

Gilt replacement strategies used in two swine production areas in Quebec in regard to porcine reproductive and respiratory syndrome virus

Marie-Ève Lambert, DVM, PhD; Martine Denicourt, DVM, MSc; Zvonimir Poljak, DVM, MSc, PhD; Sylvie D'Allaire, DVM, MSc, PhD

Summary

Objectives: To describe gilt replacement strategies in regard to porcine reproductive and respiratory syndrome virus (PRRSV) and to assess differences between high density (HD) and moderate density (MD) pig areas.

Materials and methods: A cross-sectional study was conducted in breeding sites located in an HD (n = 68) and an MD area (n = 52) in Quebec between May 2005 and August 2008. A questionnaire on strategies used to introduce replacement gilts was completed and PRRSV status was assessed by enzyme-linked immunosorbent assay or reverse-transcription polymerase chain reaction. Sites housing at least one pig positive

by either test were classified as PRRSV-positive. Strategies were described according to herd characteristics, PRRSV status, and area.

Results: Self-replacement and purchase of mature or immature gilts were observed on 37%, 35%, and 28% of sites, respectively. In positive sites purchasing mature gilts, 18% had a PRRSV-positive supplier, and gilts were introduced either directly into the sow herd (15%) or after isolation (41%) or acclimatization (44%). Most positive sites purchasing immature gilts practiced acclimatization (93%), either by commingling gilts with commercial pigs (93%) or inoculating serum (7%). Acclimatization processes were rarely monitored through diagnostic

procedures. Lower sow inventory, higher prevalence of PRRSV infection, and higher frequency of self-replacement were observed in the HD compared to the MD area. Negative and positive sites practicing voluntary exposure to PRRSV both clustered spatially within the MD area.

Implication: Replacement strategies may have weaknesses that should be addressed to facilitate PRRSV management at the herd and regional levels.

Keywords: swine, porcine reproductive and respiratory syndrome virus, gilt, acclimatization, spatial cluster

Received: June 14, 2011

Accepted: May 11, 2012

Resumen - Estrategias de reemplazo de hembras primerizas, utilizadas en dos áreas de producción porcina en Quebec, en relación al virus del síndrome reproductivo y respiratorio porcino

Objetivos: Describir las estrategias de reemplazo de hembras primerizas en relación al virus del síndrome reproductivo y respiratorio porcino (PRRSV por sus siglas en inglés) y valorar las diferencias entre las áreas de alta densidad porcina (HD por sus siglas en inglés) y densidad moderada (MD por sus siglas en inglés).

Materiales y métodos: Entre Mayo 2005 y Agosto 2008, se realizó un estudio transversal, en sitios de gestación y maternidad localizados en un área de HD (n = 68) y un área de MD (n = 52) en Quebec. Se elaboró un cuestionario sobre estrategias utilizadas para introducir hembras de reemplazo y se valoró el estatus de PRRSV por medio de la prueba de ensayo por inmunoadsorción ligado a enzimas o la reacción en cadena de polimerasa de transcriptasa reversa. Los sitios que albergaron, al menos un cerdo positivo de cada una de estas pruebas se clasificaron como positivos al

PRRSV. Las estrategias fueron descritas de acuerdo a las características del hato, estatus de PRRSV, y áreas.

Resultados: En 37%, 35%, y 28% de los sitios, se practicó el auto reemplazo y compra de hembras maduras o inmaduras, respectivamente. En los sitios positivos que compraron hembras maduras, el 18% tenía un proveedor positivo al PRRSV, y las hembras se introdujeron directamente al hato (15%) o después del aislamiento (41%) o de la aclimatación (44%). La mayoría de los sitios positivos que compraban hembras inmaduras, practicaron la aclimatación (93%), ya fuera al mezclar las hembras con cerdos comerciales (93%) o inoculando suero (7%). Rara vez se monitorearon los procesos de aclimatación a través de procedimientos de diagnóstico. Se observó un menor inventario de hembras, alta prevalencia de la infección de PRRSV, y una frecuencia más alta de auto reemplazo en el HD comparado con el área MD. Los sitios positivos y negativos que practicaron la exposición voluntaria al PRRSV se agruparon espacialmente dentro del área MD.

MEL, MD, SDA: Faculty of Veterinary Medicine, University of Montreal, St-Hyacinthe, Quebec, Canada.

ZP: Department of Population Medicine, Ontario Veterinary College, University of Guelph, Ontario, Canada.

Corresponding author: Dr Marie-Ève Lambert, Faculté de médecine vétérinaire, CP 5000, St-Hyacinthe, QC J2S 7C6, Canada; Tel: 450-773-8521 ext 8674; Fax: 450-778-8120; E-mail: marie-ve.lambert@umontreal.ca.

This article is available online at <http://www.aasv.org/shap.html>.

Lambert ME, Denicourt M, Poljak Z, et al. Gilt replacement strategies used in two swine production areas in Quebec in regard to porcine reproductive and respiratory syndrome virus. *J Swine Health Prod.* 2012;20(5):223-230.

Implicación: Las estrategias de reemplazo pueden tener puntos débiles que deben manejarse para facilitar el manejo del PRRSV a niveles de hato y regional.

Résumé - Stratégies de remplacement des cochettes utilisées dans deux régions de production porcine au Québec relative-ment au virus du syndrome reproducteur et respiratoire porcin

Objectifs: Décrire les stratégies de remplacement des cochettes relativement au virus du syndrome reproducteur et respiratoire porcin (PRRSV) et évaluer les différences entre une région à haute densité porcine (HD) et une à densité porcine modérée (MD).

Matériels et méthodes: Une étude transversale a été menée sur des sites de reproduction situés dans une région HD (n = 68) et une région MD (n = 52) du Québec entre mai

2005 et août 2008. Un questionnaire sur les stratégies utilisées pour introduire les cochettes de remplacement a été complété et le statut relativement au PRRSV a été évalué par un test immuno-enzymatique ou une réaction d'amplification en chaîne par la polymérase utilisant la transcriptase réverse. Les sites hébergeant au moins un porc positif par l'une ou l'autre des deux épreuves étaient classés comme PRRSV-positif. Les stratégies étaient décrites en fonction des caractéristiques du troupeau, du statut PRRSV, et de la région.

Résultats: L'auto-remplacement et l'achat de cochettes matures ou immatures ont été observés sur, respectivement, 37%, 35%, et 28% des sites. Sur les sites positifs qui achetaient des cochettes matures, 18% avaient un fournisseur PRRSV-positif, et les cochettes étaient introduites soit directement dans le troupeau de truies (15%) ou après isolement

(41%) ou acclimatation (44%). La plupart des sites achetant des truies immatures pratiquaient l'acclimatation (93%) soit en mélangeant les cochettes avec des porcs commerciaux (93%) ou en inoculant du sérum (7%). Le processus d'acclimatation était rarement évalué par des procédures diagnostiques. Un inventaire plus faible en truies, une prévalence d'infection par PRRSV plus élevée, et une fréquence plus grande d'auto-remplacement étaient observés dans la région HD comparativement à la région MD. Des sites négatifs et positifs pratiquant une exposition volontaire au PRRSV étaient regroupés spatialement dans la région MD.

Implication: Les stratégies de remplacement peuvent avoir des faiblesses et devraient être examinées afin de faciliter la gestion du PRRSV au niveau du troupeau et à un niveau régional.

Porcine reproductive and respiratory syndrome (PRRS) is a viral disease that has a major economic impact on the swine industry.¹ In breeding herds experiencing clinical outbreaks, decreased farrowing rates, premature farrowings, abortions, heterogeneous litters composed of stillborns, mummies, or weak piglets, and increased pre-weaning mortality can be observed.² Once introduced into a herd, the virus can be horizontally transmitted through direct contact with various fluids from infected pigs or through an indirect pathway, eg, aerosols, insects, or fomites such as boots, coveralls, or needles.³⁻⁸ Viremic sows can also transmit the infection to their progeny either transplacentally or through direct contact with vaginal, mammary, or other secretions.⁹ Viral circulation can be further maintained by persistently infected pigs, complicating PRRS management.¹⁰

Gilt replacement represents a real challenge for on-farm prevention and control of PRRS virus (PRRSV). Indeed, PRRSV-positive replacement animals represent a risk factor for not only introducing the strain to negative herds, but for re-infecting positive herds, since only partial immune response has been reported after such heterologous challenge infections.¹¹⁻¹⁴ Combined with the marked genetic and antigenic diversity among PRRSV strains, this lack of cross-protection precludes complete effectiveness of disease control through the sole use of commercially available modified-live vaccines, which are

each composed of a single virus strain.¹⁵ Consequently, preventive measures must be established to avoid introduction of new PRRSV strains into a herd. Furthermore, a producer with a PRRSV-positive sow herd buying PRRSV-negative replacements without introducing gilts properly could enhance circulation of the endemic viral strain. Gilt acclimatization is actually the most common and effective strategy to address this problem.^{16,17} It can be defined as voluntary exposure of incoming gilts to the herd's PRRSV endemic strain, followed by a cool-down period, which represents the interval between the cessation of exposure and entrance of gilts into the sow herd. This period is necessary to ensure that gilts are no longer shedding the virus when introduced into the herd.^{18,19}

Several gilt replacement strategies are performed in the field. However, noncompliance with basic principles could seriously compromise PRRSV management at the herd or regional level. The objectives of this study were to describe replacement strategies used for gilts in regard to PRRSV and to assess regional differences between a high-density (HD) and a moderate-density (MD) pig area.

Materials and methods

This study was approved by the Comité d'éthique de l'utilisation des animaux of the University of Montreal.

Study design and source population

As part of a larger study on the epidemiology of PRRSV, a cross-sectional study was conducted on strategies used to introduce replacement stock in sites located in two regions of Québec, a province in eastern Canada, between May 2005 and August 2008. An HD area was selected which corresponded to 10 adjacent municipalities located in the Monteregie administrative region. The entire Estrie region of Québec was selected as an MD area to ensure a comparable number of breeding sites in both areas for comparison of several potential risk factors.^{20,21}

The unit of interest was the production site, defined as one or more barns located within 300 m of one another, belonging to the same owner (individual or corporate), and having the same animal source(s). In order to select sites, all producers listed in the Fédération des producteurs de porcs du Québec database and located in the HD or MD area were contacted by mail. This list included every producer with at least CaD \$5000 annual income from agriculture. A written description of the project and a participation form to be signed and returned were initially sent by mail. Participation was voluntary. Producers who did not respond or refused to participate were contacted by phone to inquire about their participation or to follow up on the reasons for their refusal.

Questionnaire

A questionnaire with semi-closed and open questions was developed to assess and describe the different strategies of replacement. Four veterinarians specialized in swine production and three producers were consulted to assess relevance, clarity, and completeness of questions. The questionnaire was filled out by the first author during a 15-minute in-person interview with the owner of each independent farm or with the site manager of farms under contract. On a few occasions, the questionnaire was completed during a telephone interview because the producer was not available for a visit on the site. Information was gathered on several herd characteristics, such as sow and pig inventory, type of production, ownership, and commercial PRRSV vaccines used on the site. Questions also addressed self-replacement or purchase of gilts, approximate weight of gilts at introduction, source(s) of gilts, PRRSV status of supplier (defined in a broad sense according to the best knowledge of the producer), interval between gilt introductions, number of gilts per group, use of back-to-back transportation (transfer of gilts from the supplier's truck to the owner's truck), diagnostic procedures performed on gilts, and strategies used to introduce them into the sow herd. Self-replacement referred to sites that had not purchased gilts from external sources for at least 6 months prior to the survey. When gilts were provided by an external source to the site, introduction strategies were investigated. Isolation was defined as a period of time that purchased gilts were segregated before being introduced into the sow herd, without voluntarily exposing them to endemic pathogens from the recipient herd. In contrast, acclimatization referred to the process where a voluntary exposure of purchased gilts to the herd endemic pathogens (eg, direct or indirect contact with live animals, serum inoculation) was allowed prior to their introduction into the sow herd. This latter strategy was combined or not with a cool-down period. Another strategy was to introduce purchased gilts directly into the sow herd, without any isolation or acclimatization process. Questions pertained to practices at the time of questionnaire completion or during the previous 6 months, depending on the questions. Geographical coordinates of the site (latitude, longitude) were obtained using a global positioning system (GPS).

Herd PRRSV-status assessment

PRRS virus status was determined by a commercially available enzyme-linked immunosorbent assay (ELISA) (Idexx HerdChek PRRS 2XR; Idexx Laboratories Inc, Westbrook, Maine) or by reverse-transcription polymerase chain reaction (RT-PCR) (Qiagen OneStep RT-PCR Kit, Qiagen Inc, Mississauga, Ontario, Canada). Animal sampling strategy within each herd and laboratory analyses varied according to PRRSV history and clinical signs as reported by the producer. Sampling was performed on the farm following questionnaire completion or shortly thereafter. Sites housing at least one pig positive for PRRSV or antibodies were classified as PRRSV-positive.

Sites with clinical history of PRRS. For sites with clinical signs compatible with PRRS at the time of sampling, animals were sampled in order to confirm PRRSV infection and also to maximize the probability of identifying a PRRSV strain needed for another part of the larger project.²² We collected either tissue (pooled lungs, tonsils, and tracheobronchial lymph nodes) from one to three dyspneic suckling piglets, weaners, or finishers, or sera from sows that had aborted, according to the stage of production most clinically affected by the disease. In the absence of clinical signs compatible with PRRS at the time of sampling, a minimum of 10 blood samples were drawn from animals at higher risk of viremia (gilts recently introduced into the breeding site or piglets in mid-nursery) and were pooled (maximum five samples per pool) for further analyses. Ribonucleic acid was extracted from sera or homogenates of lungs, tonsils, and lymph nodes with a QIAamp Viral RNA Mini Kit according to the manufacturer's instructions. Reverse-transcription PCR was accomplished using the Qiagen OneStep RT-PCR Kit and primers 5FN and 5DN under PCR conditions for detection of viral RNA as described by Larochelle et al.²³ In the presence of an RT-PCR-negative result, additional samples (from sera or from necropsy samples of lungs, tonsils, and lymph nodes) were tested using RT-PCR when pigs showing clinical signs were available for necropsy. If RT-PCR results were again negative, or in the absence of clinical signs, the same strategy was used as that described for sites without history of PRRS.

Sites without history of PRRS. When no clinical history of PRRS was reported by the

producer and no PRRSV commercial vaccination was performed on the site, samples were taken in order to confirm absence of infection. For that purpose, 30 sows of various parities were conveniently selected and blood sampled. This strategy allowed for detection of infection in larger sites, assuming a 10% seroprevalence with a 95% herd confidence level. Sera were tested for antibodies as originally described by Albina et al²⁴ and performed as recommended by the manufacturer (Idexx HerdChek PRRS 2XR). Seropositivity threshold was fixed to a sample-to-positive (S:P) ratio ≥ 0.4 . When two or fewer sample(s) of 30 were positive, samples were retested by ELISA. The site was classified as positive when at least one sample was ELISA-positive after retesting.

In order to describe replacement strategies according to PRRSV status, sites were classified as PRRSV-positive if at least one RT-PCR-positive or ELISA-positive result was obtained. Otherwise, they were classified as PRRSV-negative. All diagnostic procedures were performed at the Faculty of Veterinary Medicine of the University of Montreal in St-Hyacinthe, Quebec.

Statistical analyses

Validation and descriptive statistics were performed on data using SAS version 9.1 software (SAS Institute Inc, Cary, North Carolina). Sites having more than one missing value were excluded from subsequent analyses. Gilt introduction strategies according to PRRSV status of the recipient herd, type of gilts purchased (self-replacement, purchase of mature or immature gilts), and herd characteristics were described. For PRRSV-positive sites purchasing and acclimatizing gilts, differences in the acclimatization process (exposure and cool-down period) between types of gilts purchased were assessed using the Pearson exact chi-square ($\alpha = .05$) for categorical variables²⁵ and the Wilcoxon rank sum test ($\alpha = .05$) for continuous data. Differences between the HD and MD areas were also assessed using the same statistical procedures described above regarding herd characteristics, type of gilts purchased, gilt introduction strategies, PRRSV status, PRRSV commercial vaccination of gilts or sows, and exposure of gilts to the herd's endemic PRRSV or modified-live PRRSV vaccine strain(s).

Spatial analyses

PRRS virus-negative sites, PRRSV-positive sites not exposing gilts, and PRRSV-positive

sites exposing gilts to herd endemic strains or to one or more modified-live vaccine strains were geographically interpolated using the Thiessen polygon method in ArcInfo version 9.3 software (Esri, Redlands, California) to preserve confidentiality of producers. The area perimeter was hand-defined in order to produce a compact zone comprising all sites and excluding areas with no sites. A small gap was allowed between sites and area boundaries. Spatial clusters of PRRSV-negative sites or of PRRSV-positive sites exposing gilts were assessed using the spatial scan test performed in SaTScan version 8.0 software (Boston, Massachusetts), based on a purely spatial Bernoulli distributional assumption model and scanning for circular clusters using a 50% population size maximal threshold. Analyses were performed separately for each area. The first analysis considered PRRSV-negative sites to be cases and PRRSV-positive sites to be controls. In the second analysis, the cases were PRRSV-positive sites exposing gilts and the controls were the remaining sites (eg, PRRSV-positive not exposing gilts and PRRSV-negative sites). The statistical significance of clusters was determined through 9999 permutations and the most likely significant cluster ($P < .05$) was mapped.

Results

Overall participation was 77%.²¹ Four sites that each had more than one missing value were excluded. A total of 120 breeding sites (HD = 68, MD = 52) having a median (Q1-Q3) sow inventory of 196 (139-333) were included in the study population. Included sites were either farrow-to-finish (69%), farrow-to-grow (11%), or farrow-to-wean (20%), and most sites (90%) were managed by an independent producer. PRRS-virus status was positive in 85% of the sites (102 of 120). PRRS-virus commercial vaccination was routinely used on gilts or sows in 30% of the sites (36 of 120). On these 36 sites, either both sows and gilts (75%), gilts only (22%), or sows only (3%) were vaccinated. Sites where commercial vaccinations were performed used live attenuated vaccines, mainly (83%) modified live virus (MLV) strain (Ingelvac PRRS MLV or ReproCyc PRRS-PLE; Boehringer Ingelheim Canada Ltd, Burlington, Ontario, Canada), with 11% of sites using atypical virus strain (Ingelvac PRRS ATP; Boehringer Ingelheim Canada Ltd), and two sites using both PRRSV vaccine strains (6%). Self-replacement and purchase of mature

(≥ 95 kg) or immature (< 95 kg) gilts were observed on 37%, 35%, and 28% of the 120 sites, respectively. Self-replacement ($n = 44$) was mainly performed on independently owned (100%) and farrow-to-finish sites (89%) having a median (Q1-Q3) sow inventory of 158 (108-225). When immature or mature gilts were bought from an external source ($n = 76$), 97% of the sites had only one supplier. Immature gilts (34 of 76 sites) were purchased on average at 6-week intervals at a median (Q1-Q3) weight of 5 kg (5-8 kg), and 90% of the sites bought gilts at < 20 kg. Mature gilts (42 of 76 sites) were bought on average at 7-week intervals at a median (Q1-Q3) weight of 114 kg (110-120 kg). Back-to-back transportation was applied for gilt delivery on 26% of the sites, with the proportion differing significantly between farms buying immature (44%) and mature gilts (14%) ($P < .01$; Pearson exact chi-square test).

Table 1 shows gilt introduction strategies used in PRRSV-positive ($n = 102$) and negative sites ($n = 18$) according to the type of gilts purchased. In PRRSV-negative sites, all purchased gilts were from PRRSV-negative sources. In PRRSV-positive sites, all immature gilts ($n = 31$) were purchased from a negative supplier, whereas 18% (six of 34) of the sites buying mature gilts were supplied by a PRRSV-positive herd ($n = 5$) or received PRRSV-vaccinated gilts ($n = 1$). Sites purchasing mature gilts from PRRSV-positive external sources had different gilt introduction strategies. Among PRRSV-positive sites practicing isolation, acclimatization, or introducing their gilts directly into the sow herd, 63% (10 of 16), 30% (13 of 44), and 40% (two of five), respectively, also used PRRSV commercial vaccination of gilts. On both PRRSV-positive and PRRSV-negative sites, most isolation periods ($n = 19$) occurred on premises or in rooms adjacent to the principal unit (89%), and more than one third (37%) operated with a continuous flow without washing and disinfecting between groups of gilts. The median (Q1-Q3) duration of isolation was significantly different between producers purchasing mature and immature gilts ($P < .01$; Wilcoxon exact rank sum test), with a period of 7 (4-7) and 15 (14-17) weeks without any contact with animals from the recipient herd, respectively. Diagnostic procedures for PRRSV at the end of the isolation period and before entering gilts into the sow herd were routinely performed

on 11% (two of 19) of the sites, using ELISA or a combination of ELISA and RT-PCR.

An acclimatization process where mature or immature gilts were exposed to a PRRSV endemic strain before their introduction into the sow herd was observed on 43% (44 of 102) of the PRRSV-positive sites (Table 1). Exposure mostly occurred through direct or indirect contact with live animals only ($n = 38$), sometimes combined with feeding placenta ($n = 3$), or by serum inoculation ($n = 3$). All sources of exposure were provided by the recipient herd. Among the three sites performing serum inoculation, two purchased immature gilts and exposed them in off-site facilities, observing an all-in, all-out (AIAO) pig flow and a cool-down period of 30 weeks. On the other site, mature gilts were inoculated within the principal unit and the pig flow was continuous. Table 2 shows descriptive statistics on exposure and cool-down periods as practiced on PRRSV-positive sites exposing gilts to live animals (41 of 44). To expose mature gilts ($n = 14$), direct or indirect contact was promoted with one or more animals from the following categories: culled sows or boars, finishers, weaners, or suckling pigs. This exposure period was mostly carried out within the principal unit (79%; 11 of 14) and often performed with a continuous pig flow (64%; nine of 14). Table 2 also shows parameters for exposure of immature gilts ($n = 27$). On these latter sites, gilts were commingled with commercial pigs to expose them to the PRRSV herd-endemic strain during the weaner-finisher phase (81%; 22 of 27), the weaner phase only (15%; four of 27), or in a separated unit located on the site (4%; one of 27). When exposure occurred within the principal unit, gilts were housed in a pen separate from commercial pigs in 69% (18 of 26) of the sites. After the exposure period of immature gilts, 44% (12 of 27) of the sites applied a cool-down period (Table 2). This period was carried out in a gilt finisher unit, preventing further contact with commercial pigs for one third of the sites (four of 12). With the remaining two thirds (eight of 12), the cool-down most often took place in the principal unit (86%), often observing a continuous pig flow (38%). Among PRRSV-positive sites practicing acclimatization, only 23% (10 of 44) of sites monitored PRRSV health status before gilts entered the sow barn, using either ELISA (two of 10), RT-PCR (five of 10), or a combination of both methods (three of 10).

Table 1: Frequency distribution of gilt introduction strategies used for porcine reproductive and respiratory syndrome virus (PRRSV) in PRRSV-positive and PRRSV-negative breeding sites according to type of gilts purchased*

Gilt introduction strategies	Type of gilts purchased			
	None purchased†	Immature‡	Mature¶	Total
	n = 44	n = 34	n = 42	n = 120
PRRSV-positive sites (n = 102)				
Self-replacement	37	0	0	37
Isolation§	0	2	14	16
Acclimatization**	0	29	15	44
Introduction directly into the sow herd††	0	0	5	5
Subtotal	37	31	34	102
PRRSV-negative sites (n = 18)				
Self-replacement	7	0	0	7
Isolation§	0	0	3	3
Acclimatization‡‡	0	3	2	5
Introduction directly into the sow herd††	0	0	3	3
Subtotal	7	3	8	18

* Cross-sectional study in breeding sites in a high (HD) and a moderate density (MD) area in Quebec, Canada, May 2005 to August 2008. Producers in the Fédération des producteurs de porcs du Québec database were contacted. For each site, a questionnaire on gilt replacement strategies was filled out during in-person interview with the owner or site manager. Tissues or blood samples were collected and PRRSV status was assessed by enzyme-linked immunosorbent assay or reverse-transcription polymerase chain reaction. Sites housing at least one pig positive by either test were classified as PRRSV-positive.

† Self-replacement.

‡ Purchased from external source at a weight of < 95 kg.

¶ Purchased from external source at a weight of ≥ 95 kg.

§ Period without exposure to endemic PRRSV strain.

** Period with exposure to endemic PRRSV strain.

†† Introduction of gilts into the same air space as sow herd, without isolation or acclimatization.

‡‡ Period with exposure to endemic pathogens (except PRRSV).

Some differences between the HD (354 pigs per km²)²⁶ and MD areas (44 pigs per km²)²⁶ were obtained. A lower median sow inventory (175 versus 230), a higher prevalence of PRRSV-positive sites (94% versus 74%) and a higher frequency of breeding sites practicing self-replacement (46% versus 25%) were observed in the HD compared to the MD area ($P < .05$). No other variable differed significantly between areas. Geographical distribution of PRRSV-negative sites, PRRSV-positive sites exposing gilts to herd endemic or vaccine PRRSV strains, and PRRSV-positive sites not

practicing exposure are presented for HD and MD areas in Figure 1. In the northern part of the MD area, a cluster of PRRSV-negative sites ($P = .01$) in a 40-km radius was revealed in a grouping of eight sites, of which seven were PRRSV-negative (Figure 1). In the same area and with a 15-km radius, a significant spatial cluster of PRRSV-positive sites exposing gilts ($P = .04$) was also identified in a grouping of 13 sites, of which 12 exposed their gilts. No significant spatial cluster was identified in the HD area (Figure 1).

Discussion

Self-replacement was most commonly practiced by independently owned farrow-to-finish sites having a small sow inventory. This practice could have been adopted for economic reasons in that it reduces the cost of replacement, but it may also have been used as a preventive method to limit introduction of new PRRSV strain(s) through external sources of gilts. Herd closure with self-replacement of gilts could also have been implemented in response to a PRRSV problem, in an effort to stabilize infection within the sow herd.²⁷ This hypothesis would be in line with the higher frequency of self-replacement observed in the HD area, where a higher prevalence of the disease was observed.

Purchasing gilts from an external source, a practice that could put a herd at risk for introducing a new PRRSV strain, was observed on both PRRSV-positive and negative sites. Indeed, a few sites bought gilts from a PRRSV-positive source or did not require any isolation period. On some sites, mature gilts may be introduced directly into the sow herd because no facilities specifically designed for isolation are available, or due to time limitations in meeting breeding targets. Even if purchased from a certified PRRSV-negative supplier, gilts should not be introduced directly into the sow herd, because there is still a risk of contamination before departure or during transport if vehicles are not washed or disinfected properly.²⁸ For gilt deliveries, the back-to-back procedure can be used to lower the risk of PRRSV transmission between gilt supplier and commercial producer.²⁹ Furthermore, a certificate testifying PRRSV-negative status of incoming gilts, combined with a quarantine for disease detection, should be required to reduce the risk of PRRSV introduction.³⁰ In our study, although the length of the isolation period was sufficient for disease detection (≥ 7 weeks), other essential criteria to insure its effectiveness were seldom met. A quarantine should be in a location separated from the main unit and operated with an AIAO pig flow.^{17,31,32} Diagnostic procedures to detect viremia or seroconversion were under-utilized and should also be performed shortly after delivery to confirm negative status at arrival and before entering gilts into the sow herd, since the absence of clinical signs does not guarantee the absence of infection.³³

Several practices that may increase the risk of PRRSV circulation within positive breeding

Table 2: Frequency distribution of parameters regarding acclimatization process of gilts using contact with live animals in PRRSV-positive sites (n = 41 sites)*

Parameters of acclimatization process	Immature†	Mature¶
	n = 27	n = 14
Exposure		
Median (Q1-Q3) exposure time (weeks)	20 (16-24) ^a	7 (4-8) ^b
Cool-down		
No. of sites with cool-down	12 ^a	2 ^a
Median (Q1-Q3) cool-down time (weeks)	12 (7-17) ^a	4 (2-7) ^a

* Details of study described in Table 1.

† Purchased from external source at a weight of < 95 kg.

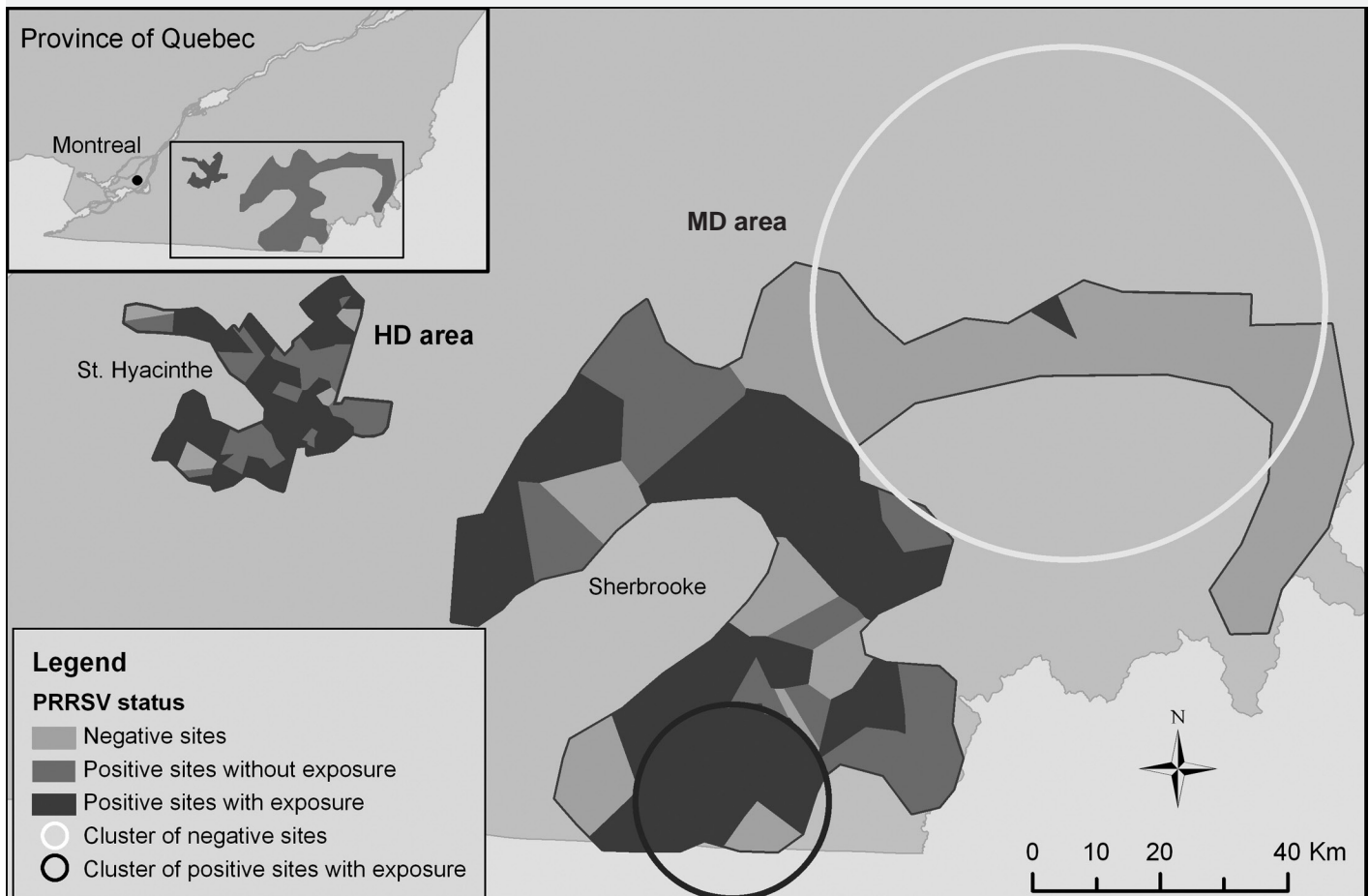
¶ Purchased from external source at a weight of ≥ 95 kg.

^{ab} Within a row, values with different superscripts are significantly different ($P < .05$; Pearson exact chi-square test for categorical data or Wilcoxon rank exact sum test for continuous variables).

PRRSV = porcine reproductive and respiratory syndrome virus.

sites were highlighted. Indeed, one third of the positive sites purchasing gilts from an external source did not attempt any acclimatization process, resulting in the entrance of non-immune subpopulations and potentially promoting PRRSV recirculation within the sow herd.¹⁷ Furthermore, when acclimatization was used, several basic principles were violated, compromising the efficacy and safety of the controlled process. Many factors might have influenced the success of gilt exposure, such as the type of “infectious” sources (culled sows, dyspneic piglets, placenta, serum), the method of exposure (direct or indirect contact, inoculation), or on-farm dynamic of PRRSV transmission.¹⁸ Location and pig flow during the exposure period should also be considered. On some sites, mature gilts were exposed to PRRSV-endemic strains in a room adjacent to the sow herd, potentially causing accidental re-infection of the sow herd.^{13,34} Furthermore, the lack of AIAO pig flow observed during the process does not allow control

Figure 1: Geographical distribution of swine breeding sites in the high density (HD) and moderate density (MD) areas according to their porcine reproductive and respiratory syndrome virus (PRRSV) status and the use of voluntary gilt exposure to PRRSV herd endemic or vaccine strains. The background map represents the Province of Quebec, Canada, with the two selected areas along with major cities. Table 1 shows details of the study.



of the duration and the end of the exposure period.¹⁸ Ideally, a timely and simultaneous exposure of all incoming gilts should be favored to determine more precisely the onset and duration of the cool-down period. This last step of the acclimatization process is essential to allow time for gilts to recover clinically, to develop adequate immune response, and to cease viral shedding before entering the sow herd.³⁴ The length of cool-down is therefore dependent on virus persistence in infected animals. In fact, PRRSV has been isolated and identified by RT-PCR in tonsils up to 157 and 251 days post infection, respectively.^{10,35} Unfortunately, only one third of the sites performing acclimatization by contact with live animals respected a cool-down period, and the length of the cool-down period was too short, especially for mature gilts.³⁶ On most sites, no diagnostic procedures were used to monitor the success of the acclimatization process. This could lead to introduction of either seronegative gilts (failure of exposure) or viremic gilts (failure of cool-down), both scenarios being a threat to PRRSV control.

The regional impact of exposing gilts to PRRSV endemic strains through an inadequate acclimatization process or exposing them to commercial vaccine strains is not largely documented in the literature. PRRSV virus endemic strains actively maintained within herds may contribute to the area spread of the virus between farms through aerosols, insects, vehicles, or other fomites in the absence of proper biosecurity.^{6,8,28,37} Furthermore, considering that vaccine strains can be shed, can persist in vaccinated animals, and can be transmitted to nonvaccinated pigs, their ability to circulate in the field also has to be considered.³⁸⁻⁴⁰ Difficulties in controlling PRRSV at the herd or regional level could also emerge from co-circulation of either wild-type or vaccine strains, since PRRSV is highly susceptible to mutations and even recombinations.^{41,42} Regional approaches to PRRSV control should therefore take into account exposure to PRRSV strain(s) within individual breeding sites. In the MD area, the presence of two spatial clusters, one with mainly negative sites and the other with sites practicing exposure to a wild-type or vaccine PRRSV strain, suggests the possibility of targeting smaller zones for interventions to facilitate disease control. In fact, whereas PRRSV-negative areas should be protected from virus introduction, the method of gilt exposure should be addressed for zones practicing exposure. In contrast, PRRSV management

in the HD area could be seriously impeded due to the greater density and proximity of sites which increase the probability of area spread, the higher prevalence of disease, and the random distribution of negative sites and of positive sites either practicing voluntary exposure or not doing so.

Assuming that some producers who could not be reached were out of business, we might have underestimated our participation rate. Nevertheless, the participation obtained improved the internal validity of the study, even if the total absence of selection bias cannot be warranted. Producers recorded on the Fédération des producteurs de porcs du Québec list from which the samples were selected were restricted to those having more than CaD \$5000 annual income from agriculture. Gilt replacement strategies discussed in the current paper might not represent those used on sites with lower or higher pig inventory. This study also principally included commercial breeding sites owned by independent producers located in two specific areas. Consequently, results should not be extrapolated to other areas or to multipliers or sites belonging to integrated systems that might have different introduction strategies due to the availability of external gilts and the facilities to implement these strategies.

A single interviewer performed all the questionnaires with people working directly on each site, and most questions referred to practices observed during the previous 6 months, minimizing recall bias. However, some misclassification bias regarding the PRRSV status of suppliers of replacement animals and the number of sources is possible, since responses given by the producers were not validated by gilt suppliers. Misclassification regarding herd PRRSV status should be minimal, considering the high sensitivity and specificity of serological testing (> 97% and > 99%, respectively).⁴³ Due to the absence of a standardized definition of the various gilt-introduction strategies in the field, definitions were set a priori. Descriptive results regarding the strategies are therefore conditional on the previous classification. Most discussion about practices that potentially risk re-circulating PRRSV within the sow herd pointed out gaps in the acclimatization process. However, since the project did not aim to evaluate the effect of every gilt introduction strategy on within-herd viral circulation, other strategies might also represent a certain risk. Finally, spatial

analyses are conditional on the subset of participating sites. According to the overall low standard of applying even basic principles of the acclimatization process, spatial cluster of PRRSV-positive sites practicing voluntary exposure might suggest higher potential for viral circulation and thus higher potential for infecting neighboring herds. However, additional studies are required in order to better quantify the impact of the acclimatization process on the neighborhood.

In conclusion, this study identified specific issues regarding gilt replacement that could increase the probability of either introducing a PRRSV strain into a herd or promoting the circulation of an endemic strain. Acclimatization, which should be an effective method for controlling PRRSV in a sow herd, was the most poorly applied gilt introduction strategy pertaining to basic principles, particularly for mature gilts. Voluntary exposure of gilts to either PRRSV herd-endemic or vaccine strains has to be considered in any targeted intervention to control the disease regionally. Producers have to be well informed about the steps and rules they should adhere to and be supervised by veterinarians in order to improve their success rates in stabilizing their herds and minimizing potential impact on the neighborhood.

Implications

- Under the conditions of this study, gilt replacement strategies may incorporate several weaknesses that should be addressed to facilitate PRRSV management at the farm or regional level.
- Producers should be informed about acclimatization techniques and comply with the steps and rules for introducing gilts in order to improve the success rate of the procedure in the field and also to minimize the potential impact on the neighborhood.

Acknowledgments

The authors would like to acknowledge the Fonds québécois de la recherche sur la nature et les technologies for a scholarship to the first author, and the Fédération des producteurs de porcs du Québec, the Conseil pour le développement de l'agriculture du Québec, the Centre d'insémination porcine du Québec, and many other swine industry partners for project funding. Special thanks to practicing veterinarians for encouraging producers to participate in the study and to all producers for their interest in the project and the time they donated.

References

1. Neumann EJ, Kliebenstein JB, Johnson CD, Mabry JW, Bush EJ, Seitzinger AH, Green AL, Zimmerman JJ. Assessment of the economic impact of porcine reproductive and respiratory syndrome on swine production in the United States. *JAVMA*. 2005;227:385–392.
2. Christianson WT, Joo HS. Porcine reproductive and respiratory syndrome virus: a review. *Swine Health Prod*. 1994;2:10–28.
3. Yoon KJ, Hoo HS, Christianson WT, Morrison RB, Dial GD. Persistent and contact infection in nursery pigs experimentally infected with porcine reproductive and respiratory syndrome (PRRS) virus. *Swine Health Prod*. 1993;4:5–8.
4. Wills RW, Zimmerman JJ, Yoon KJ, Swenson SL, Hoffman LJ, McGinley MJ, Hill HT, Platt KB. Porcine reproductive and respiratory syndrome virus: routes of excretion. *Vet Microbiol*. 1997;57:69–81.
5. Otake S, Dee SA, Jacobson L, Torremorell M, Pijoan C. Evaluation of aerosol transmission of porcine reproductive and respiratory syndrome virus under controlled field conditions. *Vet Rec*. 2002;150:804–808.
6. Otake S, Dee SA, Rossow KD, Deen J, Molitor TW, Pijoan C. Transmission of porcine reproductive and respiratory syndrome virus by fomites (boots and coveralls). *J Swine Health Prod*. 2002;10:59–65.
7. Otake S, Dee SA, Rossow KD, Joo HS, Deen J, Molitor TW, Pijoan C. Transmission of porcine reproductive and respiratory syndrome virus by needles. *Vet Rec*. 2002;150:114–115.
8. Otake S, Dee SA, Rossow KD, Moon RD, Pijoan C. Mechanical transmission of porcine reproductive and respiratory syndrome virus by mosquitoes, *Aedes vexans* (Meigen). *Can J Vet Res*. 2002;66:191–195.
9. Kranker S, Nielsen J, Bille-Hansen V, Bøtner A. Experimental inoculation of swine at various stages of gestation with a Danish isolate of porcine reproductive and respiratory syndrome virus (PRRSV). *Vet Microbiol*. 1998;61:21–31.
10. Wills RW, Zimmerman JJ, Yoon KJ, Swenson SL, McGinley MJ, Hill HT, Platt KB, Christopher-Hennings J, Nelson EA. Porcine reproductive and respiratory syndrome virus: a persistent infection. *Vet Microbiol*. 1997;55:231–240.
11. Mousing J, Permin A, Mortensen S, Bøtner A, Willeberg P. A case-control questionnaire survey of risk factors for porcine reproductive and respiratory syndrome (PRRS) seropositivity in Danish swine herds. *Vet Microbiol*. 1997;55:323–328.
12. Lager KM, Mengeling WL, Brockmeier SL. Evaluation of protective immunity in gilts inoculated with the NADC-8 isolate of porcine reproductive and respiratory syndrome virus (PRRSV) and challenge-exposed with an antigenically distinct PRRSV isolate. *Am J Vet Res*. 1999;60:1022–1027.
13. Pesente P, Rebonato V, Sandri G, Giovanardi D, Ruffoni LS, Torriani S. Phylogenetic analysis of ORF5 and ORF7 sequences of porcine reproductive and respiratory syndrome virus (PRRSV) from PRRS-positive Italian farms: a showcase for PRRSV epidemiology and its consequences on farm management. *Vet Microbiol*. 2006;114:214–224.
14. Prieto C, Alvarez E, Martinez-Lobo FJ, Simarro I, Castro JM. Similarity of European porcine reproductive and respiratory syndrome virus strains to vaccine strain is not necessarily predictive of the degree of protective immunity conferred. *Vet J*. 2008;175:356–363.
15. Meng XJ. Heterogeneity of porcine reproductive and respiratory syndrome virus: implications for current vaccine efficacy and future vaccine development. *Vet Microbiol*. 2000;74:309–329.
16. Dee SA, Joo H, Pijoan C. Controlling the spread of PRRS virus in the breeding herd through management of the gilt pool. *Swine Health Prod*. 1995;3:64–69.
17. Dee SA. Principles of prevention, control and eradication. In: Zimmerman JJ, Yoon KJ, eds. *2003 PRRS Compendium Producer Edition*. Des Moines, Iowa: National Pork Board; 2003:78–87.
18. Vashisht K, Erlandson KR, Firkins LD, Zuckermann FA, Goldberg TL. Evaluation of contact exposure as a method for acclimatizing growing pigs to porcine reproductive and respiratory syndrome. *JAVMA*. 2008;232:1530–1535.
19. Corzo CA, Mondaca E, Wayne S, Torremorell M, Dee S, Davies P, Morrison RB. Control and elimination of porcine reproductive and respiratory syndrome virus. *Virus Res*. 2010;154:185–192.
20. Lambert ME, Arsenault J, Poljak Z, D'Allaire S. Epidemiological investigations in regard to porcine reproductive and respiratory syndrome virus (PRRS) in Quebec, Canada. Part 2: Prevalence and risk factors in breeding sites. *Prev Vet Med*. 2012;104:84–93.
21. Lambert ME, Poljak Z, Arsenault J, D'Allaire S. Epidemiological investigations in regard to porcine reproductive and respiratory syndrome (PRRS) in Quebec, Canada. Part 1: Biosecurity practices and their geographical distribution in two areas of different swine density. *Prev Vet Med*. 2012;104:74–83.
22. Lambert ME, Arsenault J, Poljak Z, D'Allaire S. Correlation among genetic, Euclidean, temporal and herd ownership distances of porcine reproductive and respiratory syndrome virus strains in Quebec, Canada. *BMC Vet Res*. 2012;8:76. doi:10.1186/1746-6148-8-76.
23. Larochelle R, D'Allaire S, Magar R. Molecular epidemiology of porcine reproductive and respiratory syndrome virus (PRRSV) in Québec. *Virus Res*. 2003;96:3–14.
24. Albina E, Leforban Y, Baron T, Plana Duran JP, Vannier P. An enzyme linked immunosorbent assay (ELISA) for the detection of antibodies to the porcine reproductive and respiratory syndrome (PRRS) virus. *Ann Rech Vet*. 1992;23:167–176.
25. Stokes ME, Davis CS, Koch GG. *Categorical Data Analysis Using the SAS System*. Cary, North Carolina: SAS Institute Inc; 2000.
26. Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec (MAPAQ), FLORA: Identification and Registration of Québec Pig Herds Database. 2010.
27. Schaefer N, Morrison R. Effect on total pigs weaned of herd closure for elimination of porcine reproductive and respiratory syndrome virus. *J Swine Health Prod*. 2007;15:152–155.
28. Dee SA, Deen J, Otake S, Pijoan C. An experimental model to evaluate the role of transport vehicles as a source of transmission of porcine reproductive and respiratory syndrome virus to susceptible pigs. *Can J Vet Res*. 2004;68:128–133.
29. American Association of Swine Veterinarians (AASV). PADRAP: Production Animal Disease Risk Assessment Program. Available at: <http://www.padrapp.org/>. Accessed 29 May 2012.
30. Weigel RM, Firkins LD, Scherba G. Prevalence and risk factors for infection with porcine reproductive and respiratory syndrome virus (PRRSV) in swine herds in Illinois (USA). *Vet Res*. 2000;31:87–88.
- *31. Moore C. Biosecurity and minimal disease herds. In: Tubbs RC, Leman AD, eds. *Vet Clin North Am Food Anim Pract*. Philadelphia, Pennsylvania: W. B. Saunders Company; 1992:461–474.
32. Dee SA. An overview of production systems designed to prepare naive replacement gilts for impending PRRSV challenge: A global perspective. *Swine Health Prod*. 1997;5:231–239.
33. Cuartero L, Dee SA, Deen J, Ruiz A, Pijoan C. Association between clinical signs and high serum titers of porcine reproductive and respiratory syndrome virus (PRRSV) in nursery pigs under field conditions. *J Swine Health Prod*. 2002;10:118–121.
34. Batista L, Pijoan C, Torremorell M. Experimental injection of gilts with porcine reproductive and respiratory syndrome virus (PRRSV) during acclimatization. *J Swine Health Prod*. 2002;10:147–150.
35. Wills RW, Doster AR, Galeota JA, Sur JH, Osorio FA. Duration of infection and proportion of pigs persistently infected with porcine reproductive and respiratory syndrome virus. *J Clin Microbiol*. 2003;41:58–62.
36. Batista L, Dee SA, Rossow KD, Deen J, Pijoan C. Assessing the duration of persistence and shedding of porcine reproductive and respiratory syndrome virus in a large population of breeding-age gilts. *Can J Vet Res*. 2002;66:196–200.
37. Otake S, Dee SA, Corzo C, Oliveira S, Deen J. Long-distance airborne transport of infectious PRRSV and *Mycoplasma hyopneumoniae* from a swine population infected with multiple viral variants. *Vet Microbiol*. 2010;145:198–208.
38. Nielsen TL, Nielsen J, Have P, Bækbo P, Hoff-Jørgensen R, Bøtner A. Examination of virus shedding in semen from vaccinated and from previously infected boars after experimental challenge with porcine reproductive and respiratory syndrome virus. *Vet Microbiol*. 1997;54:101–112.
39. Mengeling WL, Vorwald AC, Lager KM, Clouser DF, Wesley RD. Identification and clinical assessment of suspected vaccine-related field strains of porcine reproductive and respiratory syndrome virus. *Am J Vet Res*. 1999;60:334–340.
40. Nielsen HS, Oleksiewicz MB, Forsberg R, Stadeljek T, Bøtner A, Storgaard T. Reversion of a live porcine reproductive and respiratory syndrome virus vaccine investigated by parallel mutations. *J Gen Virol*. 2001;82:1263–1272.
41. Kiss I, Sámi L, Kecskeméti S, Hanada K. Genetic variation of the prevailing porcine respiratory and reproductive syndrome viruses occurring on a pig farm upon vaccination. *Arch Virol*. 2006;151:2269–2276.
42. Shi M, Lam TT, Hon CC, Murtaugh MP, Davies PR, Hui RK, Li J, Wong LT, Yip CW, Jiang JW, Leung FC. Phylogeny-based evolutionary, demographical, and geographical dissection of North American type 2 porcine reproductive and respiratory syndrome viruses. *J Virol*. 2010;84:8700–8711.
43. Mateu E, Tello M, Coll A, Casal J, Martin M. Comparison of three ELISAs for the diagnosis of porcine reproductive and respiratory syndrome. *Vet Rec*. 2006;159:717–718.

* Non-refereed reference.

