DIFFERENT APPROACHES TO HANDLING PRRS

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ABSTRACT

Developing methods to control PRRS is both a critical and long-standing challenge for the swine industry. To quote Dr. Mark Fitzsimmons, Swine Graphics, Webster City, Iowa: ***Abasic PRRS virus information, particularly in the area of immunity and transmission, is conspicuous by its absence.*** In short, there is a lot we do not know yet about PRRSV and hence its *predictable* and *effective* control which I wish I could share with you today. However, we have come a long way from the days of Mystery Swine Disease, Abortus Blau, Porcine Epidemic Abortion and Respiratory Syndrome, and EMC virus. **PRRS control strategies that work have been developed**, each however usually limited to specific types of situations and production types. This presentation will attempt to clearly define essential concepts of PRRSV-pig "biology" and then review control strategies for PRRS, both conventional and unconventional. It is by necessity only an overview, hopefully providing a clear basis and framework for weighing different approaches to PRRS control. For more details on PRRS control I strongly urge you to read the applicable sections of the Producer Edition of 2003 PRRS Compendium produced by the National Pork Board (United States) and edited by Drs. Zimmerman, Yoon, (Iowa State University) and Neumann (National Pork Board). This is an excellent document which provides a practical review of scientific knowledge as well as current, albeit untested methods used for controlling PRRS (scientific proof often follows practical and effective innovations).

For a more detailed review of current scientific knowledge read the 2003 PRRS Compendium, second edition. Both are available (for $30US) in a single CD from the US National Pork Board at:
http://porkstore.pork.org/customer/product.php?productid=202&cat=260&page=1.

PRRSV-PIG "BIOLOGY" AND IMMUNOLOGY

PRRS is particularly a disease of LARGE three-site or single-site swine herds which use management short-cuts that don't meet the needs of the pigs or designs which compromise both internal and external biosecurity.

Continuous flow rooms, buildings, and possibly sites, as well as breeding barns which receive susceptible gilts regularly enable **continuous virus replication** (constant source of susceptible pigs), **holding-back** of poor-doers (Typhoid Marys), short time for cleaning,

disinfecting, and DRYING of rooms and transports, inadequate isolation and testing of breeding stock, semen, inadequate pre-immunization of breeding replacements prior to entry, etc, etc, etc. Continuous virus replication enables maximum PRRSV mutation and ultimately "escape" from the herds' initial immune responses. Typhoid Mary **hold-backs infect younger groups of pigs**, ensuring they repeat the same PRRSV-associated disease-losses of their predecessors.

Definitions:

Modified-Live Virus (MLV): PRRSV which has been altered in the laboratory to reduce its virulence or pathogenicity in an effort to make it safe**(r)** for use as a live-virus vaccine.

Virulent Live Virus (VLV): ***Unaltered*** or Wild-Type PRRSV isolated from a diseased pig. VLV can be grown and multiplied unchanged in Porcine Alveolar Macrophage (PAM) cultures, from blood or lung tissue from purposefully infected PRRSV-free pigs, or from diseased pigs within herds during PRRS outbreaks. In the last case, it is imperative to collect the blood or tissues from febrile aborting sows or weak-born febrile piglets if it is to be used later to immunize / acclimatize gilts in isolation or perform whole-herd exposure and closure (see below).

Horizontal infection (transmission): PRRSV infection comes from another pig of the same age or production group (all-in all-out flow) or within the same room where pigs of different ages are housed together (CF production ***and breeding herds***). Virus transfer occurs by exchange of saliva, blood, or semen. Therefore, **mixing pigs** from different litters or pens (causes fighting and exchange of saliva and blood) or **not changing needles or blades** between litters, pens, or at times pigs, ***helps horizontal transmission.*** We all know the impact PRRSV-infected semen can have!

Vertical infection (transmission): PRRSV infection comes from the sow either ***in utero*** (across the placenta **~ 70 days at the *earliest***) or from milk, oral / nasal contact. *In utero* infection has the most severe impact on piglet immune system and duration of (persistent) PRRSV infection.

Persistent infection or "persistence": Ability of PRRSV to stay in an infected pig for weeks and months after infection. PRRSV may persist in these pigs, be shed, and infect other pigs over 80 to 100 days (maybe longer??). Persistence seems to be a result of a slowed development of FULLY protective immunity (ability to eliminate the virus) by some unknown effects of PRRSV on the pig. PRRSV persistence after infection is the reason recommendations are made for both long periods of time for herd closures when attempting herd virus elimination or for duration of isolation during acclimatization after exposing new gilts to VLV.

PRRSV BIOLOGY

Post-infection PRRSV "timeline": (Long time needed to develop "full" immunity / clear PRRSV infection)

Days post-infection

10 – 30	Viremia (virus can be isolated from blood), strong PRRSV ELISA antibody response.
20 – 30	Earliest time neutralizing antibody can be detected in blood
60+	Full or peak titer of neutralizing antibody in blood reached
100 – 150	Tonsil / Lymph Nodes become PRRSV negative (nursery infection)
100 – 150	Many pigs become PRRSV ELISA negative (< 0.4 S/P ratio), still SN+.
150++	Tonsil / Lymph Nodes become PRRSV negative (*in utero* infection)
200	Duration of herd-closure needed post-outbreak to eliminate PRRSV from the herd.

If total, fully-protective immunity (elimination of PRRSV from ALL tissues of the pig) requires up to 150 days after infection, then this "fact" may explain why piglets born to gilts are the most likely to be PRRSV-infected *in utero*. Endemic PRRS most likely results from gilt litter *in utero* infected piglets carrying the virus for months and subsequently infecting the rest of the pigs in their production group and causing disease losses in either the nursery or finisher phase. This hypothesis would also explain the success of Parity Segregation production for eliminating endemic PRRS in piglets born to sows in the P2+ herds, limiting endemic PRRS to only the P1 gilt herd pig flow.

PRRSV IMMUNE RESPONSES: TO PROTECT or NOT TO PROTECT, *THAT* is the QUESTION

Introduction: The ability of pigs to produce protective immune responses to PRRSV infections that can also "cross-protect" against other "strains" of the virus often appears limited or even non-existent. The anti-PRRSV immune response seems "narrow" in scope, potentially much like the HIV of AIDS and its cousin, Feline Immunodeficiency Virus. This poor ability to cross-protect possibly is due to a relatively high rate of genetic mutation (changes in genetic sequence) which results in far more "strains" (viruses that don't cross-protect) than even Influenza viruses. Therefore PRRSVs are nearly impossible to "immunologically categorize" or predict cross-protection between since 1) the mechanisms needed for protection are poorly understood, 2) the location of the targets of immune responses are unknown, so 3) we don't know which mutations or changes in genetic sequence are important! PRRSV immunity is often discussed in scientific terms, common descriptive terms, and ***hybrid combinations of both (slang)!*** This common verbal practice of veterinarians and veterinary "scientists" adds to the frequent and sometimes serious confusion that we all experience when talking about control of PRRSV.

PRRSV Immune Response Definitions:

Homologous PRRSVs are two virus isolates tested in the laboratory which have the SAME GENETIC SEQUENCE.

Heterologous PRRSVs are two virus isolates tested in the laboratory which have DIFFERENT GENETIC SEQUENCES. By definition they can differ by a couple of

mutations (> 99.5% "homology" or "sequence sameness"), or many, many mutations (<85% "homology"). The degree of difference or "heterogeneity" plays a role in the amount of cross-protection against disease between PRRSV isolates. **HOWEVER, "% genetic homology" between two PRRSV isolates cannot be used what-so-ever to predict protection!** This is because % homology does not include any information about the LOCATION of the mutations or genetic differences, i.e. are they located in immune response target genes.

Protected pigs: Pigs and pregnant sows which are totally resistant to disease when challenged or injected with live Wild-Type PRRSV. The most complete and only predictable protection is **against the same or homologous WT PRRSV.**

Susceptible (unprotected) pigs:

1. Naïve or uninfected pigs are obviously susceptible to disease following WT-PRRSV infection.
2. **WT-PRRSV immune pigs can be very susceptible to disease with a new, genetically-different or *"heterologous" WT-PRRSV!!!***
3. ***Vaccinated pigs also can be very susceptible to WT-PRRSV infection and disease!*** infection. By definition, Modified-Live Virus vaccines are genetically-different or heterologous to all WT-PRRSVs.
4. Susceptibility is the opposite of protection against PRRS disease. There is a **full-spectrum of pig responses to heterologous PRRSV infection** ranging from full protection (we never know the pigs were exposed) to no protection / full susceptibility and severe disease. To date, we cannot predict the amount of cross-protection between two different PRRSVs by comparing their genetic sequences. We do not know where the targets of any Cell-Mediated Immune responses are for PRRSV. We do know where at least one target is of serum-neutralizing antibodies. Measuring the cross-neutralizing ability of serum neutralizing antibody against the heterologous PRRSV may provide some information about cross-protection.

PRRSV strains are virus isolates which are **1) genetically different** or ***heterologous*** and **2) immunity against one does not cross-protect "well" against the other**. One isolate stimulates immune responses which do not cross-protect against clinical disease following challenge of immune pigs with the other heterologous isolate. The most extreme types of heterologous strains were the Acute PRRSVs which caused severe disease initially in frequently vaccinated herds in 1996 – 1998, and again from 2001 - 2004 in Wild-Type PRRSV-immune herds in the US. There is currently no exact definition of how severe the clinical disease should be, what specific clinical signs or neutralizing antibody / immune response test outcomes to conclude there is a "lack of cross-protection" between two isolates. Regrettably, this conclusion is only established retrospectively, i.e. after a severe PRRS outbreak and economic loss occurs following mixing two groups of pigs, sows and replacement gilts, or using semen infected with a heterologous PRRSV.

Subpopulations are subgroups of sows or gilts in the sow herd which are susceptible to PRRSV infection (naïve) or re-infection (lost their protective immunity). They have low or no immunity against PRRS. All animals in a PRRSV infected herd can be susceptible to

infection and disease by a new and DIFFERENT enough strain of PRRSV. Subpopulation has become a slang term which has clear meaning when discussing naïve, non-infected groups of pigs within a herd such as newly introduced "negative" gilts. The meaning of subpopulation becomes less clear when discussing animals that have "lost their immunity" to the herd's original or "homologous" PRRSV. Lastly, there may be "subpopulations" of pigs or sows within a "positive" herd following a disease outbreak caused by introduction of a second, different, heterologous PRRSV "strain".

PRRSV immunity (protection) "slang subtypes":

Homologous immunity is produced against the same PRRSV isolate or strain that initially infected the pig. It is generally thought that this immunity is long-term, however, it may not be life long. Homologous immunity is the highest level of immune response efficacy a pig can produce, i.e. protection against re-infection with the same virus is almost total.

Heterologous immunity describes the protection pigs possess against challenge with a different virus strain. The "amount" of cross-protection provided by "heterologous" immune responses to heterologous virus challenge is often less than the "full cross-protection" seen of "homologous" immune responses to homologous PRRSV challenge. Sometimes it seems heterologous immunity is nearly nonexistent. The amount of heterologous immunity a virus can stimulate in a pig is probably due to how genetically similar that virus is to the new challenging strain of PRRSV, i.e. how few immune response targets on the different viral proteins have been mutated. The totally frustrating problem for veterinarians and scientists alike is that we do not know what virus genes must have identical sequences to stimulate fully cross-protective immunity.

PRRSV Immune Response Principles:

Relevance of Virus Genetic Sequence Homology to Producers and Veterinarians efforts to control PRRS: Typically, only PRRSV Open Reading Frame (ORF) 5 is sequenced and compared in Veterinary Diagnostic Laboratories. It "codes for" the major glycoprotein sticking out from the outer envelope or "shell" of the virus. **The ORF 5 sequences reported by Veterinary Diagnostic Laboratories describes only 4.4% of the whole PRRSV genome (~ 660 of 15,000 "base-pairs").** This ORF 5 sequence data is used by veterinarians to track and compare PRRSV isolates within and between herds. There is a single known target of neutralizing antibody coded by ORF 5. However, there are probably many other unidentified and significant antibody and Cell-Mediated Immunity targets coded in ORF 5 and the other 7 parts or ORFs of the PRRSV genome.

Therefore:

1. ORF 5 genetic sequence data alone probably is very incomplete for prediction of cross-protective immunity between two virus isolates from different time-points within a herd or from different herds.
2. ***We do not know where the key targets of antibodies or cell-mediated immunity are located even in ORF 5 (only one neutralizing antibody target in ORF5 is known).***
3. Predictions of cross-protection between vaccine and wild-type **PRRSVs using**

measurements of ORF 5 sequence "sameness" such as "RFLP cut-patterns" or % homology are nearly ***WORTHLESS*.

There are field reports both of PRRSVs with very similar ORF 5 sequences causing severe disease problems and of viruses with 10% or more different ORF 5 sequences causing nearly no disease when infecting pigs known to be immune to the other virus (Dr. Mark Wagner, personal communication). Therefore, attempts to predict the amount of cross-protection between two PRRSV isolates by % genetic sequence homology is a frustrating exercise of ignorance and futility. Two viruses with identical ORF 5 sequences have the best chance of stimulating fully cross-protecting "homologous immune responses, but even this is not guaranteed if they came from different herds. Identical ORF 5 sequences from pigs in the same herd isolated at different times can be used to predict that they are / the herd is protected against that PRRSV. It is most likely, **but not guaranteed**, that these two PRRSV isolates would be very similar throughout their whole genomes.

PRRSV DIAGNOSTIC TESTS AND INFECTION MONITORING

Testing to confirm groups of pigs are virus-free is difficult with PRRS. Pigs can lose their ELISA antibody response by 4 to 6 months after infection, even when they are being re-exposed to the same virus isolate. Some pigs can be persistently infected (are PCR positive on tonsil scrapings) and be ELISA antibody negative. Pigs retain serum neutralizing antibody titers for much longer, however, mutations have been found in the SN antibody target that can cause serum samples to test false negative or very low titer. Therefore, antibody testing of large numbers of pigs is needed to make decisions on whether a group of pigs or herd is PRRSV-free. Enough PRRSV can be carried by just a few pigs at weaning to infect a finisher full of pigs, but yet remain undetected in the nursery if only 10 to 30 pigs are tested at 10 weeks of age. Testing 30 animals can reliably detect at least ONE PRRSV-infected animal **only if** more than 10% of the group is infected. Retesting and finding negative ELISA results repeatedly over time also increases confidence that the group of pigs / herd is PRRSv negative. Tonsil scraping and testing by PCR is the best antemortem test available for detecting persistently infected pigs. This may be a very valuable method for routinely testing critical animals which are entered in low numbers such as boars to boar studs. **To certify that a group of pigs is PRRSV-free, ALL ANIMALS must be tested and found to be antibody test and / or PRRSV PCR negative.**

PRRS CONTROL OPTIONS BY HERD STATUS OR PRODUCTION PROBLEM

Acute PRRS (outbreaks with both reproductive and growing pig disease losses)

PRRS clinical outbreaks are times of anger and despair. However, they are also times where critical decisions need to be made that may minimize both current as well as long term losses due to PRRS. To minimize piglet and weaned pig losses implement McREBEL (limited cross-fostering) management immediately (procedures attached). Optimal results may not be seen during the first couple of weeks of the outbreak if sows are sick, not eating well, and

therefore not lactating well. If piglets are not moved between litters you should see only some litters with sick, poor doing piglets in them. This allows you to focus intensive care and treatment toward fewer litters than if fostering is practiced. Success is expected by both reduced mortality and disease as well as most pigs being weaned with fat irregardless of their body weight. PRRSV can be spread by needles or other means of carrying blood (knife blades, etc). Therefore minimize treatment to only pigs and litters that need it. Do not use one needle to treat two or more litters, and treat affected pigs for up to 5 days. PRRS causes severe damage to the piglet's immune system and their ability to fight bacterial disease, therefore treatment is needed for longer periods of time. Euthanize any piglets that do not respond to treatment, do not move them into the nursery to infect other healthier pigs. If McREBEL is followed correctly, nursery pig mortality will also be reduced and gain maximized even though pigs are placed into nursery pens by size and sex. It is essential to follow strict all-in all-out pig flow from farrowing through finishing to minimize the risk or duration of endemic PRRS associated-diseases.

Critical decisions also must be made for the sow herd during PRRS outbreaks. Long-term problems with PRRS come from variations in immunity to the virus between sows within infected herds. PRRSV actually does not spread easily or uniformly through herds, especially previously infected and possibly vaccinated herds (personal observation of differences in seroconversion in various breeding groups). Groups of non-immune or susceptible sows remain after the outbreak has ended (subpopulations). These subpopulations are thought to be the source of new clinical outbreaks and losses once PRRSV starts to spread in the herd again and finally reaches these susceptible animals. Some veterinarians and producers therefore have chosen to make sure all animals are exposed to PRRSV during the outbreak. They 1) vaccinate the whole herd or 2) ensure exposure to the homologous WT PRRSV. Exposure to the homologous WT PRRSV can be done by 1) moving aborted animals around to all areas of the gestation and breeding barn, 2) feeding back tissues or inoculating with serum from aborted sows and/or weak-born viremic piglets, 3) purchasing and infecting 4-6 months of naïve replacement gilts, 4) closing the herd to new additions for 200 days (more if there are still viremic piglets being born).

The goal is to get all animals in the herd immune to the virus, to stop shedding the virus, and therefore to deny the virus any new, susceptible hosts to continue to multiply in. ***If we fail to stop the circulation (shedding by one sow resulting in infection of new sows) long term problems with PRRS (see below) will reoccur / continue in the breeding herd and nursery / finisher.*** Therefore, many veterinarians choose to ensure all animals get infected during the outbreak and close the herd as the currently most predictable, effective, and easiest way to achieve whole herd immunity to end both horizontal and vertical virus infection in the herd. Frequently they report that abortions and birth of weak PRRSV-infected piglets ends quicker and completely. ***This information is provided in an attempt to be complete and is not a blanket recommendation to or not to use virulent live virus exposure. This decision is a complex one and needs to be done on a herd by herd basis. Factors and methods to consider which are intended to minimize the risk of this approach are listed in a document in the appendix below.*** Previously infected herds need to quickly determine whether the current outbreak is due to infection by the original herd PRRSV or a heterologous one.

Decisions of whether to spread virus through the herd or what source of virus to use may be changed if a new, heterologous virus has infected the herd.

Success of whole herd commercial MLV vaccination during an outbreak depends upon how much the vaccine will cross-protect with the wild-type virus. Short-term success may be observed just because the clinical outbreak would have ended quickly as a result of rapid whole-herd infection and hence establishment of herd immunity, not because of MLV vaccination. Failure of MLV vaccination to control PRRS long-term following outbreaks may be due to a low level of cross-protection with the WT PRRSV that infected the herd. This would leave gilts vaccinated at or prior to entry into the herd susceptible to infection by the herd's WT PRRSV. Gilts would be infected by the herd's WT PRRSV (with or without clinical signs) sometime during gestation and their piglets potentially would become infected *in utero.* The *in utero* infected piglets from gilt litters would then carry the virus into the nursery and finisher ultimately infecting and causing endemic PRRS disease losses in their production group. If PRRS disease losses persist or return in the face of continued vaccination and the original outbreak virus is isolated from both affected pigs and gilt litter piglets, then the vaccine did not stimulate sufficient levels of cross-protective immunity against the herd's WT PRRSV, particularly in replacement gilts. In this scenario vaccination with a poorly cross-protective MLV vaccine allows the herd to progress into "endemic PRRS" and **long-term economic losses.** Ultimately, the decision to use MLV vaccine in sows or pigs must be made upon whether with it, you are profitable and without it, you are not. Many US producers have determined they cannot produce pigs profitably with any permutation of MLV vaccination schedules because the reductions in disease losses achieved was unable to stop continuation of significant economic losses. Therefore they have turned to methods known to stimulate full homologous immune responses throughout the breeding herd, or have decided to eliminate the virus from their herds and pray the herd is not reinfected.

Endemic PRRS (reoccurring nursery / finisher disease)

PRRSV and common secondary diseases often continue to reoccur in the nursery and finisher phases for a long time following PRRS outbreaks. This may be the result of either horizontal spread between groups (holding back poor-doing INFECTED / SHEDDING Typhoid Mary pigs to younger age groups, virus transfer by boots or veterinary tools, etc.) or from vertically infected piglets (infected *in utero* or during lactation) who then carry the virus into the nursery and finisher. To control and eliminate endemic PRRS you must identify where the virus is coming from (nursery group cross-contamination vs. sow herd virus circulation causing *in utero* PRRSV infection). The virus is from contaminated nursery rooms or holding back of sick pigs if no PRRSV is detected by PCR from newborn piglets, serologic testing of the sow herd shows no evidence for active spread, and there is no evidence of active PRRS disease in sows (abortions, early farrowing, increased % mummies, and weak viremic piglets). In this case successful elimination of PRRS disease can be accomplished by total nursery depopulation, partial nursery depopulation, or whole nursery / finisher vaccination in addition to partial depopulation. All pigs are recommended to be vaccinated twice, 30 days apart. Some vets recommend also closing the nursery and finisher to any new pigs for 60 days following the first vaccination. **Strict all-in all-out pig flow with very thorough cleaning and disinfection is essential to success of these programs.** Also, to further ensure success,

assign workers to only work with clean or PRRSV-infected pigs until the project is completed. If the finisher is affected, continue these methods through all buildings until all infected groups of pigs have been marketed. **Depopulation or vaccination programs cannot stop PRRS in the nursery or finisher if pigs are getting infected *in utero* or in lactation**. Suggestions for how to stop virus circulation in the sow herd and therefore vertical spread to piglets are discussed below.

Methods for Long-Term Control of PRRSV Infection in Sow Herds

Summary: Long-term control of PRRS in herds depends HEAVILY upon stopping circulation or spread of the virus between sows in the herd. **PRRS losses in growing pigs cannot be controlled if the virus is circulating among sows in the herd.** Newly added gilts or susceptible subpopulations of sows will get infected and transmit the virus to their piglets *in utero* or during lactation (vertical spread) if there is circulating virus in the breeding herd. The following Critical Control Concepts and Procedures are useful to fully understand the different methods used to control PRRS in sow herds.

IN ALL CASES Biosecurity Flaws must be found and fixed first if the herds are to successfully **control PRRS** (remain "stable" but infected and immune to a single WT PRRSV) or **eliminate PRRSV** for long periods of time.

Isolation, acclimatization, and cool down of incoming gilts is designed to immunize gilts against the herds' homologous PRRSV. This should produce a homologous ("fully" protective) immune response against the WT PRRSV isolate in the herd. Acclimatization attempts to prevent the build-up of a subpopulation of animals in the breeding herd (gilts) which is susceptible to the herd's homologous WT PRRSV. Obviously these animals would spark a new outbreak of disease if they subsequently get infected. To **acclimatize gilts in isolation** they can be exposed to non-pregnant cull gilts or sows (unreliable method for infection), nursery pigs (inconsistent infection of gilts and risk of severe PRRS outbreak by introduction of a mutated heterologous PRRSV), or inoculated with serum or lung tissue from infected suckling piglets. The intent is to infect and immunize ALL gilts with the homologous herd virus. An extended time for cool down (90 days in all-in all-out isolation) is needed to get past the persistent infection period where acclimatized gilts could still shed the virus to susceptible sows in the herd or possibly to their piglets *in utero*. This procedure is used in herds that have demonstrated that MLV vaccines do not provide adequate cross-protective immunity against their herd's strain of PRRSV. This conclusion is established by consistently detecting WT PRRSV in suckling piglet serum which has an ORF 5 sequence homologous with virus isolates from the herd's PRRS affected nursery and finisher pigs. **These producers are experiencing significant and sustained economic losses** resulting from severe endemic PRRS.

Vaccination with MLV vaccines is approved for use at least twice before entry into the herd / breeding and then during every lactation to try to control PRRS in both sows and their progeny. A new approach called mass vaccination is now being advocated to control virus circulation in the sow herd. It requires working with a veterinarian since the MLV vaccine is not approved for use in pregnant animals. The goal is to get sufficient herd-wide cross-

protective immunity to stop circulation of PRRSV among sows and therefore vertical infection of their piglets. The herd is vaccinated twice, 30 days apart and is closed to any new gilts for 60 days. The herd may continue to be mass vaccinated quarterly, or attempt to eliminate PRRSV by introduction of naive non-vaccinated gilts. If elimination is desired, then the herd should be closed for 200 days (see herd closure below). The herd can then be checked by introducing and monitoring a few unvaccinated negative sentinel gilts to see if WT PRRSV and MLV virus circulation has stopped before starting routine introduction of naïve replacement gilts. Success of this approach, like all others, depends upon how complete the MLV immunity cross-protects against the WT PRRSV infecting the herd.

Killed PRRSV vaccine is safer because it can not shed to other animals in the herd. Killed vaccine is labeled for use in pregnant animals and therefore can be used without question in mass vaccination programs. Some producers have used mass vaccination with killed vaccine to stop virus circulation in the herd before starting PRRSV eliminations in their herds. There has been much debate with limited scientific evidence that killed vaccines can stimulate effective immunity alone. However, there are a couple of studies that indicate killed PRRSV vaccine appears to boost the immune response of pigs previously infected with live PRRSV. This effect may be dependent upon the % genetic homology between the herd's WT PRRSV and the killed PRRSV vaccine.

Success of any of the discussed vaccination options depends upon whether the vaccine is genetically similar enough to the herd virus to stimulate a protective immunity. Some wild viruses are similar enough that the vaccine will work, others appear not to be. The biggest frustration for veterinarians is that the information in PRRSV genetic sequence reports cannot be used to accurately predict whether vaccine will effectively cross-protect against the strain of virus infecting their client's herd (see above discussion).

Herd closure or depopulation / repopulation have been used to eliminate PRRSV from infected herds. Herd closure is most successful in farrowing only herds (no on-site nursery or finishing pigs) which have not had evidence of active PRRS reproductive disease or seroconversion in offsite nursery pigs for over a year. PRRSV-free sentinel gilts or vasectomized boars can be used to check the sow herd for PRRSV circulation before starting the elimination program. These herds are closed to new additions for approximately 140 days during which PRRSv free replacement gilts are bred offsite. The first of the offsite bred gilts are scheduled to farrow 6 weeks after the last of the on-farm bred gilts have farrowed. PRRSV free gilts are continually added and previously infected sows culled naturally until the herd is populated with only PRRSV-free sows. The biggest challenge is getting PRRSV to stop circulating in the sow herd before starting a herd closure project. This is very difficult in large herds, and herds which have onsite nursery or finishing pigs. In these cases total depopulation of the herd will eliminate PRRSV. If possible, depopulation of nursery and finisher buildings, subsequent sale of all weaned pigs and herd closure for 200 days may also successfully eliminate PRRSV from one-site production herds. Replacement gilts should be bred offsite to minimize the down time between farrowings. Depopulation appears to be the only viable option for herds infected with multiple strains of PRRSV where vaccination or gilt acclimatization has failed to control reproductive and finisher disease problems. Integrated

pig production company Production Managers estimate the costs of total depopulation can be recovered **if the herd can remain PRRSV-free for one year**.

Serum therapy or VLV immunization is a desperate yet logical procedure being used in herds to insure exposure of replacement gilts in isolation to acclimatize them to the herd's strain of WT PRRSV. It is a form of autogenous vaccination which is intended to ensure stimulation of homologous (full) immunity against the herd virus in a short period of time. This procedure is most effective and predictable in herds infected with a single strain of PRRSV. It is chosen by herds which are certain that all other options including vaccination cannot control their PRRS disease problems. This procedure has definite risk since gilts are being infected with live WT virus in the serum from infected pigs from the producer's herd. The greatest risk is bringing in inoculated gilts into the herd too soon such that they are still shedding the virus (still persistently infected) to sows or to their own piglets *in utero*. Additionally, the isolation /acclimatization unit must be run all-in all-out to minimize the risk of virus shedding gilts and continuous mutation of the herd's WT PRRSV. Other producers have considered using serum immunization of all sows in the herd during PRRS rebreaks (SAME virus causing reproductive disease that originally infected the herd) to make sure all sows are exposed, and all become immune simultaneously. This provides an opportunity for farrowing-only herds to eliminate PRRSV if 4 to 6 months of replacement gilts can be obtained, exposed to the outbreak virus, and the herd closed for 200 days. At the end of this time period negative sentinels are added to check for virus circulation. If no PRRSV circulation is present, then regular introduction of PRRSV-free gilts is started as described in Herd Closure above. **Serum immunization of pregnant sows will likely cause abortion in some later-term sows or gilts and also infection of piglets *in utero* that will probably cause PRRS-associated disease problems in the nursery and finisher.** This is a desperate measure to be considered only as a last resort. The amount of losses are difficult to predict, and should be weighed against the cost of depopulation of the herd. Elimination of PRRSV from the nursery and finisher sites will have to be accomplished, probably by depopulation or production breaks, to gain full economic benefit of herd closure following a re-break or initial outbreak.

Alternatively, the advantage of serum immunization is that during outbreaks, it ensures both a whole-herd exposure to the PRRSV and also brings a quicker end to abortions, weak-born and mummified piglets. In turn a quicker end to viremic groups of weaned pigs is achieved and thereby nursery / finisher pig PRRS losses. Therefore, while this process MAY increase the total number of aborted litters, it **ensures all sows are exposed at the same time.** Otherwise some sows in mid-gestation (feti apparently not susceptible) which do not initially get exposed, will become exposed later, now during late gestation when their piglets are susceptible to infection. These piglets, born into later production groups, will be viremic due to *in utero* infection, and will increase the number of production groups that are infected and affected by PRRS. Therefore, it has been observed that serum therapy or VLV inoculation of pregnant sows and 4 months of replacement gilts combined with herd closure during PRRS outbreaks will decrease BOTH the number of breeding groups that have abortions and certainly that have piglets infected *in utero*, ultimately stopping PRRS-affected production groups much sooner.

CONCLUSIONS

This presentation purposely does not advocate one control strategy over another. Decisions of which to use can be complex and must be tailored to each individual herd situation. Factors such as number of strains which infect the herd, breeding stock source(s) PRRSV status, availability of isolation and acclimatization facilities, density of pig production in your area, economic status of the herd, risk aversion (or desperation), production type / flow, herd size, biosecurity measures used, etc. need to be weighed. Decisions must be made based upon collection of all needed information to answer these and other questions. What makes PRRS challenging to control is that this information (herd PRRSV circulation status in particular) can change over time, and hence, affect which control methods to use and their likely success.

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APPENDIX

McREBEL

LIMITED CROSSFOSTERING PRODUCTION PROCEDURES

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1) Don't crossfoster piglets after 24 hours of age

a) move the minimum number of pigs necessary to load functional teats
b) don't crossfoster to create uniform size or sex litters
c) when EXTRA medium or large pigs must be moved, do match them by size and milking ability of receiving sows and litter
d) ensure smallest piglets are given lowest priority for functional teat assignment, leave on birth sow or move as Aextras@ when more piglets than available teats

MAXIMIZE THE NUMBER OF PIGLETS REMAINING ON THEIR BIRTH MOTHER!

Otherwise, maximize the number of piglets remaining on colostrum mother.

2) Don't move piglets between rooms

a) follow strict All In - All Out production

THE LITTER IS NOW THE ALL IN - ALL OUT UNIT!

3) Remove very sick, moribund, or bad body condition pigs from the system

a) sell or eliminate piglets at weaning that are too light to survive in the nursery and have poor body condition
b) eliminate immediately piglets that don't quickly get better after treatment
c) eliminate very thin, starve-out, lame, light body weight, long-haired, chronically sick piglets as they are found

A PIGLET HELD-BACK FROM WEANING TAKES A TEAT AWAY FROM A YOUNGER, POTENTIALLY HEALTHIER PIG!

4) Nursery care practices to maximize piglet survival and performance

a) size piglets into pens carefully
b) place smallest piglets in warm, non-drafty part of room
c) hand feed smallest piglets 4 times a day for 5 days
d) switch rations based upon weight of pen, not room
e) use heat lamps and / or plastic lying pads for small piglets
f) lower one nipple / pen and jam it open for the first 24 hours to help piglets find water.

DON'T EXPECT TO WEAN ANY MORE QUALITY PIGLETS THAN THERE ARE FUNCTIONAL TEATS IN A FARROWING ROOM.
TO MAXIMIZE THE NUMBER OF PIGLETS WEANED PER ROOM, MAXIMIZE THE NUMBER OF FUNCTIONAL TEATS BY PROPER GILT SELECTION AND SOW CULLING.