopulation and repopulation required 25 months until the break-even point was reached.

McRebel Procedures

McRebel Overview: The McRebel (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses from PRRS) management program is a set of procedures which are aimed at reducing the spread of the PRRS virus within the sow farm and that allows the farm to return to PRRS negative status. McRebel has three major components comprised of: 1) eliminating cross-fostering; 2) eliminating contact with other litters (i.e. via needles, gloves etc.) and; 3) euthanizing weak piglets. McRebel recommendations require that piglets remain with their birth sow; the exception is if a sow dies, in which case the entire litter can be moved to a nurse sow, but not mixed with piglets from other litters (McCaw, 1995).

McRebel is based on the assumption that the biggest impact of a herd being PRRS positive is increased susceptibility to secondary infection. Therefore, keeping piglets within the birth litter decreases exposure to outside pathogens, decreases virus circulation, and can increase

growth (McCaw, 1995). At a commercial sow farm, pre-weaning mortality can increase during McRebel implementation due to lack of cross-fostering. However, the program can control nursery mortality due to reduction in exposure to secondary pathogens such as *Mycoplasma* and *Streptococcus suis* (McCaw, 2000).

Eliminating the spread of disease and, ultimately the elimination of the virus are the most important goals of the commercial sow farm so that the herd can be reopened to allow the introduction of new breeding stock. McRebel is most influential in herds where the PRRS virus is still present and circulating and the herd is not affected by commercial vaccines. One limitation of McRebel is that the procedures can be difficult to implement due to farm staff being unwilling to cease cross-fostering and increase euthanasia of piglets (McCaw, 1995).

Cross-Fostering: The McRebel program consists of a number of procedures including eliminating cross-fostering. One method to manage piglets in a commercial production setting is to practice cross-fostering, which involves moving piglets between birth litters. This practice is widely used in the swine industry. Straw et al. (1998) estimated that cross-fostering is practiced in 98% of herds in the Mid-west of the US. Heim et al. (2012) defined cross-fostering as, "the transference of piglets to equalize litter size according to birth weight, aiming at a reduction in pre-weaning mortality". The major restriction to this practice is that the number of piglets placed on a sow should be equal to or less than the number of functional teats on the sow. If there are more piglets than functional teats available not all piglets will be able to suckle leading to decreased growth and increased mortality. Producers practice cross-fostering early after the end of farrowing, normally within 24 hours of birth but up to a few days post-farrowing. Piglets experience significant competition for teats during the process of establishing teat order (Straw et al., 1998). Teat order refers to the arrangement of piglets on specific teats during suckling.

Piglets have been shown to favor a teat or location of teat depending on size of teat and birth weight of piglet (McBride, 1963). Straw et al. (1998) identified that teat order starts to be established around two days after farrowing and that piglets are aggressive with others who try to use the teat they prefer. This generally decreases the opportunity for smaller piglets to receive sufficient nutrients for maintenance and growth. Straw et al. (1998) showed that herds that fostered piglets by day 7 post-parturition had lower pre-weaning mortality compared to herds that continued to foster piglets after day 7 post-parturition (11.4 and 13.5%, respectively).

The importance of cross-fostering has increased recently due to the considerable increase in litter size that has occurred, largely as a result of genetic improvement of this trait (Ferrari et al., 2014). Larger litters have a lower average piglet birth weight but also greater within litter variation in birth weight (Ferrari et al., 2014). A study conducted by Quesnel et al. (2008) showed that litters with less than 9 piglets had an average birth weight of 1.88 kg, whereas litters with more than 16 piglets had an average birth weight of 1.38 kg. Quesnel et al. (2008) also reported that the coefficient of variation in birth weight for litters of less than 10 piglets and greater than 15 piglets was 15% and 24%, respectively. Based on this, the percentage of piglets with low birth weights will increase with litter size. Piglets with lower body weights are more likely to die pre-weaning and have decreased growth performance compared to heavier litter mates (Beaulieu, et al., 2010). A study by Ferrari et al. (2014) found that the highest incidence of mortality (28%) of the total pre-weaning mortality occurred within the first 24 hours post-farrowing with the most common reasons being low birth weight and starvation. This study also reported that sufficient colostrum intake in piglets can lead to decreased pre-weaning mortality.

Colostrum is a source of energy, aids in intestinal growth, and provides immunoglobulins to provide passive immunity. Piglets are born without plasma immunoglobulins and, therefore,

ingesting colostrum is crucial for piglets to develop immunity against common infections. Absorption of intact immunoglobulins across the gut occurs mainly in the first 12 hours post-farrowing (Ferrari, et al., 2014). However, piglets are normally not cross-fostered until approximately 24 hours after birth to allow colostrum intake, therefore, cross-fostering should have a limited effect on colostrum consumption (Ferrari et al., 2014).

Cross-fostering involves mixing of piglets from a number of litters which can increase exposure to disease (Wills et al., 1997; Kirkden et al., 2013). A common practice is for a producer to cross-foster some piglets more than once in order to minimize pre-weaning mortality. In addition, some producers will delay the weaning of smaller piglets and foster them onto other sows to allow them more time to grow before weaning. However, it has been shown that limiting piglet movement decreases the spread of the PRRS virus within a herd (Mason et al., 2014). This study analyzed the effect of cross-fostering (none compared to at 24 hours, or 5 or 10 days post-farrowing) on PRRS transmission at weaning. The study confirmed that cross-fostering at 10 days of age resulted in a significant increase of PRRS positive piglets at weaning by 8.8% among piglets compared to 1.4% at 5 days, 2.4% at 24 hours, and 4.2% with no cross-fostering.

Effects of Herd Closure: An important part of eliminating PRRS is to close the herd once it becomes PRRS positive. This means that no replacement animals are brought into the farm until piglets that are negative for PRRS are consistently produced (Torremorell et al., 2002). The goal is to return the unit to a PRRS-negative status as quickly as possible and producers will implement herd closure immediately after a break with PRRS as a means to reach that goal. Herd closure aims to eliminate introduction of a new PRRS infection into an infected herd and, thus, prevent further virus transmission to piglets (Schaefer, 2007).

It is common practice to vaccinate the entire herd with either a live or a modified-live virus to ensure all pigs have been exposed to the virus. Eventually, the virus infects all pigs and the pigs will cease to shed the virus around the same time. The animals all attain the same immunity level to the virus and since pigs are able to eliminate PRRS from the body the virus will eventually be eliminated from the farm (Schaefer, 2007). According to Torremorell et al. (2002) immunity can take up to six months for pigs to develop. The closure period typically can last for six to eight months and ends once all the piglets weaned are PRRS negative (Schaefer, 2007).

Herd closure has been shown to be successful for eliminating PRRS from a herd with, according to Linhares et al. (2012), a success rate of 85%. Other methods that can be used to eliminate PRRS from a unit are either partial or whole herd depopulation. In both of these instances, the farm will be depopulated of the PRRS positive animals and will purchase PRRS-negative replacement animals to repopulate the farm. Both methods are costly and do not ensure that the health status of the replacement animals is better than that before depopulation. Herd closure can decrease production temporarily since sows that die or are culled cannot be replaced (Schaefer, 2007). However, herd closure offers producers the ability to continue production with the sows already on site and to reopen the herd once PRRS-negative status is attained (Linhares et al., 2012).

Biosecurity: Disease is a major cause of economic and productive loss for swine producers. One method to reduce the opportunity for disease to enter into a herd and decrease the spread of diseases already present in the herd is to implement biosecurity practices (Armass et al., 1999). Armass et al. (1999) defines biosecurity as protection from the introduction and spreading of infectious agents (viral, bacterial, fungal or parasitic). Good biosecurity practices can maintain

a healthy herd status and eliminate disease from an already infected herd. Herd status refers to the disease activity in the herd (Lambert et al., 2009).

Biosecurity practices differ by herd and farm type because the spread of disease is affected by geographical location, pig density, time of year, supplies entering facility, and how semen and replacement gilts enter the farm. There are numerous opportunities to improve biosecurity, however, effectiveness and cost are factors in implementation (Holtkamp et al., 2010). Veterinarians and farm managers work closely together to put a biosecurity plan in place both before and after PRRS infection (Cornell and Kopcha, 2007). There are multiple biosecurity practices which can be implemented. A study by Holtkamp et al. (2010) determined several risk factors for disease entry into a unit that were manageable such as: average parity of females on site, serum testing of replacement animals, location of animal isolation, transportation vehicle cleaning procedures, and implementation of employee training . Another avenue for pigs to be exposed to disease is through organic matter. Removal of contaminated material and thorough cleaning and disinfection of the area is required to control the disease spread (Pritchard et al., 2005).

Introducing new animals into a facility risks bringing new diseases into a unit. Pigs can be a carrier for disease without symptoms being present. Stress (i.e. transportation, mixing) can activate disease and increase transmission (Pritchard et al., 2005). Replacement gilt acclimation is crucial to control the health status of the herd. Gilt acclimation involves exposing replacement gilts to the strains of the viruses present in the facility that they will be entering. The gilts will develop immunity and, therefore, eliminate the circulation of the virus within the herd (Corzo, et al., 2010). Two PRRS exposure methods reported by Corzo et al. (2010) include introducing gilts to nursery pigs that are positive for the PRRS strain present in the herd or by using vaccines.

Time to Stability/Time to Baseline Production: Porcine Reproductive and Respiratory Syndrome is costly to the producer due to a decrease in production. Since PRRS has a high prevalence in the United States, many producers and veterinarians focus on controlling rather than eliminating PRRS within a herd due to the fact that reinfection is likely (Brouwer et al., 1994). Methods used to assess if PRRS has been controlled in a unit is time to stability (time to produce PRRS negative pigs at weaning) and time to baseline production (time for the herd to return to the same production performance levels as before the PRRS infection). Linhares (2016) carried out an experiment in a PRRS positive herd to evaluate the effect of injecting pigs with two forms of PRRS virus, either a modified-live virus (MLV) or a live-virus inoculation (LVI), on the time to stability and time to baseline production. The LVI treatment reached time to stability seven weeks earlier than the MLV treatment (median of 25.1 and 32.0 weeks, respectively). However, the MLV treatment reached time to baseline production 11 weeks earlier than the LVI treatment (median of 10 and 21 weeks, respectively). Interestingly, the conclusion from this study was that using the MLV compared to the LVI reduced the time for the herd to return to baseline production levels. However, the opposite was the case for the time for the herd to reach stability, with the LVI treatments producing PRRS negative piglets at weaning earlier than the MLV treatment. The author concluded that the time to return to baseline production was the more economically important of the two measurements (Linhares, 2016).

Conclusions

- Porcine Reproductive and Respiratory Syndrome is common in the United States and can significantly decrease production. There is a need to understand effective methods to control and eliminate PRRS from a breeding herd.

- McRebel is one method used to decrease the spread of PRRS within a farm and return to the farm to a PRRS negative status. A major component of McRebel is elimination of cross-fostering.
- Cross-fostering is carried out to equalize piglet numbers and weights across litters to decrease pre-weaning mortality and is particularly useful with large litter sizes.
- Herd closure is commonly implemented once a breeding farm becomes PRRS positive allowing the farm to remain productive until the virus is eliminated. However, replacement animals are not brought into the farm and, therefore, the objective of the farm is to reopen the herd as quickly as possible.
- PRRS is persistent; can remain present in the animal for an extended period of time, however, the animal can eliminate the virus.
- PRRS costs the United States swine industry $560 million a year and also affects the global industry.

This review of the literature has highlighted that McRebel is an industry accepted set of procedures which are aimed at reducing the spread of the PRRS virus within the sow farm and, thus, allow the farm to return to PRRS negative status as soon as possible. A major component of these procedures is to eliminate cross-fostering of piglets between litters. Cross-fostering mixes piglets from multiple litters which can increase exposure to the PRRS virus. However, cross-fostering of piglets between litters within one day after birth is a standard management procedure that is carried out to align the number of piglets that the sow suckles with the number of functional teats on the sow. This approach has been shown to reduce pre-weaning mortality. The hypothesis behind the proposed study was that delaying the start of the implementation of the McRebel program for a period of time after a sow farm has become infected with PRRS will

result in a reduction in pre-weaning mortality (due to the continued use of cross-fostering) with little impact on the number of litters testing positive for PRRS. Therefore, the objective of this study was to determine the effects of the McRebel procedures on pre-weaning mortality and timing of increase in the percentage of litters testing negative for the PRRS virus.

Literature Cited

Albina, E. 1997. Epidemiology of porcine reproductive and respiratory syndrome (PRRS): an overview. Veterinary microbiology. 55(1): 309–316.

Armass, S.F. and L.K. Clark. 1999. Biosecurity considerations for pork production units. Swine Health Prod. 7 (5): 217–228.

Bandrick, M., M. Pieters, C. Pijoan, S.K. Baidoo, and T.W. Molitor. 2011. Effect of cross-fostering on transfer of maternal immunity to *Mycoplasma hyopneumoniae* to piglets. Vet. Rec. 168: 100.

Beaulieu, A.D., J.L. Aalhus, N.H. Williams, and J.F. Patience. 2010. Impact of piglet birth weight, birth order, and litter size on subsequent growth performance, carcass quality, muscle composition, and eating quality of pork. J Anim Sci. 88: 2767–2778.

Benfield, D.A., E. Nelson, J.E. Collins, L. Harris, S.M. Goyal, D. Robison, W.T. Christianson, R.B. Morrison, D. Gorcyca, and D. Chladek. 1992. Characterization of swine infertility and respiratory syndrome (SIRS) virus (isolate ATCC VR-2332). J Vet Diagn Invest. 4:127–33.

Blaha, T. 2000. The" colorful" epidemiology of PRRS. Veterinary research. 31(1): 77–83.

Brouwer J., K. Frankena, M.F. de Jong, R. Voets, A. Dijkhuizen, J. Verheijden, and R.E. Komijn. 1994. PRRS: effect on herd performance after initial infection and risk analysis. The Veterinary Quarterly. 16: 95–100.

Cho, J.G. and S.A. Dee. 2006. Porcine reproductive and respiratory syndrome virus. Theriogenology. 66(3): 655–662.

Cornell, K.K. and M. Kopcha. 2007. Client-veterinarian communication: Skills for client centered dialogue and shared decision making. Vet Clin North Am Small Anim Pract. 37: 37–47.

Corzo, C.A., E. Mondaca, S. Wayne, M. Torremorell, S. Dee, P. Davies, and R.B. Morrison. 2010. Control and elimination of porcine reproductive and respiratory syndrome virus. Virus Res. 154: 185–192.

Dee, S.A., J. Deen, S. Otake, and C. Pijoan. 2004. An assessment of transport vehicles as a source of porcine reproductive and respiratory syndrome virus transmission to susceptible pigs. Can J Vet Res. 68:124–33.

DiPietre, D. and L. Mulberry. The Economics of PRRS. Economists, KnowledgeVentures, LLC. [Online],; 2017. Available from: http://prrs.com.

Ferrari, C. V., P.E. Sbardella, M.L. Bernardi, M.L. Coutinho, I.S. Vaz Jr., J. Wentz, and F.P. Bortolozzo. 2014. Effect of birth weight and colostrum intake on mortality and performance of piglets after cross-fostering in sows of different parities. Prev. Vet. Med. 114: 259–266.

Gabler, N. K., W. Schweer, J.F. Patience, L. Karriker, J.C. Sparks, G. Gourley, M. FitzSimmons, K. Schwartz, and T.E. Burkey. 2013. The impact of PRRSV on feed efficiency, digestibility and tissue accretion in grow-finisher pigs. In Allen D. Leman Swine Conference. 40: 135–136.

Halbur P.G., P.S. Paul, X.J. Meng, M.A. Lum, J.J. Andrews, and J.A. Rathje. 1995. Comparison of the pathogenicity of two U.S. porcine reproductive and respiratory syndrome virus isolates with that of the Lelystad virus. Vet Pathol. 32: 648–60.

Heim, G., A.P.G. Mellagi, T. Bierhals, L.P. de Souza, H.C.C. de Fries, P. Piuco, E. Seidel, M.L. Bernardi, I. Wentz, and F.P. Bortolozzo. 2012. Effects of cross-fostering within 24 h after birth on pre-weaning behaviour, growth performance and survival rate of biological and adopted piglets. Livest. Sci. 150: 121–127.

Holtkamp, D. J., J.B. Kliebenstein, J.J. Zimmerman, E. Neumann, H. Rotto, T. Yoder, and C. Haley. 2012. Economic analysis of PRRS virus elimination from a herd. Animal Industry Report. 658(1): 10.

Holtkamp, D., D. Polson, C. Wang, and J. Melody. 2010. Quantifying risk and evaluating the relationship between external biosecurity factors and PRRS-negative herd survival. AASV. 109–113.

Horter, D.C., R.M. Pogranichiny, C.C. Chang, R.B. Evans, K.J. Yoon, and J.J. Zimmerman. 2002. Characterization of the carrier state in porcine reproductive and respiratory syndrome virus infection. Vet. Microbiol. 86: 213–228.

Kirkden, R.D., D.M. Broom, and I. L. Andersen. 2013. INVITED REVIEW: Piglet mortality: Management solutions. J. Anim. Sci. 91:3361–3389.

Lambert, M.È. and S. Dallaire. 2009. Biosecurity in swine production: Widespread concerns. Advances in Pork Production. 20: 139–148.

Le Poitier, M.F., P. Blanquefort, E. Morvan, and E. Albina. 1995. Results of a control program for PRRS in the French area 'Pays de Loire.' In Proceedings of the 2nd International Symposium on PRRS, Copenhagen, Denmark. 34.

Linhares, D. 2016. Factors associated with shorter time-to-stability and time-to-baseline-production. In Proc James D. McKean. 91–93.

Linhares, D., M. Torremorell, and R. Morrison. 2012. How long does it take for a breeding herd to produce PRRSv-negative piglets. In Proc Allen D. Leman Conf. 95–96.

Lunney, J.K., D.A. Benfield, and R.R. Rowland. 2010. Porcine reproductive and respiratory syndrome virus: an update on an emerging and re-emerging viral disease of swine. Virus Res. 154: 1–6.

Mason, B., M.F. Billing, and J. Seate. 2014. The effect of cross fostering on PRRS transmission and litter performance. AASV. 117–119.

Mateu, E. and I. Diaz. 2008. The challenge of PRRS immunology. The Veterinary Journal. 177(3): 345–351.

McBride, G. 1963. The “teat order” and communication in young pigs. Anim. Behav. 11:53–56.

McCaw, M.B. 1995. MCREBEL PRRS: Management procedures for PRRS control in large herd nurseries. In Proc Allen D. Leman Conf. 22: 161–162.

McCaw, M.B. 2000. Effect of reducing crossfostering at birth on piglet mortality and performance during an acute outbreak of porcine reproductive and respiratory syndrome. Jf Sw. Hlth Prod. 8(1): 15–21.

Meredith, M.J. 1994. Porcine reproductive and respiratory syndrome (PRRS). Pig Disease Information Centre (PDIC). 1: 1–60.

Neal, S.M. and K.M. Irvin. 1991. The effect of crossfostering pigs on survival and growth. J. Anim. Sci. 69(1): 41–46.

Neumann, E., J. Kliebenstein, C. Johnson, J. Mabry, E. Bush, A. Seitzinger A.L. Green, and J.J. Zimmerman. 2005. Assessment of the economic impact of porcine reproductive and respiratory syndrome on swine production in the US. J Am Vet Med Assoc. 227: 385–392.

Otake, S., S.A. Dee, L. Jacobson, M. Torremorell, and C. Pijoan. 2002. Evaluation of aerosol transmission of porcine reproductive and respiratory syndrome virus under controlled field conditions. Vet Rec. 150:804–808.

Pejsak, Z. and I. Markowska-Daniel. 1997. Losses due to porcine reproductive and respiratory syndrome in a large swine farm. Comp Immunol Microbiol Infect Dis. 20:345–352.

Pijoan C., G. Solano, and J. Segalés. 1994. PRRS virus and secondary disease. In Proc Allen D. Leman Swine Conf. 21: 225–226.

Plagemann P.G.W. and V. Moennig. 1992. Lactate dehydrogenase-elevating virus, equine arteritis virus, and simian hemorrhagic fever virus: a new group of positive-stranded RNA viruses. Adv Virus Res. 41: 99–192.

Pritchard, G., I. Dennis, and J. Waddilove. 2005. Biosecurity: reducing disease risks to pig breeding herds. In Practice. 27(5): 230–237.

Quesnel, H., L. Brossard, A. Valancogne, and N. Quiniou. 2008. Influence of some sow characteristics on within-litter variation of piglet birth weight. Animal. 2(12): 1842–1849.

Rowland, R.R.R., M. Steffen, T. Ackerman, and D.A. Benfield. 1999. The evolution of porcine reproductive and respiratory syndrome virus: quasispecies and emergence of a virus subpopulation during infection of pigs with VR-2332. Virology. 259: 262–266.

Salak-Johnson, J. L. and J.J. McGlone. 2007. Making sense of apparently conflicting data: Stress and immunity in swine and cattle. J. Anim. Sci. 85: 81–88.

Schaefer, N. and R. Morrison. 2007. Effect on total pigs weaned of herd closure for elimination of porcine reproductive and respiratory syndrome virus. Jf Sw. Hlth Prod. 15(3): 152–155.

Straw, B.E., C.E. Dewey, and E.J. Burgi. 1998. Patterns of crossfostering and piglet mortality on commercial U.S. and Canadian swine farms. Prev. Vet. Med. 33: 83–89.

Torremorell, M., and W.T. Christianson. 2002. PRRS eradication by herd closure. Adv. Pork Prod. 13: 169–176.

Wagstrom, E.A., C.C. Chang, K.J. Yoon, and J.J. Zimmerman. 2001. Shedding of porcine reproductive and respiratory syndrome virus (PRRSV) in mammary secretions of sows. Am J Vet Res. 62:1876–80.

Wills, R., J.J. Zimmerman, K.J. Yoon, S.L. Swenson, M.J. McGinley, H.T. Hill, K.B. Platt, J. Christopher-Hennings, and E.A. Nelson. 1997. Porcine reproductive and respiratory syndrome: a persistent infection. Vet. Microbiol. 55: 231–240.

Wills, R.W., J.J. Zimmerman, K.J. Yoon, S.L. Swenson, L.J. Hoffman, M.J. McGinely, H.T. Hill, and K.B. Platt. 1997. Porcine reproductive and respiratory syndrome virus: routes of excretion. Vet Microbiol. 57:69–81.

Zimmerman, J.J., K.J. Yoon, R.W. Wills, and S.L. Swenson. 1997. General overview of PRRSV: a perspective from the United States. Vet. Microbiol. 55: 187–196.

Chapter II: Impact of the Implementation of the McRebel Management Program in a Commercial Breed-to-Wean Unit Following an Infection with Porcine Reproductive and Respiratory Syndrome

Introduction

Porcine Reproductive and Respiratory Syndrome (PRRS) is an infectious disease that can affect all stages of production and cause major economic losses to the global swine industry. There is not a cure for PRRS and there is much speculation on how to best eliminate the disease from a swine herd. The clinical symptoms of the disease are variable. The most common clinical symptoms are: reduced growth performance, increased secondary respiratory infections, and increased mummies, still-births, and weak piglets. One important property of PRRS is its persistence; PRRS can remain within a herd for extensive periods of time. Because of this persistence, the virus can be present in a herd at low levels for over 150 days (Cho et al., 2006).

Once a herd becomes PRRS positive, the goal of the swine producer is to return the herd to a PRRS negative status as quickly as possible in order for the herd to be reopened to stock introductions and production to return to normal. Producers have several options to choose from to try to achieve PRRS negative status including whole herd depopulation and repopulation, partial depopulation, and herd closure. With herd closure, no new replacement animals are brought into the herd until the herd becomes PRRS negative. In the United States, herd closure is most commonly implemented because of the cost benefits. Eliminating the introduction of any new animals will reduce the circulation of the virus within the herd and also will prevent the introduction of other secondary pathogens. This allows production to continue in the herd (Linhares et al., 2012).

One method to increase the rate of elimination of PRRS from the herd is to implement McRebel (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses from PRRS) procedures. These management procedures include the cessation of piglet movement between litters with all piglets remaining with their birth sow and litter (i.e., no cross-fostering is allowed). Also, additional biosecurity measures are put in place to eliminate cross-contamination. These procedures aim to end the circulation of the virus throughout the herd and also reduce clinical symptoms associated with PRRS (McCaw, 1995).

Cross-fostering of piglets between litters within one day after birth is a standard management procedure that is carried out to align the number of piglets that the sow suckles with the number of functional teats (Hein et al., 2012). Eliminating cross-fostering results in elevated pre-weaning mortality since piglets are disadvantaged if there are more piglets in the litter than the sow has functional teats (Straw et al., 1998).

Therefore, the objective of this research was to determine relationship between implementation of McRebel procedures on 1) pre-weaning mortality and; 2) timing of improvements in the percentage of litters testing negative for PRRS.

Materials and Methods

The study was conducted at the Maschhoff Pork Farm, a breed-to-wean facility, owned and operated by The Maschhoff's, LLC located near Carlyle, Illinois. Experimental protocols were approved by the University of Illinois Institutional Animal Care and Use Committee.

Experimental Design and Treatments: The study was carried out in a herd that experienced a recent PRRS infection to investigate the effects of implementing an epidemic disease management treatment (McRebel procedure). The source of the virus entering the herd is

unknown. The study was carried out as a Randomized Complete Block Design (RCBD) with farrowing date as the blocking factor and compared the following two treatments: McRebel procedure and Control. McRebel procedure included: No cross-fostering with additional biosecurity procedures used to prevent cross-contamination between litters. The Control treatment allowed cross-fostering between litters according to industry standards for PRRS negative farms but no additional biosecurity procedures. The additional biosecurity procedures implemented in the McRebel treatment were changing or disinfecting all equipment used between litters and no personnel entry into crates. Typical biosecurity procedures followed at this facility include: showering into facility, fumigating all items entering farm, required time before people who have visited other farms are allowed to enter, and quarantining animal 90 days before entry into farm.

Disease Status: The farm on which this study was carried out had previously experienced a PRRS infection with the 1-12-4 strain about 2 years prior to this most recent break with the disease. At the time of the previous infection, the herd was closed for 48 weeks. Previous history with this farm would suggest that it typically experiences a break with PRRS after approximately one year of being reopened. In the case of the recent PRRS infection that was the focus of this study, the farm manager reported that sows were off feed and that there had been an increase in the incidence of abortions. Subsequently, the farm was tested for PRRS with serum from 30 sows from various locations on the farm and collected within 2 days of the report of clinical signs. The farm tested positive for the 1-7-4 wild type strain of PRRS. Consequently, the study began two weeks later. The last group of replacement gilts was brought into the herd the week before PRRS was diagnosed; subsequently, no replacement gilts were brought into the herd during the study period. The whole herd received an Ingelvac PRRS MLV (Boehringer

Ingelheim Vetmedica GmbH, Ridgefield, NJ) vaccination one week after the PRRS diagnosis and again four weeks later. Prior to this study, the sows had been vaccinated with PRRS MLV and the herd was being operated as a vaccinated PRRS positive facility.

Facilities: The unit used for the study was a standard breed-to-weaning farm that held approximately 11,500 sows. It consisted of typical breeding, gestation, and farrowing buildings. Sows were weaned into the breeding facility which consisted of individual crates. They were bred using AI and were checked for pregnancy which was carried out at approximately day 30. After pregnancy confirmation, sows were moved to standard gestation crates. The breeding and gestation crates had solid concrete floors at the front half of the crate and slatted floors at the back half, a trough at the front, and a drop feeder. The floor space was 1.12 m^2 floor space per animal. Water was continuously available in the trough. Feed was provided daily and according to body condition score.

The farrowing facility, where sows were housed during farrowing and lactation, consisted of 7 buildings with a total of 55 rooms with 20 to 60 farrowing crates per room. The temperature in the room was maintained using a thermostat linked to an automatic ventilation system. The thermostat had set points that changed over the period that the sows were in the farrowing room. Initially when the sows were moved into the room, the thermostat temperature was set at 22.8 ºC and the setting was gradually decreased to 17.2 ºC by weaning.

The farrowing pens consisted of a farrowing crate located in the center of the pen and surrounding pen divisions. The dimensions for the sow area were 0.54 m x 1.95 m giving a total of 1.07 m^2 of floor space and for the piglets were 0.98 m x 2.04 m giving a total of 1.99 m^2 of floor space The floors were made of plastic and the farrowing crate was equipped with a feed trough, and a nipple-type water drinker located in the feed trough. Plastic mats were provided for

the piglets within the piglet area. Piglets were provided with a supplemental heat source via a heat lamp suspended over the plastic mat.

Animals and Allotment to Study: A total of 4,238 litters (sows) housed in 109 farrowing rooms were used in the study. The experimental unit was the farrowing room and a replicate was two farrowing rooms in close proximity within the building and housing sows with similar farrowing dates. Thus, there were 54 farrowing rooms on the Control treatment and 55 on the McRebel treatment.

Each farrowing room was clearly labeled with a color-coded treatment card and a copy of the protocol was displayed in each room. Sows were moved into a farrowing room at approximately day 110 of gestation. Prior to movement into farrowing rooms, sows were kept in gestation crates and managed according to the standard procedures of the company.

Farrowing Management: If sows had not farrowed by day 115 of gestation they were induced using 2 cc of Prostaglandin F-2α (Zoetis, Parsippany, NJ). Trained personnel monitored sows during the farrowing process and assisted with delivery of piglets as needed. Split-suckling was practiced on both treatments when trained personnel deemed necessary; the McRebel treatment remained with birth litter and equipment was replaced in order to allow to contamination from other litters. For the Control treatment cross-fostering was carried out within the first 24 hours after birth; there was no cross-fostering on the McRebel treatment with piglets remaining with their birth sow. Piglets on both treatments received 1 mL iron dextran (Pharmacosmos Inc, Watchung, NJ) and 0.5 mL Draxxin (Zoetis, Parsippany, NJ) via intramuscular injections at day 1 after birth. In addition, a second 1 mL injection with iron dextran (Pharmacosmos Inc, Watchung, NJ) was given at day 8 when castration and tail docking were also carried out. Piglets were weaned at approximately day 21 of age. Diets used during lactation were formulated to

meet or exceed the nutrient requirement proposed by the NRC (2012) for lactating sows. Sows were fed twice daily until farrowing occurred, and then given *ad-libitum* access to feed for the remainder of lactation. Parity 1 sows were given additional feed as a top dressing which was formulated to meet their amino acid requirements. Sows had *ad-libitum* access to water throughout their time in the farrowing facility.

Measurements: The sow cards that were maintained by the farm staff were used to collect the litter performance data. The information recorded on the sow cards included barn, room, crate numbers, parity, sow identification, number piglets born alive and weaned. Pre-weaning mortality was determined from the sow cards as the difference between the number of piglets born alive and the number weaned.

Starting 6 weeks from when the farm became PRRS positive (Figure 1), fluid was collected at processing from litters on the McRebel treatment only. On the day of processing (approximately day 8 after farrowing), ten percent of litters were sampled with half from parity 1 sows and the other half from sows of parity 2 or greater. Fluid collection at processing involved collection of tails and testicles from an entire litter and placing these in a bag which was suspended to allow fluid to drain. Fluid was collected for each litter and sent to the diagnostic laboratory at Iowa State University for determination of the presence of PRRS which was carried out using a polymerase chain reaction (PCR) assay. Fluid samples were collected each week over a 4-week period through week 9 of the study.

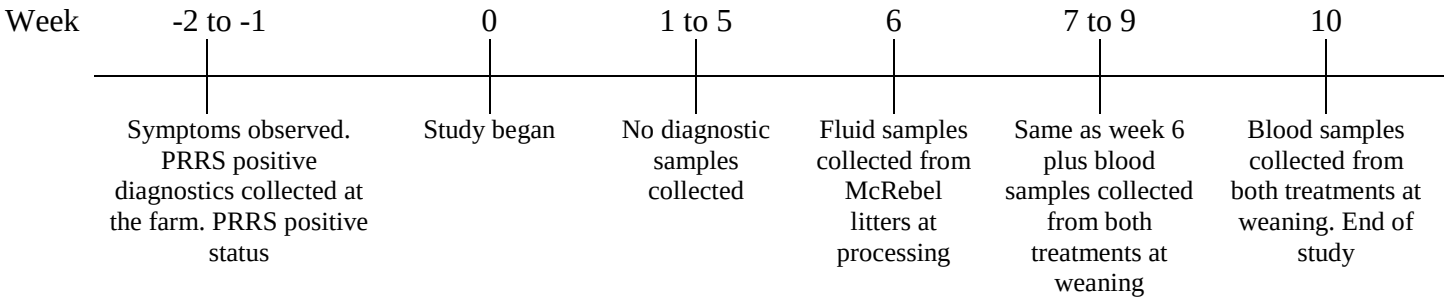
The original plan was that once ten percent of the litters tested negative for PRRS, blood sampling would be carried out at weaning to determine the PRRS status of piglets from both treatments. However, all of the fluid samples collected from litters on the McRebel treatment at week 6 tested negative for PRRS and consequently, collection of blood samples at weaning from

litters on both treatments was started in week 7 of the study. For the blood samples collected from the piglets on the day of weaning, twenty percent of litters were sampled with half being from parity 1 sows and the other half from sows of parity 2 or greater. Within each litter that was sampled, 2 piglets were randomly selected for blood sampling. Blood was collected from the ear vein and the samples from the two piglets were pooled. A PCR assay was carried out on the blood sampling from each litter at the diagnostic lab at Iowa State University. Blood sampling was carried out for 4 weeks until week 10 of the study when the study was terminated.

Statistical Analysis: Litter performance data from each room were tested for normality and homogeneity of variance using the PROC UNIVARIATE procedure of SAS (SAS Inst. Inc., Cary, NC). Data that were normally distributed were analyzed using the PROC MIXED procedure. The model used included the effects of treatment as a fixed effect, and replicate and farrowing date as random effects. Mortality and diagnostic data were analyzed using a Chi-square test using the PROC FREQ procedure of SAS. Means were considered different at $P \leq 0.05$.

Study Timeline:

Figure 1.



Results and Discussion

Litter Performance. For reference purposes, litter performance for all of the sows in the herd used in this study for the period from 6 weeks prior until 6 weeks after the PRRS infection was confirmed is presented in Appendix Table 1. There was considerable variation between weeks for litter performance measures and no clear trends in performance levels between the period before and after confirmation of the infection.

Least square means for the effect of McRebel treatment on litter performance are presented in Table 1. There were 4,238 sows on trial with 2,127 on the McRebel treatment and 2,111 on the Control treatment with a mean parity of 4.32 and 4.69, respectively. There was no difference ($P > 0.05$) in the number of piglets born alive per room between the two treatments. There was also no difference ($P > 0.05$) in the number of piglets weaned per room although the McRebel treatment was numerically lower compared to the Control treatment. Although the difference between the treatments was relatively small, it would be commercially important. These results suggest that implementing McRebel procedures (which involved no cross-fostering or additional biosecurity procedures) decreased the number of piglets weaned. Part, but not necessarily all, of the reduction in number weaned for the McRebel treatment could be due to not using cross-fostering which is a widely used practice aimed at reducing pre-weaning mortality. Most studies that have evaluated the effects of cross-fostering on pre-weaning mortality have shown positive results. For example, Arango et al. (2006) reported that cross-fostering decreased pre-weaning mortality by 10% compared to litters that had remained with the birth sow. Similarly, Neal et al. (1991) reported that pre-weaning mortality in cross-fostered litters compared to those that were not cross-fostered was 13.7% and 25.0%, respectively. However, Heim et al. (2012) found no effect of cross-fostering on pre-weaning mortality.

Pre-weaning mortality. Pre-weaning mortality was calculated for each week of the study after the PRRS infection was confirmed based on the number of piglets born alive and weaned from each room and these results are presented in Table 2. At each week post-infection the McRebel treatment had numerically higher pre-weaning mortality and several weeks had significantly higher ($P < 0.05$) pre-weaning mortality compared to the Control treatment. The total pre-weaning mortality for the trial was 16.87% for the McRebel treatment and 13.13% for the Control treatment. Therefore, these results suggest that postponing implementation of McRebel procedures would decrease pre-weaning mortality. There has been limited research to evaluate the impact of implementation of the McRebel program on sow and piglet performance pre-weaning. In contrast to the results of the current study, McCaw (2000) showed that implementation of McRebel procedures reduced pre-weaning mortality from 14.94 to 10.08%. As previously discussed, the McRebel program eliminates cross-fostering, as well as implementing additional biosecurity measures to eliminate contact between litters. Further research is needed to clearly establish the impact of implementation of McRebel program on pre-weaning mortality.

Diagnostic results. The first collection of fluids at processing to test for the PRRS virus began at week 6 after the start of the study and samples were only collected from litters on the McRebel treatment. When the study was originally designed, the expectation was that litters on the McRebel treatment would become PRRS negative before those on the Control treatment due to reduced cross-contamination between litters for the McRebel treatment. In addition, previous research had suggested that piglets can remain PRRS positive for up to 100 days post infection (Horter et al., 2002;McCaw, 2006). On this basis, it was decided to start fluid sampling to test for PRRS 6 weeks after the start of the study and, initially, only for the McRebel treatment. The

intention was to start sampling litters on the Control treatment as soon as 10% of samples collected from the McRebel treatment were PRRS negative.

The results of the diagnostic tests on the fluid and blood samples are presented in Table 3. All of the fluid samples collected at processing from litters on the McRebel treatment in weeks 6, 8, and 9 tested negative for PRRS, with 50% testing negative in week 7 (Table 3). This high incidence of PRRS negative litters relatively early after the initial infection was unexpected. Consequently, collection of blood samples at weaning for both treatments for diagnosis was started at week 7. Based on these blood samples, the percentage of litters that tested negative for PRRS was relatively high for both treatments in all weeks, ranging between approximately 78 and 90% (Table 3). The exception to this was for week 7 where the percentage of litters testing negative for PRRS was lower ($P < 0.05$) for the Control than the McRebel treatment (40 vs. 90%, respectively). Subsequently, for weeks 8 to 10 the percentage of litters testing negative for PRRS was similar ($P > 0.05$) for both treatments (Table 3). By the conclusion of the study (Week 10), 82.5% of samples on the McRebel treatment and 85.0% of the samples on the Control treatment were PRRS negative (Table 3). Prior to the start of this study the farm was being run as a vaccinated PRRS positive farm, results may differ at a naïve farm.

Conclusions

These results highlight one of the difficulties in carrying out research into the PRRS virus and also in developing control strategies to use under commercial conditions because of the inability to predict the transmission and development of the virus. The original objective of the study was to determine the most advantageous time to implement McRebel procedures after a site was infected with the PRRS virus. The idea was that if this time could be predicted then the implementation of the McRebel program could be delayed until then which should increase

overall numbers weaned compared to introducing the McRebel program immediately after the PRRS infection is confirmed which is the normal approach adopted on most units. Based on the historical literature and previous experience of the veterinary staff from this company, it was decided to delay the start of sample collection for diagnostic testing until week 6 post infection. However, by this time all of the litters on the McRebel treatment were PRRS negative. Thus, the study failed in relation to one of the major objectives and cannot be used to give recommendations about the optimum timing of implementation of the McRebel program. There is a need to repeat this study and to initiate diagnostic testing much earlier than was the case in the current study.

In conclusion, there is evidence from this study that implementing the McRebel program can increase pre-weaning mortality. However, the pigs on this study on both treatments became PRRS negative much earlier than expected after the initial infection. As a consequence, this study needs to be repeated to establish the optimum time to implement a McRebel program after a PRRS infection.

Tables

Table 1 Least square means for the effect of McRebel implementation on litter performance.

	Treatment			
Item	McRebel	Control	SEM	*P*-value
Parity[1]	4.32	4.69	.	.
Number of litters	2127	2111	.	.
Number of piglets per litter				
Born alive	12.58	12.37	0.010	0.14
Weaned	10.68	10.85	0.091	0.15

[1]Nurse sow parities not included.

Table 2. The effects of McRebel treatment on the incidence of pre-weaning mortality by week of study.

Item[1]	Treatment		SEM	*P*-value
	McRebel	Control		
Start-week 1				
Total number of rooms	3	3	.	.
Average number of pigs born alive	12.36	12.50	0.404	0.66
Average number of pigs weaned	10.61	11.14	0.242	0.26
Mortality, %	17.34	13.12	1.82	0.24
Week 1-week 2				
Total number of rooms	9	9	.	.
Average number of pigs born alive	12.40	12.45	0.266	0.90
Average number of pigs weaned	10.71	11.11	0.268	0.27
Mortality, %	15.13	12.87	1.69	0.37
Week 2-week 3				
Total number of rooms	5	5	.	.
Average number of pigs born alive	12.18	12.28	0.313	0.76
Average number of pigs weaned	10.04	10.23	0.284	0.60
Mortality, %	18.75	16.36	1.58	0.31
Week 3-week 4				
Total number of rooms	9	8	.	.
Average number of pigs born alive	12.84	12.80	0.248	0.91
Average number of pigs weaned	10.94	11.23	0.217	0.38
Mortality, %	16.82[a]	11.92[b]	1.35	0.03
Week 4-week 5				
Total number of rooms	5	5	.	.
Average number of pigs born alive	12.05	12.72	0.202	0.10
Average number of pigs weaned	10.82	10.50	0.207	0.23
Mortality, %	18.46[a]	12.91[b]	1.14	<0.01
Week 5-week 6				
Total number of rooms	9	10	.	.
Average number of pigs born alive	12.41	12.09	0.213	0.21
Average number of pigs weaned	10.56	10.69	0.159	0.60
Mortality, %	16.92[a]	12.90[b]	0.868	<0.01
Week 6-week 7				
Total number of rooms	5	5	.	.
Average number of pigs born alive	12.14	12.38	0.301	0.60
Average number of pigs weaned	10.06	10.41	0.239	0.36
Mortality, %	17.38	15.63	1.02	0.29
Week 7-week 8				
Total number of rooms	8	8	.	.
Average number of pigs born alive	13.29	12.54	0.298	0.12
Average number of pigs weaned	11.23	11.12	0.228	0.74

Table 2 (Cont)				
Mortality, %	16.46[a]	11.75[b]	1.02	<0.01
Week 8-week 9				
Total number of rooms	2	1	.	.
Average number of pigs born alive	12.24	12.27	.	.
Average number of pigs weaned	10.61	10.98	.	.
Mortality, %	15.90	12.30	.	.
Overall				
Total number of rooms	55	54	.	.
Average number of pigs born alive	12.58	12.39	0.099	0.18
Average number of pigs weaned	10.68	10.85	0.091	0.15
Mortality, %	16.87[a]	13.13[b]	0.476	<0.0001

[a,b]Means within a row with different superscripts differ ($P < 0.05$).

[1]Means were compared using the PROC MIXED procedure of SAS.

Table 3. The effects of McRebel treatment on the number of samples testing positive or negative for Porcine Reproductive and Respiratory Syndrome (PRRS)[1].

	McRebel			Control			
Item[2]	Positive	Negative	% Negative	Positive	Negative	% Negative	*P*-value
Processing[3]							
Week 6	0	6	100	.	.	.	.
Week 7[4]	2	2	50.0	.	.	.	.
Week 8	0	4	100	.	.	.	.
Week 9	0	4	100	.	.	.	.
Weaning[5]							
Week 7	1	9	90.0[a]	6	4	40.0[b]	0.02
Week 8	6	22	78.6	3	25	89.3	0.28
Week 9	7	35	83.3	4	38	90.5	0.33
Week 10	7	33	82.5	6	34	85.0	0.76

[a,b]Means within a row with different superscripts differ ($P < 0.05$).
[1]One sample was taken from each litter evaluated unless otherwise noted.
[2]All tests were conducted at Iowa State University diagnostic laboratory using polymerase chain reaction (PCR).
[3] Fluid samples were collected at processing.
[4]Samples from 9-13 litters were pooled.
[5] Blood samples were collected at weaning.

Supplementary Table 1. Summary of farrowing performance per litter by week of study.

	Week of Study												
Item	-6	-5	-4	-3	-2	-1	0*	1	2	3	4	5	6
Number Sows	631	632	674	583	581	531	591	535	601	504	575	588	550
Number Born	8678	8669	9290	8125	8127	7311	8039	7276	7930	6715	8004	7893	7302
Average	13.75	13.72	13.78	13.94	13.99	13.77	13.60	13.60	13.19	13.32	13.92	13.42	13.28
Number Live Born	8039	8098	8713	7479	7403	6623	7269	6672	7311	6299	7121	7246	6677
Average	12.74	12.81	12.93	12.83	12.74	12.47	12.30	12.47	12.16	12.50	12.38	12.32	12.14
Number Still Born	639	571	577	646	724	688	770	604	619	416	883	647	625
Average	1.01	0.90	0.86	1.11	1.25	1.30	1.30	1.13	1.03	0.83	1.54	1.10	1.14
Number Mummified	210	217	192	210	205	205	254	230	216	139	233	174	206
Average	0.33	0.34	0.28	0.36	0.35	0.39	0.43	0.43	0.36	0.28	0.41	0.30	0.37
Number Weaned	6761	6682	7057	5755	5511	5130	5485	5409	5656	5102	5554	5653	5158
Average	10.71	10.57	10.47	9.87	9.49	9.66	9.28	10.11	9.41	10.12	9.66	9.61	9.38

*Week 0 is start of study.

Literature Cited

Arango, J., I. Misztal, S. Tsuruta, M. Culbertson, J.W. Holl, and W. Herring. 2006. Genetic study of individual preweaning mortality and birth weight in Large White piglets using threshold-linear models. Livest. Sci. 101(1): 208–218.

Cho, J.G. and S.A. Dee. 2006. Porcine reproductive and respiratory syndrome virus. Theriogenology. 66(3): 655–662.

Heim, G., A.P.G. Mellagi, T. Bierhals, L.P. de Souza, H.C.C. de Fries, P. Piuco, E. Seidel, M.L. Bernardi, I. Wentz, and F.P. Bortolozzo. 2012. Effects of cross-fostering within 24 h after birth on pre-weaning behaviour, growth performance and survival rate of biological and adopted piglets. Livest. Sci. 150: 121–127.

Horter, D.C., R.M. Pogranichiny, C.C. Chang, R.B. Evans, K.J. Yoon, and J.J. Zimmerman. 2002. Characterization of the carrier state in porcine reproductive and respiratory syndrome virus infection. Vet. Microbiol. 86: 213–228.

Linhares, D., M. Torremorell, and R. Morrison. 2012. How long does it take for a breeding herd to produce PRRSv-negative piglets. In Proc Allen D. Leman Conf. 95–96.

McCaw, M.B. 1995. MCREBEL PRRS: Management procedures for PRRS control in large herd nurseries. In Proc Allen D. Leman Conf. 22: 161–162.

McCaw, M.B. 2000. Effect of reducing crossfostering at birth on piglet mortality and performance during an acute outbreak of porcine reproductive and respiratory syndrome. Jf Sw. Hlth Prod. 8(1): 15–21.

McCaw, M. 2006. Different approaches to handling PRRS. In Proc London Swine Conf. 21-33.

Neal, S.M. and K.M. Irvin. 1991. The effects of crossfostering pigs on survival and growth. J. Anim Sci.. 69(1): 41–46.

Straw, B.E., C.E. Dewey, and E.J. Burgi. 1998. Patterns of crossfostering and piglet mortality on commercial U.S. and Canadian swine farms. Prev. Vet. Med. 33: 83–89.