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# Gilt introduction in PRRSV-positive systems

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## Introduction

Gilt introduction has become a key step for controlling PRRS. In the mid 1990s, Dee et al. (1996) described the existence of subpopulations of PRRSV-negative animals in PRRSV-positive herds. Presence of subpopulations were associated with the inconsistent introduction of PRRSV-exposed gilts (Dee et al., 1997); that is, in a given herd, gilt replacements were not all of the same immunological status at entry. Some gilt groups may have been introduced as PRRSV-negative, while others would have been PRRSV-positive. In turn, this created the presence of subpopulations or areas in the breeding herd that could perpetuate virus circulation. This virus recirculation would potentially result in the presence of clinical signs in the breeding, gestating, and farrowing areas as well as nursery and growing pigs.

In order to avoid economic losses associated with the destabilization of the herd, it was clear that replacement introduction should be managed accordingly. Gilts should be introduced all as either PRRSV-positive or PRRSV-negative, and PRRSV status for gilt introduction should be consistent month after month.

When we think about gilt introduction for PRRSV, gilts can be introduced either as PRRSV-positive or PRRSV-negative. PRRSV-negative gilts should be introduced into PRRSV-negative systems or systems undergoing PRRSV elimination programs. PRRSV-positive replacements may be preferred in PRRSV-positive systems where control of PRRSV infection without elimination of the virus is the objective. In this situation, PRRSV-negative gilts should be used as the starting point and should be converted to PRRSV-positive prior to introduction. In addition, producers can be certain that new viral strains are not being added only if PRRSV-negative replacements are used.

## Introduction of replacement gilts into PRRSV-positive herds

How should gilts be introduced? How can we make sure that they are exposed to the farm's pathogens and have had enough time to develop immunity?

*PRRSV-negative gilts are a must.* PRRSV infection in negative animals is expected to result in a traditional immunological response with the subsequent viremia, development of antibodies, and recovery. Exposure in previously PRRSV-positive animals may result in a less consistent response as described by Batista et al. (2002). If gilts are selected from the farm's finisher or gilt grow-out and are PRRSV-positive, they should not be exposed further and should be taken into quarantine and allowed to recover from infection. PRRSV is expected to be eliminated from the gilt's body during this time. In addition, gilts are expected to develop protective immunity against the PRRSV strain responsible for the original infection. In this scenario, replacement animals should be moved as all-in/all-out groups through the quarantine stage.

The following question still remains: How can we expose negative gilts to PRRSV consistently?

As an exposure source, the following options can be considered:

- Adult sows (from a farm's gilt grow-out): This is convenient, but exposure is not reliable; animals are exposed at different times, and some are not exposed. Adult animals are not a good source of virus since they may have already recovered from infection and eliminated the virus. They simply are not shedding anymore. This strategy may only work temporally after a recent PRRSV infection.
- Cull sows in quarantine: Exposure may be very inconsistent in this case as well. Only young, culled gilts may still be shedding virus, but the farm's supply may be inconsistent.
- Nursery pigs: Better than culled sows, but difficult to guarantee good nose-to-nose contact between animals of different ages. Pigs in early grower stage could also be considered. It is recommended that the nursery and growing stages be monitored to better determine time of seroconversion and virus recirculation. Seeder pigs for gilt exposure should be selected at the beginning of the seroconversion period (7-10 days post-infection) to allow maximum virus shedding when mixing with the negative animals happens.

- Tissues from PRRSV-infected animals: This source of material could be considered after a recent break. Processed tissues could be given as feed-back. Under those circumstances, it is advisable to collect blood and tissues for storage (via freezing).

All these systems above expose gilts to other pathogens in addition to PRRSV. Also, exposure is still not guaranteed, since it depends of a successful contact transmission. Other methods are used across the industry, although their efficacy and safety may be questionable.

- Modified live vaccine: This is the ideal tool needed for gilt acclimatization (“virus in a bottle”). This method would insure universal, one-time exposure at the chosen time. The major inconvenience would be the limited cross-protection that exists between field strains of PRRSV.
- Purified virus from culture: This method may be illegal, certainly if used across sow farms, even within companies. Problems with this protocol are mostly associated with the cells used to culture the virus, which may be contaminated with other agents. The advantage here would be the exposure to the system’s PRRSV strain. However, most commercial systems may have multiple PRRSV strains, and the decision must then be made as to which “appropriate” PRRSV strain to select. Iatrogenic administration of more than one strain is not recommended due to the possibility of recombination.
- Nursery pig serum: This may be legal as long as its used in-farm and not across State borders. The inconvenience here would, again, be selecting viremic nursery pigs. Another serious consequence could be the possibility of spreading other pathogens also present in the blood and not desired in the production system.

When any of the methods mentioned above are used, they should be used in discrete all-in/all-out groups, similar to the method described as “mass exposure.” In addition these methods may also be problematic in the face of exotic disease outbreaks and in “needle” transmitted microorganisms such as PRRSV. Also, according to research data from experimentally infected animals, duration of viremia may be extended when the virus is given intramuscularly as opposed when it is given intranasally. Intranasal exposure should be considered as an option, although consistent administration will require more specialized handling.

*Age of exposure* is also a very important factor to consider. In the case of PRRSV, animals may remain viremic or persistently infected for prolonged periods of time. Experimentally, animals have been found to be carriers for periods longer than 100 days. In addition, successful contact transmission has been possible for up to 80 days.

All these factors must be taken together; it is important to emphasize early exposure of the replacement animals.

- Replacement animals should be exposed early in life (maximum of four months of age) to a consistent viral source.
- Replacement animals should be all exposed at a single point in time; exposure should cease after that.
- Replacement animals need a minimum of three months to recover. In the past, quarantine was recommended for at least 60 days. Newer data may suggest 60 days quarantine was not sufficient, and extension to 90 days should be seriously considered.

In addition, to appropriately acclimatize gilts to PRRSV, we cannot forget simultaneous exposure to other pathogens and protection. As gilts are moved and isolated at a younger age, naïve populations to other microorganisms are being created. A clear example is the case for parvovirus. Susceptible, non-exposed populations of pigs to parvovirus may be created under such circumstances. It is therefore crucial to design comprehensive vaccination programs directed to protect against a broad spectrum of economically significant pathogens beyond PRRSV.

## **Summary**

In summary, gilt acclimatization becomes a challenge when a sustainable and consistent source of virus is not available. Even when the source of virus is available, ensuring proper exposure may also be difficult. Successful gilt acclimatization is potentially hampered by the presence of limited protection against different PRRSV strains present in the herd. As general guidelines, PRRSV-negative gilts are recommended as the starting animal for gilt acclimatization. Gilts should be exposed as early as possible in age to allow enough time for the animals to become infected, immune, and to eliminate the virus from their body. In addition, exposure to the farm’s PRRSV strains should provide the best results for gilt acclimatization, although the delivery mechanism may be unclear.

In addition, a successful gilt acclimatization will not be complete unless it targets other pathogens of interest specific to that herd. Proper vaccination programs also need to be implemented.

## **References**

- Dee SA, Joo HA, et al.(1996). Detecting subpopulations after PRRSV infection in large breeding herds using multiple serologic tests. *Swine Health and Prod.* 4:181-184.
- Dee SA, Joo HS, Pijoan C (1997). Controlling the spread of PRRSV in the breeding herd through management of the gilt pool. *Swine Health and Prod.* 3:64-69.

Batista L, Pijoan C, Torremorell M (2002). Experimental injection of gilts with porcine reproductive and respiratory syndrome virus (PRRSV) during acclimatization. *J Swine Health Prod.* 10(4):147-150.

