Effect of reducing crossfostering at birth on piglet mortality and performance during an acute outbreak of porcine reproductive and respiratory syndrome

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Summary

Objective: To determine whether limiting crossfostering even within 24 hours of birth was essential to the success of the **m**anagement **c**hanges to **r**educe **e**xposure to **b**acteria to **e**liminate **l**osses (McREBEL) protocol in a 1800-sow herd.

Methods: During the first 10 weeks after an outbreak of acute porcine reproductive and respiratory syndrome (PRRS) in a herd but prior to implementing McREBEL management, crossfostering was performed only within 24 hours of birth; however, because the manager was sizing and sexing all litters at birth, > 85% of piglets were being raised by a foster dam. Under the McREBEL protocol, crossfostering practices were changed so that only the minimal number of piglets needed to fill functional teats (fewer than 15%) were moved between litters.

Results: Preweaning and nursery mortality (which included euthanized culls) and weight at sale were all improved within 1 week of implementing the minimal-crossfostering protocol, although PRRS, manifested as early farrowings, weak-born piglets, increased mummies, and virus circulation among nursery pigs, continued for 9 subsequent weeks.

Implications: Crossfostering of piglets should be minimized even within the first 24 hours of age and throughout lactation during acute outbreaks of PRRS. Excellent production performance was achieved without vaccination, nursery depopulation, or waiting for virus circulation to end in the breeding herd and nursery. Continued minimal fostering between litters (McREBEL management) did not adversely affect production performance after the PRRS outbreak subsided.

Keywords: Porcine reproductive respiratory syndrome virus, PRRSV, McREBEL, crossfostering, mortality.

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One of the main challenges in an acute outbreak of porcine reproductive and respiratory syndrome (PRRS) is the need to control preweaning and nursery mortality. Vaccinating piglets is a recommended method for controlling PRRSV in nursery pigs.[1] Sow vaccination has also been attempted to prevent reproductive and possibly nursery pig disease, but vaccine should be administered only to nonpregnant sows. Several investigators have questioned the use of modified-live PRRSV vaccine in late-term naive gestating sows (an unapproved use) to control acute reproductive losses and to minimize preweaning mortality.[2,3] Furthermore, other investigators have noted outbreaks of disease in well-vaccinated herds, suggesting that the vaccine might not protect against the full range of different field isolates.[4,5] None of these measures controls PRRS-associated disease losses in suckling pigs during acute outbreaks or in herds with endemic PRRS.

We have used a set of farrowing house management practices—**m**anagement **c**hanges to **r**educe **e**xposure to **b**acteria to **e**liminate **l**osses (McREBEL)—to control mortality rates in both suckling and nursery pigs during acute outbreaks of PRRS. McREBEL is based on three principles:

- piglets are born essentially free of bacteria, with the notable exception of *Streptococcus suis*;[6]
- none of nine different field isolates of PRRSV were able to kill caesarian-derived, colostrum-deprived (CDCD) piglets after these bacteria-free pigs were challenged with the virus;[7] and
- the majority of clinical signs and mortality associated with field cases of PRRS is the result of secondary bacterial infections.[8]

McREBEL has been successfully used in herds with endemic PRRS in the nursery in which virus is continuing to circulate among sows and in herds that appear to be infected with strains of the virus that are not controlled by commercial vaccines.[9] In this study, we investigated what effect minimizing the amount of crossfostering within the first 24 hours of life, as part of a McREBEL protocol, would have on preweaning and nursery mortality in a herd in which PRRSV was still actively circulating.

Case history

An 1800-sow farrow-to-feeder pig herd (PIC Camborough 15 genetics) experienced an outbreak of acute PRRSV, with a sudden increase in nursery and then preweaning mortality (Figure 1), late-term abortions, and early farrowings. The herd was using an all-in–all-out (AIAO) animal flow strategy in both the farrowing and nursery sections at the time of the outbreak.

During each week of the first month of the outbreak, 14%–19.5% of litters were aborted at > 80 days gestation or were born early. The percentages of stillborn piglets and mummified fetuses increased sharply in the third to fourth weeks of the outbreak (Figure 2). The percentage of mummies remained elevated (> 1.8%) for 16 weeks.

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Figure 1: Study timeline:

1/1/96	Herd initiates minimal crossfostering post-24-hours-old policy (extensive sizing and sexing occurring on the day after birth)
2/12/96	PreMcREBEL period begins (birth of first production group).
2/19/96	Reproductive losses begin.
4/19/96	Initial farm visit, McREBEL implemented. —minimal fostering within first 24 hours; —euthanasia of all existing poor-doing pigs and those unresponsive to therapy in lactation and nursery phases.
	First production group sold.
4/22/96	McREBEL 1 period begins (birth of 11th production group).
6/9/96	Birth of last group observed with weakborn piglets/early farrowings. This group was also the last one to test seropositive for PRRSV by ELISA at 9 weeks of age before nursery became seronegative.
6/23/96	Last group with elevated rates of mummified fetuses born.
6/27/96	First McREBEL 1 production group sold.
7/1/96	McREBEL 2 period begins (birth of 21st production group).
8/5/96	First production group of McREBEL 2 sold.
8/9/96	McREBEL 3 period begins (birth of 31st production group).
11/7/96	First production group of McREBEL 3 sold.
11/11/96	End of McREBEL 3 (birth of production group 40).
1/16/97	Sale of 10th production group of McREBEL 3.

In many litters, weakborn piglets were observed during this outbreak. Treatment by mass injection of all piglets in affected litters or rooms with different antibiotics (penicillin, gentamycin, and ceftiofur) was not successful in controlling preweaning mortality. Nursery mortality (> 3%) was also not improved by antibiotics (tetracycline or amikacin) administered in the drinking water or via intramuscular injection of affected pigs.

Facilities

The herd was housed in four parallel freestanding buildings. The breeding and gestation facilities were two curtain-sided buildings ventilated with large fans at one end and air inlets at the other in the "tunnel ventilation" configuration. Sows were maintained in gestation crates or breeding pens. Water was delivered by a common trough running the length of the building in the front of each crate. Waste was removed from all buildings by flush gutters under concrete slats or woven wire. Farrowing took place in the third building, which contained eight individual farrowing rooms opening off a common hallway. Each farrowing room was equipped with 36 crates. The farrowing building was solid-sided and was mechanically ventilated by drawing air into the common hallway through cool cells. The common hallway was used to temper air before it was drawn through the open attic and into individual rooms through ceiling baffles. Piglets were weaned at 18–21 days of age and moved to nursery rooms via a covered walkway.

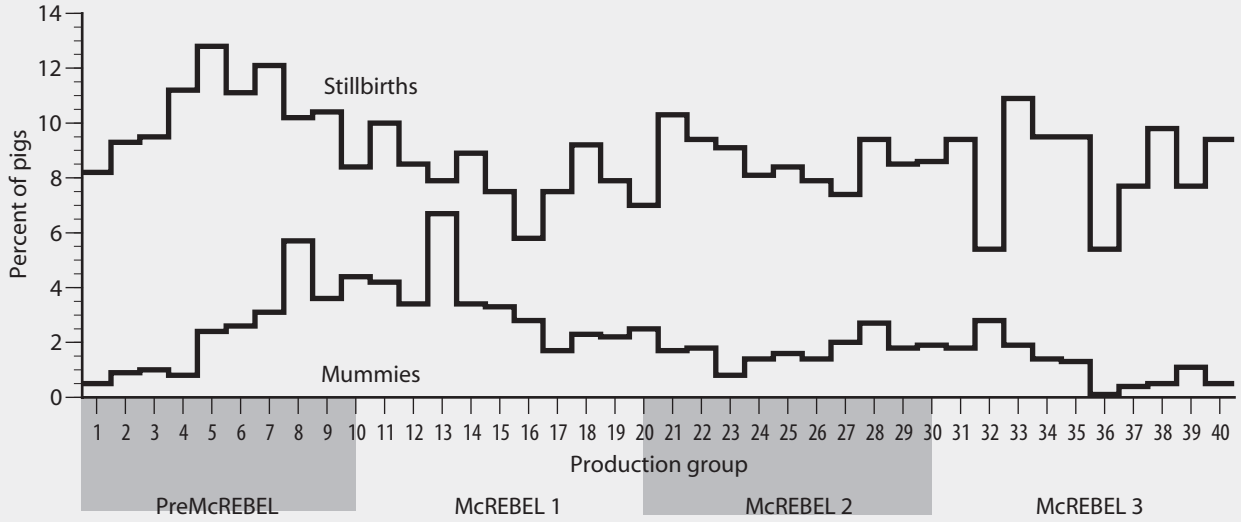
The nursery building had seven rooms opening off a common hallway. The nursery was solid-sided and ventilated with the same equipment as the farrowing building. Nursery pigs were reared to 9–9.5 weeks of age on wire mesh floors with approximately 20 pigs per pen. Starter feed was pelleted and all other stages of nursery feed were ground.

Pre-outbreak management

Gilts sent to the herd at 26 weeks of age from the company farrow-to-finish multiplier were vaccinated at 10 and 18 weeks of age for PRRSV (RespPRRS™, Boehringer Ingelheim/Nobl Laboratories; St. Joseph, Missouri). No sows in the study herd were revaccinated for PRRSV. No isolation facilities existed for housing breeding animals sent to the herd.

Although crossfostering between litters after 24 hours of age had been minimized in the case herd prior to the PRRS outbreak, the producer was moving piglets between litters at birth to match them by size and by sex. The producer stated that ≥ 85% of the piglets were fostered off their birth dam during the sizing and sexing procedure. At weaning, two to three size- and sex-matched litters would be placed into one

Figure 2: Stillborns and mummies by period



nursery pen. Pigs from two farrowing rooms were being commingled into a single nursery room in a strict AIAO protocol. All the pigs farrowed within the same week were kept together as a cohort and cohorts were not commingled. These management procedures were in place at the onset of the PRRS outbreak and continued until the McREBEL protocol was implemented 9 weeks after the initial outbreak.

Study design

Two months after the initial PRRS outbreak, the herd was still experiencing severe PRRS-associated losses. The crossfostering protocol was changed to that called for by McREBEL management:

- crossfostering of piglets between litters did not occur after 24 hours of age, and even within the first 24 hours, fostering was limited to moving only enough piglets to fill available teat spaces. (The 24-hour "rule" was established arbitrarily as a compromise between the need to limit movement of piglets among litters to reduce spread of bacterial pathogens during PRRS outbreaks and the need to use all functional teats available.) For example, only four pigs would be exchanged between a litter with 14 and a litter with six liveborn piglets, if both sows had 10 functional teats. The farrowing house personnel were instructed to disregard size variation within litters and piglet sex when making fostering decisions. Piglets were only moved to other litters within the same farrowing room. No piglets were fostered between litters after 24 hours of age except in cases of sow mortality or complete agalactia. These changes resulted in 15% of piglets being fostered away from their birth dams.
- suckling piglets and nursery pigs were moved strictly AIAO by room;
- piglets were not moved among different rooms to "nurse sows" (especially poor-doing piglets to younger age groups attempting to save them); and
- pigs that were clinically unresponsive to therapy with ceftiofur (Naxcel®, Pharmacia and Upjohn; Kalamazoo, Michigan) (three total injections at 3 mg per lb administered every other day to all litters in which affected piglets were observed), and piglets that were very thin, lethargic, gaunt, moribund, or lightweight and depressed were euthanized immediately (to minimize the exposure of other pigs in the litter or room to secondary bacteria and PRRSV). These piglets were never placed on nurse sows in rooms with younger piglets. All preweaning euthanasias were recorded as a mortality against their sow.

No sows were moved to other farrowing rooms after initial placement nor were piglets weaned to another nursery room. Piglets were weaned at approximately 18–21 days of age. Piglets in the two rooms (72 crates total) were weaned AIAO each week into a single nursery room; these piglets constituted the "cohort" for that week. At weaning, pigs were sorted into nursery pens by size and sex regardless of their litter of origin. Two to four pens per room were initially left empty to receive sick pigs that needed antibiotic therapy and reduced competition. These pens were located adjacent to exhaust fans in one end of the nursery room. Otherwise, pigs were left in their original pens throughout the nursery phase. Poor quality, lightweight, or sick pigs were euthanized prior to sale and assigned as mortalities to their group, rather than moved to a room with younger pigs. Once McREBEL was implemented, approximately 0.5% of nursery pigs were euthanized per production group until virus circulation ceased.

Data collection

Total number of piglets born live, died, and weaned were recorded for each room, and cohort group performance values were combined from the two farrowing rooms for statistical analysis. Total number of piglets placed into each nursery room, number of mortalities per week, and the total number died/sold were recorded for each nursery room. Total production group mortality from birth to sale (approximately 10 weeks old) was calculated using liveborn and combined mortality data from both farrowing and nursery room charts. Piglet sale bills were used to determine the average weight of pigs sold from each nursery room. All pigs in each nursery room were either shipped or euthanized on the day of sale. PigCHAMP® bureau records were used to determine total pigs born per litter, percent stillbirths, and percent mummies.

Statistical analysis

Data was analyzed by 10-week-long production periods, the amount of time required for a cohort to complete a "turn" or move completely through the farrowing and nursery facilities. The "PreMcREBEL" period consisted of the 10 weeks prior to implementing McREBEL management. "McREBEL 1" represented the first 10 weeks of production under McREBEL management. A total of 40 weeks of production ("PreMcREBEL," "McREBEL 1," "McREBEL 2," and "McREBEL 3") were included in the statistical analysis. Differences between mean production group preweaning mortality, nursery mortality, total mortality (production group combined preweaning and nursery mortality), and mean nursery sale weight were compared among the four production periods by one-way ANOVA.

Virus circulation monitoring

Blood samples were collected from 10 nursery pigs per sampled room at various intervals during the three postMcREBEL periods. Four different samplings were conducted on pigs that were at 3, 5, 7, and 9 weeks of age. Sows (30) and gilts (15) were also randomly selected and tested toward the end of McREBEL 2 (production week 27). All samples were analyzed by PRRSV ELISA (HerdChek PRRS®, IDEXX Laboratories, Inc; Westbrook, Maine) and some were confirmed as viremic by virus isolation on porcine alveolar macrophage cultures.

Results

Percentages of preweaning mortality, nursery mortality, and total mortality were significantly higher and mean sale weight was significantly lower during the PreMcREBEL period than during the three McREBEL periods (Tukey's comparison of means, rejection level = .010) (Table 1). Production group preweaning mortality was significantly lower ($P < .01$) for the piglets born during McREBEL 1 than for piglets born during the PreMcREBEL period (Figure 3). Nursery mortality for the production groups born during McREBEL 1 also dropped quickly (Table 1). The rapid reduction in mortality rates during McREBEL 1 occurred while fewer pigs were born per week and significantly

($P<.01$) fewer pigs were dying in lactation and the nursery (Figures 3, 4, Table 1). Mean nursery pig sale weight began to increase 4 weeks prior to the implementation of McREBEL (Figure 5). The production period mean mortality and sale weights (Table 1) were significantly different between PreMcREBEL and each of the three McREBEL periods ($P \leq .0001$). Weakborn piglets, early farrowings, and elevated percentages of mummies continued to the end of McREBEL 1 (production week 20).

PRRSV was isolated from seven of 10 9-week-old nursery pigs in production week 25 (during McREBEL 2). When nursery pigs were serosampled halfway through McREBEL 2, only the 9-week-old group contained seropositive pigs. When the same nursery pig age groups were tested later in McREBEL 2 (production week 27), all were seronegative for PRRSV sows and gilts still had an unstable PRRSV antibody profile at this time (Figure 6).

Table 1: Production parameters (±SD)

Observation period	Mean pigs born live per litter	Mortality preweaning	Mortality nursery	Mortality combined	Mean sale weight		coefficient of variation for mean period sale weight
PreMcREBEL	11.05±0.61	14.94±3.81[a]	8.03±3.50[a]	22.05±3.87[a]	20.19±4.00kg	(44.42±4.00lb[a])	9.00%
	(9.9–11.9)[c]	(7.32-22.56)	(1.03-15.03)	(14.31-29.79)	(16.55-23.83)	(40.42-48.42)	
McREBEL 1	10.57±0.43	9.75±1.69[b]	2.98±1.29[b]	12.43±2.28[b]	22.64±0.46kg	(49.81±1.07lb[b])	2.15%
	(9.6–11.1)	(6.37-13.13)	(0.4-5.96)	(7.87-16.99)	(21.72-23.56)	(47.67-51.95)	
McREBEL 2	10.78±0.55	8.67±1.20[b]	2.01±0.78[b]	10.48±1.66[b]	23.31±0.83kg	(51.28±1.93lb[b])	3.76%
	(9.6–11.6)	(6.37-11.07)	(0.45-3.57)	(7.16-13.8)	(21.65-24.97)	(47.42-55.14)	
McREBEL 3	10.11±0.37	10.08±1.14[b]	2.63±0.96[b]	12.43±1.62[b]	22.45±1.06kg	(49.39±2.47lb[b])	5.00%
	(9.5–10.6)	7.8-12.36)	(0.71-4.55)	(9.19-15.67)	(20.33-24.57)	(44.45-54.33)	

a,b different superscripts are statistically different ($P < .01$)

Figure 3: Preweaning and nursery mortality by period

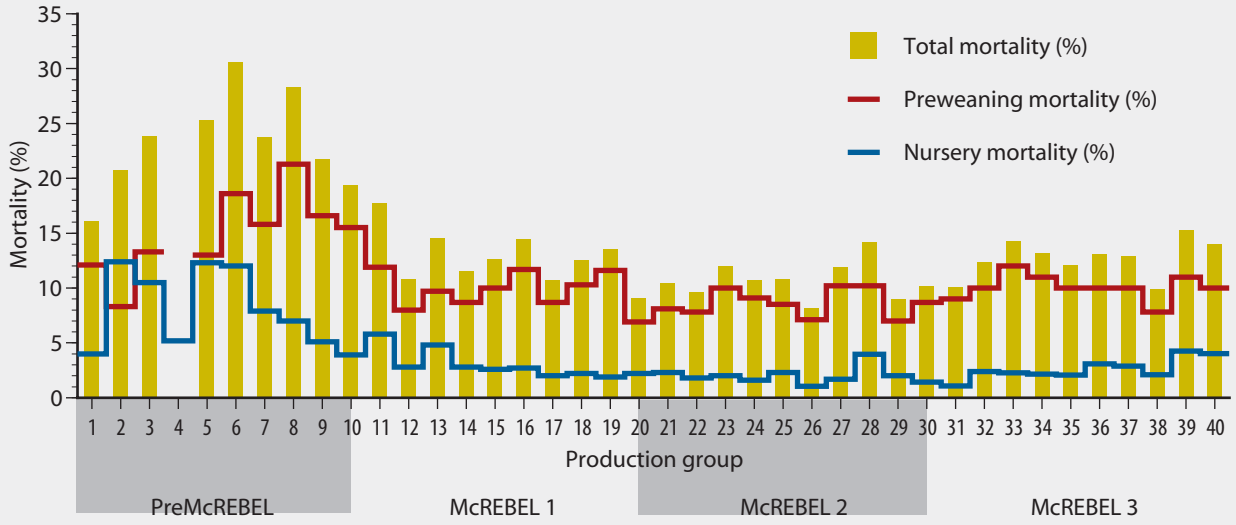


Figure 4: Number of pigs born alive and number to nursery by period

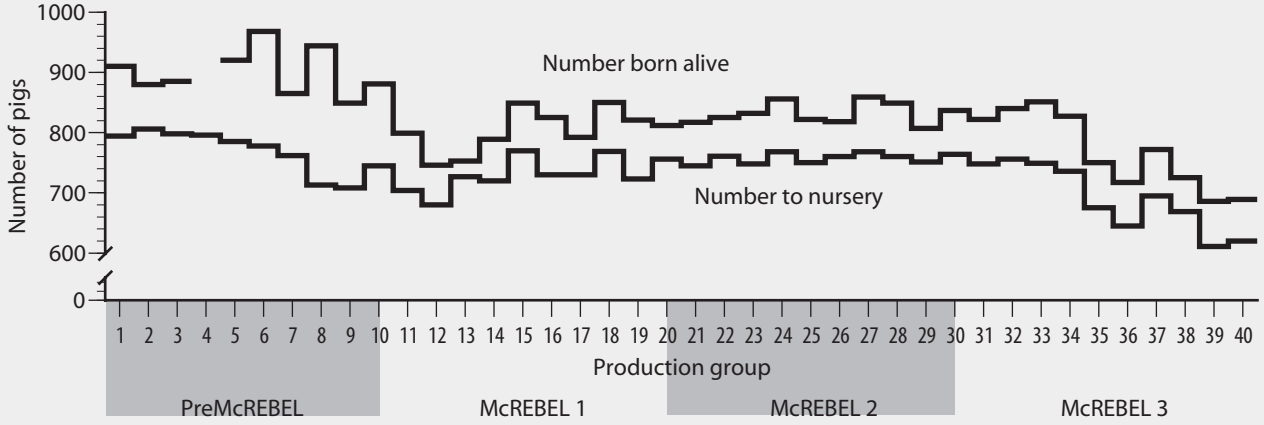
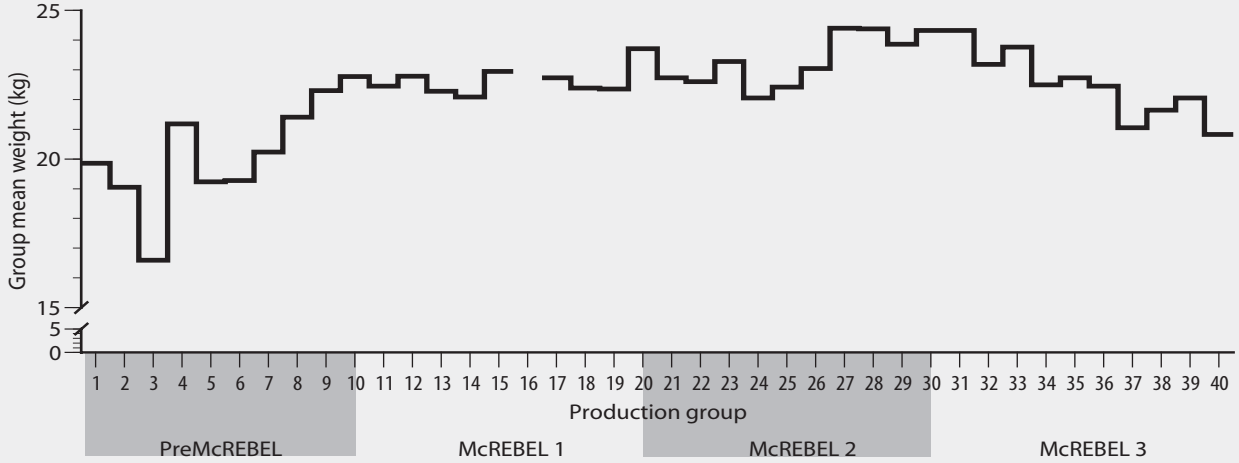


Figure 5: Mean sale weight by production group



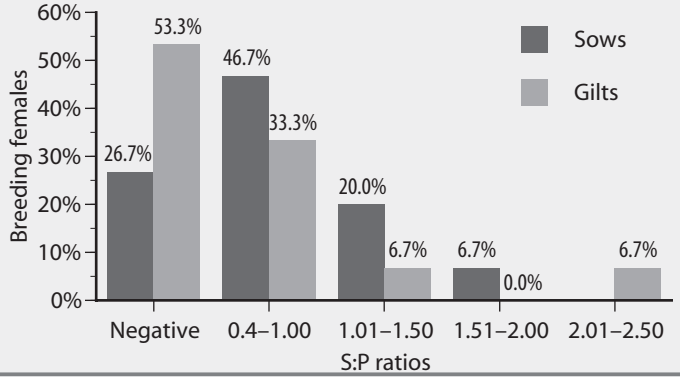
Discussion

In this herd, extensive fostering of piglets between litters within the first 24 hours of life did not appear to control piglet losses associated with an acute outbreak of PRRS. Moving most piglets from their birth dams to another litter may help spread PRRSV and other bacterial pathogens to more litters. Also, extensive crossfostering may contribute to a delay in successful lactation for several hours after the new litter is assigned.[10] Loss of regular nutrient uptake by already weak piglets may contribute to their inability to mount a successful response to environmental pathogens.

Prior to implementing McREBEL, the producer was not systematically treating whole litters with antibiotics, nor euthanizing pigs that were very sick, moribund, or nonresponsive to therapy. These practices may have increased the amount of pathogens littermates were exposed to during lactation.

The performance improvements observed in this case after McREBEL management was implemented in the herd were achieved despite evidence that the infection with PRRSV was still in the acute phase (i.e., continued birth of weakborn—potentially virus-infected—litters, elevated number of mummified fetuses, and continued virus circulation in the nursery). Early farrowings and abortions were also observed until the latter part of McREBEL 1, but were not noted because the breeding manager did not realize that they needed to be recorded as a specific event on the sow's record.

Figure 6: Breeding female PRRSV serology results during McREBEL2



The extensive crossfostering before 24 hours of age practiced in this herd before McREBEL was implemented, with little or no crossfostering after 24 hours, did not prevent elevated preweaning and nursery mortality after the outbreak of acute PRRSV. Extensive crossfostering before 24 hours of age was not practiced by previous herds in which McREBEL was used to successfully control preweaning disease and mortality.[9]

The improvement observed in nursery pig mean sale weights 4 weeks before McREBEL was implemented was probably due to the euthanasia of nursery pigs of all ages that were chronically ill, unthrifty, and unresponsive to therapy at the start of McREBEL management. Therefore, the mean sale weight for these groups could have been artificially elevated as a result of removing remaining chronically ill pigs from the production groups present in the nursery at the time of McREBEL implementation.

Target levels of production group preweaning and nursery mortality were obtained by the second week after McREBEL was implemented (10th week of the outbreak) (Figure 3). Preweaning mortality was reduced to 9.5% during McREBEL 1, well below the 14.53% Baysinger reported for the fifth- and sixth-month performance in 27 post-PRRSV-outbreak herds.[11] However, elevated rates of mummified fetuses (≥2.2%) were observed through the end of McREBEL 1. Significantly elevated rates (P <.05) of mummified fetuses were observed during the fifth and sixth months following the onset of PRRS in Baysinger's study. Therefore, our observation of elevated numbers of mummified fetuses during the eighteenth week of the outbreak (i.e., the end of McREBEL 1) appears not to be unusual.[11]

In this herd, the farrowing house supervisor observed thin, weak-born, "PRRS-like" litters through the sixteenth week, and elevated mummies were observed until the nineteenth week after the beginning of the outbreak. These litters were easily observed

since the farm was crossfostering very few piglets. Regrettably, no colostrum-deprived weakborn piglets could be found for sampling to attempt virus isolation on the days of any of the several herd visits. The thin, weakborn piglets we observed were likely to have been infected with PRRSV in utero during this period, given the elevated rates of mummified fetuses (≥ 2.2%) that were recorded (through the end of McREBEL 1). At-term birth of weakborn viremic piglets in litters with high numbers of mummified fetuses has been reported in a study in which pregnant sows (at 84 days of gestation) were experimentally infected with the Lelystad strain of the virus.[12] Viremic littermates have now been observed in litters with mummified fetuses from PRRSV-free gilts exposed to both strains of the virus as early as 20 days gestation.[13] In the present case herd, herd size and the use of three separate buildings for breeding, gestation, and farrowing may have delayed the time of infection for certain subpopulations within the herd, thus potentially prolonging the duration of reproductive losses. Therefore, it appears likely that some PRRSV-infected litters were still being born during most or all of McREBEL 1, even though the herd returned to normal preweaning mortality rates at the beginning of this period.

Spontaneous resolution of PRRSV-associated nursery disease and mortality starting with the groups of pigs raised under McREBEL management (born study week 11, McREBEL 1) appears unlikely. PRRSV commonly circulates among nursery pigs for prolonged periods of time, causing continued clinical signs following cessation of reproductive losses.[14] PRRSV continued to circulate among nursery pigs in the case herd until approximately the twenty-first production week, when the last seropositive group of nursery pigs to be sold entered the nursery. Interestingly, this group of pigs (born study week 18) was the last in which observations of weakborn litters and an elevated number of mummified fetuses was reported. Also, 25 weeks after the onset of reproductive PRRS (i.e., during McREBEL 2), serologic evidence of virus circulation could no longer be detected in the nursery. This was accomplished without depopulation or other alterations to normal AIAO pig flow.

Prior to the present study, successful control of endemic nursery pig PRRSV-related losses was possible only by depopulating the nursery after virus circulation among the sow herd had ended.[15] However, other investigations have been unable to control endemic PRRSV by depopulating the nursery.[16] This may be due in part to cessation of the birth of viremic piglets that would carry the virus and therefore reintroduce it into the newly depopulated nursery. Arresting virus circulation among sows is probably essential to ending the birth of in utero-infected viremic piglets.

The case herd in the present study may have been approaching the time when it could have successfully used nursery depopulation to stop PRRSV circulation and losses, as evidenced by the serologic results obtained near the end of McREBEL2. By implementing McREBEL management, the producer reduced the expected period of reduced gains and elevated mortality by at least eight production groups versus waiting to depopulate the nursery. If weekly average nursery mortality (deaths and euthanized poor-health pigs) for the PreMcREBEL period (8.03% ±3.50%) and average mortality for McREBEL 1 (2.98% ±1.29%) is multiplied by the average number of pigs weaned per week during McREBEL 1 (730), then by implementing McREBEL rather than waiting to depopulate the nursery, the producer realized a net increase of 295 surviving pigs for the period.

If PRRSV vaccine had been used in suckling piglets, it may also have helped reduce nursery pig losses in this herd. Some clinicians have reported good results using piglet vaccination (at the time, an off-label use) to control PRRSV-associated nursery pig losses.[1] However, others have reported that the vaccine was ineffective in controlling reduced daily gains in PRRSV-infected nurseries.[5,17] In this herd, abortions, weakborn pigs, and high rates of mummified fetuses were observed in all parities, including recently introduced and twice-vaccinated gilt litters. We chose not to revaccinate the herd based upon this observation, assuming that cross protection between the vaccine and wild-type virus was minimal. In late 1996 to mid 1997, other "well vaccinated" herds experienced devastating outbreaks of PRRS-like disease, including high rates of mid- to late-term abortions, high rates of sow mortality and adult morbidity and fever, and elevated preweaning and nursery mortality.[4,5] McREBEL may provide a successful management tool for limiting preweaning and nursery pig losses in vaccinated herds experiencing "acute PRRS" outbreaks.

Antibiotic treatment alone has not consistently controlled clinical and production losses associated with bacterial infections secondary to endemic PRRS in nursery pigs.[16] Others have reported a decrease in severity but not elimination of clinical signs resulting from antibiotic treatment programs.[18]

Besides McREBEL, no other strategy has been reported to date to control preweaning piglet losses that occur during active PRRS outbreaks. Long-term piglet and nursery pig PRRS control (prevention) efforts are now focused upon controlling sow herd virus circulation by isolating, acclimating, and vaccinating the sow.[19,20]

Preweaning and nursery performance levels of mortality and growth that met the production goals of this large sow herd were maintained for the last 29 weeks of the study, during a period when virus was actively circulating within the herd. This was achieved without depopulating the on-site, single-roof nursery, where seropositive 9-week-old pigs continued to be present. The nursery production levels achieved with McREBEL for the final two observation periods attained rates close to the 2.0% mortality reported for nursery depopulation.[15] This level of performance was attained at least 8 weeks prior to the first possible opportunity to successfully depopulate the nursery.

The producer has continued to use McREBEL management since it was implemented during the outbreak reported here. Farrowing and nursery performance levels similar to those achieved during McREBEL 1–3 have been maintained, and 9-week-old nursery pigs were still seronegative for PRRSV when sampled in June and July of 1997.

Further controlled investigations are needed to isolate the specific factors that may be responsible for the apparent need to minimize crossfostering of piglets among litters even within the first 24 hours of age to control disease losses during PRRSV outbreaks.

Implications

- Minimal crossfostering of piglets

between litters is critical to the success of McREBEL for minimizing preweaning and nursery pig mortality even during the first 24 hours of life.
- McREBEL management did not adversely affect this herd's achievement of target performance values for herd preweaning mortality, nursery mortality, or 9.5-week sale weights.
- PRRSV-related increases in preweaning and nursery pig mortality can be controlled rapidly without vaccination or depopulation of the nursery by implementing McREBEL management.
- Even in an 1800-sow herd, McREBEL effectively controlled PRRSV-associated preweaning and nursery mortality.

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