

Porcine Herd Health Management Practices for the Control of PRRSV Infection

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1. Introduction

Porcine reproductive and respiratory syndrome (PRRS) is a highly contagious viral disease that was first recognized almost simultaneously in Western Europe (Wensvoort et al., 1991) and North America in the late 1980s (Keffaber, 1989). The causative agent is PRRS virus (PRRSV), a small single-stranded positive sense RNA virus, classified in the order Nidovirales, family Arteriviridae and genus Arterivirus. Since its appearance, PRRS has devastated the worldwide swine industry with tremendous economic losses (Neumann et al., 2005). PRRSV causes reproductive failure in breeding stock (e.g. premature farrowings, late term abortions, poor farrowing rate, mummified fetuses and stillborn piglets), as well as respiratory disease, elevated mortality and reduction of growth performance in piglets and growing / finishing pigs (Cho & Dee, 2006). Generally, after an acute outbreak of a PRRSV infection, herds may undergo a chronic loss of production in growing / finishing pigs and an endemic infection of breeding stock characterized by several outbreaks throughout the year (Stevenson et al., 1993).

The severity of PRRS may result from a number of factors such as differences in virulence among the PRRSV isolates, probable recombination between the different isolates within the same farm, immune status, host susceptibility and concurrent infections (other viruses and bacteria) and hygiene monitoring programme (Goldberg et al., 2000). PRRSV infected pigs are more susceptible to some bacterial (e.g. *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, *Bordatella bronchiseptica*, *Pasteurella multocida*, *Haemophilus parasuis*, *Streptococcus suis*) and viral diseases (e.g. swine influenza virus, Aujeszky's disease virus, porcine respiratory coronavirus, porcine circovirus 2 - PCV2) (Brockmeier et al., 2002). One of the main pathogens involved in the porcine respiratory disease complex (PRDC) is PRRSV, as it has an additive or synergistic effect with the above other bacteria or viruses, that leads to a more severe and chronic respiratory disease in growing / finishing pigs (Thacker, 2006).

Before trying to control diseases at the farm level, it is very important to get information about what we really have to control. For instance, it is important to understand the pathogenesis, epidemiology and clinical forms of diseases. Therefore, the more scientific knowledge is known about PRRS, the better are the chances that this disease will be kept under control at relatively low losses. Table 1 shows some basic information of PRRSV infection at farm level (Cho & Dee 2006; Zimmerman et al., 2006).

Infection source	<ul style="list-style-type: none"> • Direct contact, nasal secretions, saliva, urine, feces, milk, semen, blood, aerosol, transplacental, fomites (e.g. boots, coveralls), equipment, insects, vehicles, human
Incubation time	<ul style="list-style-type: none"> • 3 days and over
Shedding period	<ul style="list-style-type: none"> • 99 days
Survival in the environment	<ul style="list-style-type: none"> • Less than 24 hours at 25°C on solid material • 9 to 11 days in water at 25°C • 8 days in lagoon water at 4°C • Stable at pH 6.5–7.5, but infectivity is rapidly lost at below 6 > pH < 7.5 • Survival >4 months at -70 to -20°C / its viability decreases with increasing temperature • 90% of PRRSV infectivity is lost within 1 week at 4°C, but low titers of infectious virus can still be detected for at least 30 day • In solution, PRRSV infectivity persists for 1–6 days at 20–21°C, 3–24 hours at 37°C, and 6–20 minutes at 56°C • Shedding of PRRSV in saliva, urine, and feces are high risk factors for the environmental contamination
Other properties	<ul style="list-style-type: none"> • High biological, antigenic, genetic and pathogenic heterogeneity exists among PRRSV strains / genetically diverse PRRSV strains may coexist in the same farm • Partial and variable cross protection between isolates • Frequent genetic changes or recombination • Fetuses are most susceptible to active infection after 60 days of conception • Pigs may become carriers until at least 150 days of age (PRRSV persistence) • PRRSV is associated with outbreaks of other pathogens • Lack of complete heterologous protection by commercial vaccines

Table 1. Main information about PRRSV infection for a herd health management programme

The above information should not be used as definitive, as many factors may interact with field situations that can result in potential timeline changes. Veterinary medicine, just as human medicine, is not a static science. So, it is possible that the scientific data for PRRS could change year by year. The data of Table 1 are very basic and representative of the scientific literature regarding PRRSV infection in order to take decisions on what herd health management practices should be applied in a farm. For this reason, the above data should be interpreted, since the goal at farm level is the herd and not individual animals.

2. Principles of a porcine herd health management programme in PRRSV infected farms

2.1 Biosecurity measures

Biosecurity measures are always a fundamental part of a porcine herd health management programme. Generally, the risk of a herd becoming PRRSV-positive increases with: a) density of PRRSV-positive neighboring herds, within 500 meters; b) increasing herd size; c) high number of animal introductions; d) purchase of semen for artificial insemination; e) absence of quarantine for replacement boars and gilts (Weigel et al., 2000). The aim of

porcine herd health management programmes for the prevention and control of PRRSV infection is either to stop the introduction of PRRSV into negative herds or limit the introduction of new strains into already PRRSV-infected herds. However, the control of PRRS in a herd, continuously exposed to exogenous infection, is a loss of time and money. Biosecurity measures should be reviewed with attention to both internal and external biosecurity. External biosecurity deals with the risk of introduction of new PRRSV strains or other co-infections from outside the farm. Internal biosecurity deals with the spread of PRRSV within a farm after the exogenous introduction of the virus. Table 2 shows the basic elements of a biosecurity programme for a commercial farrow-to-finish farm or a sow unit.

Gilts source	<ul style="list-style-type: none"> • PRRSV negative or at least non-shedding • Site of gilt production should be away from any other pig facilities
Semen source	<ul style="list-style-type: none"> • PRRSV negative boar stud • Unit under air filtration or far from any other pig facilities
Quarantine	<ul style="list-style-type: none"> • For a minimum of 30 days before the introduction into the herd • Separate building than the main unit
AIAO	<ul style="list-style-type: none"> • Strictly applied in farrowing rooms, nursery and finisher • Washing/disinfecting/drying between batches • Consider batch farrowing to facilitate AIAO
Environment/Feed, water, air	<ul style="list-style-type: none"> • Mechanical air filtration • Water sanitation • Feed free of mycotoxins / balanced energy and amino acids
Equipment/instruments	<ul style="list-style-type: none"> • One set of processing equipment should be disinfected while the other is in use • Boots, hands and coveralls should be kept clean • Disposable gloves can be used between litters • Separate equipment (shovels, brooms, scrapers) should be used for the manure passage and the feed alley at all times in order to reduce the risk of PRRSV spread • Needles: <i>Sows and Boars: discard after one injection / Piglets: discard after each litter or pen</i> • Equipment / instruments for castration, tooth-clipping or tail-docking: <i>Washing and heating (propane burner) between litters; Disinfection after using</i>
Transport	<ul style="list-style-type: none"> • Transport of pigs in cleaned vehicles (washing, disinfection, drying)
Hygiene	<ul style="list-style-type: none"> • Disinfectants • Washing of boots at the end of the day with a brush and disinfect in a bath of disinfectant (new disinfectant solution should be used every day) • Scrape sow's manure each day in farrowing room with a shovel (1 shovel per room) • Carcass disposal: compost or incineration
Personnel/Visitors	<ul style="list-style-type: none"> • Minimize the numbers of visitors • Wash hands • Fomites belonging to the farm • Forbid to enter equipment, tools or materials in the farm that have been in contact with pigs or pig manure of other farms

Table 2. Basic elements of a biosecurity programme for a commercial farrow-to-finish farm or a sow unit

Direct routes of PRRSV transmission within and between pig populations include infected animals and contaminated semen. For this reason, all replacement boars and gilts should originate from PRRSV negative farms and be properly isolated in quarantine facilities for a minimum of 30 days, including serological tests and PRRSV vaccinations prior to their introduction into the herd. Ideally, isolation facilities should be located on another farm site and visited at the end of the working day. Moreover, semen for artificial insemination should come from PRRSV-negative boar studs. Indirect transmission involves transmission by fomites (boots and coveralls), contaminated equipment (e.g. needles), farm personnel and visitors, transport vehicles (contaminated trailers, coolers, containers), substances (e.g., water, food), insects (e.g. houseflies and mosquitoes), or aerosols (Dee et al., 2002; Cho & Dee, 2006; Desrosiers, 2011). In general, biosecurity efforts should focus on all inputs and outputs of the farms, such as pigs, supplies and materials, feed, water, personnel, removal of manure, and reclaims. In addition, the entry of pests such as rodents, insects, and birds from all buildings should be avoided (Zimmerman et al., 2006). Finally, All-in/All-out (AIAO) pig flow is effective in controlling a variety of respiratory pathogens in weaned pigs. AIAO consists of dividing buildings into individual rooms, allowing thorough cleaning and disinfection of facilities between groups of pigs. This method is very effective in reducing the horizontal spread of PRRSV from older, infected pigs to those recently placed in the finishing stage. Although, AIAO does not directly control the transmission of PRRSV, it reduces the impact of secondary bacterial co-infections.

The disinfection is crucial for a biosecurity programme in a PRRSV-infected farm. The survival of PRRSV outside a living host is affected by factors that include the substrate, pH, temperature, relative humidity, and exposure to ultraviolet light. PRRSV is inactivated by lipid solvents, such as chloroform and ether. In addition, at "room temperature" the virus can be inactivated completely with the use of chlorine (0.03%) in 10 minutes, iodine (0.0075%) in 1 minute, and a quaternary ammonium compound (0.0063%) in 1 minute (Shirai et al., 2000). PRRSV is also relatively labile in the environment and particularly susceptible to heating and drying (Pirtle & Beran, 1996). Leaving a room to completely dry with or without supplemental heating is one of the most effective ways of killing viruses and bacteria. PRRSV can remain infectious for an extended time under specific conditions of temperature, moisture, and pH. It is stable at pH 6.5–7.5, but infectivity is rapidly lost at pH below 6 and above 7.5 (Bloemraad et al., 1994). At temperatures ranging from -70 to -20°C, PRRSV can survive more than 4 months, but when temperature increases, its survivability decreases. Approximately 90% of PRRSV infectivity is lost within 1 week at 4°C, but low titers of infectious virus can still be detected for at least 30 days. In solution, the virus infectivity persists for 1–6 days at 20–21°C, 3–24 hours at 37°C, and 6–20 minutes at 56°C (Zimmerman et al., 2006). At 25–27°C, infectious PRRSV is not detected on plastic, stainless steel, rubber, alfalfa, wood shavings, straw, corn, swine starter feed, or denim cloth (Pirtle & Beran, 1996). Cleaned and disinfected pens should be left to dry for a minimum of 24 hours before pigs are placed, while barns should be allowed to dry for a minimum of 7 - 14 days between batches. Since PRRSV persists in cold and wet conditions, all equipment and material used at the farm or for transport of pigs must be cleaned and dried (Dee et al., 2002).

Herd health management has a significant impact on disease expression, as some management parameters like environmental conditions, water and feed quality may compromise affect the animal resistance to diseases. In addition, the above management parameters if are not in compliance with the rules of animal welfare can also increase the stress and favor virus / bacteria transmission in pigs or have a negative impact on immunity against several diseases.

Water: PRRSV survives in water up to 11 days, but drying quickly inactivates it (Zimmerman et al., 2006). It is important to use chlorine or hydrogen peroxide for the water sanitation, because water sources and delivery systems may be contaminated with pathogens. However, the use of chlorine should be monitored by testing the levels of free chlorine. Well capacity, water flow rates, pressure, drinker number, type and placement are very important for the optimal water intake. Finally, factors such as mineral content, hardness, total dissolved solids, and pH should also be considered.

Feed: it is unlikely that PRRSV can be transmitted through contaminated feed, but feed suppliers present a risk acting as a vector from infected farms to other farms. However, energy and amino acids must be balanced in diet, in order to provide the required energy for the enhancement of the immune system and the basic building blocks of antibodies. Minerals and vitamins are also required in sufficient quantities for the optimal function of immune system. According to the rules of animal welfare, feeders must be managed appropriately, so that the feeder provides adequate access for each pig. Moreover, the feed needs to be free of mould and mildew, because mycotoxins can cause immune suppression, resulting in increased incidences of clinical signs of PRRS, such as pneumonia.

Environment: It plays also a main role, as PRRSV shedding in saliva, urine, and feces is a high risk factor for the environmental contamination, creating the potential for transmission via fomites (e.g. boots, coveralls), personnel and vehicles. Air exchange rates have an impact on control of PRRS. The filtering of air entering pig housing has been proposed as a means to reduce the risk of airborne transmission of PRRSV from infected herds to at-risk populations (Pitkin et al., 2009). Therefore, air filtration is an effective means to reduce the risk of external PRRSV introduction to large breeding herds located in areas with high pig population density (Dee et al., 2010). Moreover, increased relative humidity may increase the survival time of respiratory pathogens in the room environment and increased ventilation rates may increase air speed, causing chilling. Chilling, due to the wide daily and rapid small temperature fluctuations, contributes significantly to the increased prevalence of disease by increasing stress levels in affected pigs. For this reason, ventilation and temperature controllers should be adjusted so as to ensure that they are set to control temperature fluctuation and daily variability. The use of simple environmental testing equipment, such as humidity monitors, data loggers, air speed and gas testers are very important. Moreover, PRRSV can be inactivated through the process of composting or incinerating carcasses, so only these methods should be applied. Finally, PRRSV can survive in lagoon effluent for up to 3 days at 20 °C and for 7 days at 4 °C. Contact with PRRSV-positive effluent can be a source of infection to naïve pigs. Therefore, producers that utilize recycled lagoon water in their waste management protocols may be at higher risk for external PRRSV introduction than those who use deep pits.

2.3 Control strategies

PRRSV tends to circulate within a herd indefinitely after the initial infection of a herd. For example, PRRSV was isolated from nursery pigs up to 3.5 years after the initial PRRS outbreak (Larochelle et al., 2003). Several parameters related to the herd (size, management and type), or the vaccine (type, management) may determine whether PRRS can be successfully controlled in a farm. Unfortunately, the ability of the virus to persist in herds and its wide biologic, antigenic and genetic variability may further complicate control plans (Zimmerman et al., 2006). In endemically PRRSV infected herds, the virus circulates, because, in any given time, animals are in different stages of infection and immunity. Control and prevention of the virus should be based on: a) minimizing PRRSV circulation at farm level, b) limiting the effects of the virus circulation and secondary bacterial co-infections and c) maximizing and stabilizing the herd immunity (Dee, 2003).

2.3.1 Vaccination programmes

Vaccinations against PRRSV with both modified (or attenuated) live vaccines (MLV) and inactivated (or killed) vaccines have frequently been reported by many studies. The absence of complete protection has frequently been attributed to antigenic differences and the limited cross-reactivity between strains of commercial vaccines and challenge strains. Such variability is largely attributed to genetical and antigenic heterogeneity between isolates mainly among European and American isolates, but also within the same area or the same farm. The high heterogeneity among PRRSV strains is likely to be the main obstacle to effective control of PRRSV infection using current commercial vaccines (MLV and inactivated), since the immunity induced by one strain may be only partial against a different strain, even within the same genotype (Mateu & Diaz, 2008; Kimman et al., 2009). However, vaccine efficacy may be associated with an efficient cell-mediated immunity and it is not only related with its immunological properties, but also with the characteristics of the challenging strain to trigger an immune response (Martelli et al., 2009). Therefore, the ability of each strain to induce a strong cell-mediated immune response is more important than the genetic similarity between the vaccine strain and the field strains for inducing clinical protection (Mateu & Diaz, 2008). The complexity of the immune response to PRRSV and the ability of the virus to escape or modulate the host's immune system make difficult the development of an effective vaccine for control and eradication of PRRS.

MLV vaccines have been widely used in breeding stock and young piglets. The results of many studies have shown beneficial effects on PRRS clinical disease occurrence and severity, the duration of viremia and virus shedding (Scortti et al., 2006; Martelli et al., 2007; Kimman et al., 2009), as well as improvement of health status and performance of gilts/sows and their litters (Alexopoulos et al., 2005). However, the use of MLV vaccines is questionable. Virus-neutralizing (VN) antibodies against PRRSV protect against viremia, virus replication in lungs, transplacental spreading of the virus and reproductive failure (Labarque et al., 2003, 2004; Lopez & Osorio, 2007). MLV vaccination induce VN antibodies and protect against viremia, virus replication in lungs and virus induced respiratory and reproductive disorders (Labarque et al., 2003; Scortti et al., 2007; Zuckermann et al., 2007). The protective immune response induced by current commercial MLV vaccines is influenced by genetic diversity, as these vaccines do not always sufficiently protect (or only

partially) against re-infection and transplacental infections caused by heterologous strains (Scortti et al., 2006; Prieto et al., 2008; Kimman et al., 2009). However, Martelli et al. (2009) reported that vaccination of piglets at 5 weeks of age with a commercial MLV vaccine induced a partial clinical protection, associated with an efficient cell-mediated immune response, when the above vaccinated pigs were exposed to a heterologous field strain. Moreover, there are major concerns about the safety of current vaccination programmes with MLV vaccines. Experimental and field studies reported that MLV strains can cause viremia, revert to virulence and spread transplacentally and horizontally not only within the vaccinated herds (transmission and detection in non-vaccinated pigs), but also to neighbouring non-vaccinated herds (Bøtner et al., 1997; grosse Beilage et al., 2009; Kimman et al., 2009). It is possible that farmers using an MLV vaccine for the first time may experience a decrease in the herd productivity. MLV vaccinations in sows reported to cause acute PRRS-like clinical signs, characterized by increased late term abortions, increased numbers of stillborns and mummified piglets, as well as reduced numbers of live born and weaned piglets (Bøtner et al., 1997; Dewey et al., 1999). An additional problem is that the MLV vaccination has been shown to decrease the efficacy of *Mycoplasma hyopneumoniae* vaccines (Thacker et al., 2000; Le Roitha et al., 2011).

Based on the Greek experience, in farms with history of endemic PRRSV infection, the MLV vaccination of breeding stock (gilts once at the age of 180 days and sows 10 days post-partum) can have beneficial effects on their health and performance. Alexopoulos et al. (2005) reported reduction of premature farrowing rate, return-to-oestrus rate, number of dead and mummified born piglets as well as the increase of farrowing rate, the number of piglets born alive and weaning pigs per litter. Furthermore, in farms affected by both PRRSV and PCV2, the MPV vaccinations in piglets (at roughly 5 weeks old) can lead to a reduction of morbidity and mortality of growing pigs, as well as an improvement of the growth performance in vaccinated pigs (Alexopoulos et al., 2005; Kritas et al., 2007). However, recent evidences based on personal experience and field observations in endemic PRRSV-infected farms suffering by significant reproductive failure, indicate that the MLV vaccination of breeding stock can improve; a) the reproductive performance, b) the viraemic status of piglets, c) the morbidity and mortality of piglets, d) the growth performance of piglets. The vaccination protocol depends on the average breeding herd size and the current clinical expression of PRRS. In cases of; a) large scale farms with a capacity of over 700 sows under production, b) farms suffering from acute outbreaks of PRRS and c) endemic PRRSV-infected farms with 4-5 outbreaks every year, the preferable vaccination schedule includes a vaccination at 60th day of gestation and booster at 6th day of lactation. Vaccination at 2-4 weeks before mating and revaccination before each consecutive gestation is proposed for; a) farms with a capacity of over 100-200 sows under production and b) endemic PRRSV-infected farms with 4-5 outbreaks every year. Moreover, the MLV vaccination of piglets at 2-3 weeks of age can have beneficial effects on their health and growth performance, including reduction of morbidity and mortality rate due to PRRSV infection, as well as improvement of average daily gain and feed conversion ratio (Papatsiros, 2011).

Inactivated vaccines are considered safer than MLV vaccines, as the vaccine virus cannot transmit to other pigs and cannot revert to virulence. However, their efficacy has been seriously questioned. Field studies reported that the inactivated vaccines did not induce

reproductive failure in vaccinated sows and improved efficiently the reproductive parameters at a farm level (Plana-Duran et al., 1997; Papatsiros et al., 2006). However, their capacity to induce a protective immunity against challenge with wild-type virus has been questioned, as these vaccines induce poor immune responses in naïve pigs and provide weak memory responses with sequential challenge without any obvious active immune responses in the vaccinated pigs (Kim et al., 2011). On the one hand, commercially available inactivated vaccines do not induce VN antibodies and do not sufficiently protect against viremia or prevent from the clinical signs associated with PRRSV infection, i.e., post-challenge viremia and transplacental infection of the piglet (Nilubol et al. 2004; Scotti et al., 2007; Zuckermann et al., 2007). On the other hand, experimental inactivated PRRSV vaccines can induce VN antibodies and reduce the duration of viremia (Misinzo et al., 2006, Vanhee et al., 2009). However, without VN antibodies induction, a commercial inactivated vaccine reported to induce significant improvement of sow reproductive performance and litter characteristics (Papatsiros et al., 2006). In addition, some types of adjuvants were used as effective vaccine adjuvants to enhance the humoral and cellular responses of piglets against PRRSV (Linghua et al., 2007). Hence, the effectiveness of vaccination programme based on inactivated vaccine depends on the vaccine (virus strain and used cells to prepare the vaccine) and vaccination strategy (Misinzo et al., 2006).

Based on the Greek experience, the vaccination of gilts and sows with inactivated vaccines can have beneficial effects on their health and performance, as well as on their litters. The long term use of a commercial inactivated vaccine for a period of 1.5 years in breeding stock of a closed single commercial farm with persistent PRRSV infection and high seroprevalence, proved to reduce the negative effects of the virus on the breeding herd (Papatsiros et al., 2006). The vaccination scheme included a primary vaccination of all gilts/sows of the herd by administering two doses 3–4 weeks apart, except those being 1 week prior to 2 weeks post-service. The skipped females were subjected to primary vaccination, but starting 3 weeks later. All previously vaccinated animals received a booster vaccination between 55 and 60 days of next gestation, and thereafter at each gestation for a period of 1.5 years. The gilts were vaccinated twice prior to breeding (primary vaccination) and boosted in each pregnancy as described previously. Vaccinations resulted in a significant improvement of sow reproductive performance and their litters' characteristics, as are shown in Table 3. This study indicated also that under practical conditions, the use of an inactivated vaccine should be administered on a regular basis for obtaining the maximum of its effect, as it has been observed that the higher the degree of immunization of sows, the better the improvement of their reproductive parameters. It is interesting to note that while the number of booster vaccinations improved several performance parameters, it did not improve the level of immunity as measured by IPMA, suggesting that the levels of IPMA-antibodies may not always reflect protection. Moreover, the vaccination led to a significant reduction of culling rate due to reproductive failure, resulting in an improvement of longevity, herd age distribution and number of non productive days in the breeding stock (Table 4). Finally, the long-term vaccination of boars with the same inactivated vaccine was safe and no significant changes in semen characteristics after each vaccination were noticed (Papatsiros et al., 2008). The above vaccination schedule is proposed to be applied on a regular basis in breeding stock of endemic PRRSV-infected farms, in order to achieve a stabilization of the immunity status of all breeding herd and prevent the losses due to the yearly outbreaks of PRRS.

	Period relative the start of vaccination			
	1 year prior (Non vaccinated sows)	1.5 years after (Vaccinated sows)	1 year prior (Non vaccinated sows)	1.5 years after (Vaccinated sows)
Reproductive parameters	Number of piglets per litter			
Returns to oestrus rate (%)	8.9 ^a	6.8 ^b	Totally born	11.4 ^a ± 3.1 11.6 ^a ± 3.1
Abortion rate (%)	2.2% ^a	1.3% ^b	Live born	10.5 ^b ± 3.1 10.9 ^a ± 3.1
Farrowing rate (%)	87.7% ^b	90.3% ^a	Stillborn	0.7 ^a ± 1.4 0.5 ^b ± 1.2
«Empty» sows rate (%)	0.4% ^a	0.5% ^a	Mummified	0.2 ^a ± 0.6 0.1 ^b ± 0.5
Premature farrowing rate (%)	22.8% ^a	1.4% ^b	Light weight	1.0 ^a ± 1.6 0.1 ^b ± 0.6
Farrowing rate (%)	87.7% ^b	90.3% ^a	Weak	1.5 ^a ± 2.0 0.6 ^b ± 1.5
Culling rate (%)	24.5% ^a	21.9% ^b	Splay-legs	0.7 ^a ± 1.0 0.1 ^b ± 0.4
Gestation length (days)	114.3 ^b ± 2.3	115.2 ^a ± 1.4	Alive first 24h	10.1 ^b ± 3.0 10.5 ^a ± 3.0
Lactation length (days)	25.0 ^a ± 3.3	21.6 ^b ± 2.8	Weaned	8.9 ^b ± 1.5 9.3 ^a ± 1.4
Weaning-to-service (days)	6.7 ^a ± 6.3	5.6 ^b ± 3.7		

^{a,b}. Percentages and Means (± SD) in a row with different superscripts differ (P≥0,05).

Table 3. Reproductive and litter characteristics prior (non vaccinated sows) and after (vaccinated sows) the start of vaccination with inactivated vaccine: Source: Papatsiros 2006.

Causes of culling (%)	Period relative the start of vaccination	
	1 year prior (Non vaccinated sows)	1.5 years after (Vaccinated sows)
Reproductive failure	40.5% ^a	26.6% ^b
Deaths	13.7% ^a	10.4% ^a
Old age	14.8% ^a	33.9% ^b
Locomotor problems	12.5% ^a	12.7% ^a
Other	18.5% ^a	16.3% ^a

^{a, b} Percentages in a row with different superscripts differ (P≤0.05).

Table 4. Causes of culling in female breeding stock prior (non vaccinated animals) and after (vaccinated animals) the start of vaccination with inactivated vaccine. Source: Papatsiros 2006.

2.3.2 Gilt development & isolation / acclimatization

The first step in breaking virus circulation in the breeding herd is to use replacement animals that have been exposed to PRRSV and developed immunity prior to their introduction into the herd (Dee, 2003). Gilt introduction in breeding population is the key for PRRS control, consisting of 3 periods: the isolation period, the acclimatization period, and the recovery period (Pesente et al., 2006; Vashisht et al., 2008). The length of each period may range from 30 to 60 days, depending on the age of the replacement animal, the PRRS history of intra and inter farms, and the type and size of farm. The isolation period consists of serological tests (day 1 to 2) in order to determine the PRRSV infection status of the incoming animals. The PRRSV vaccination of the incoming breeding stock should be done shortly after arrival. The acclimatization period starts 30 days after the introduction of replacement gilts, aiming to expose the "new" gilts to the specific PRRSV strain of the farm. Finally, a period of recovery (at least 30 days) is implemented to reduce the risk of introducing actively infected gilts into the breeding herd. By the end of acclimatization period, the replacement gilts could be vaccinated against other diseases, such as Aujeszky's disease. Moreover, the acclimatization should be also applied in farms with grandparent nucleus of sows for producing its own gilts, which are separately housed (McCaw, 1995).

Several acclimatization programmes were described, including methods such as: a) feedback with tissues from weak-born piglets and stillbirths in the face of outbreaks, b) use of MLV and inactivated vaccines, c) contact exposure between gilts and weaning pigs that are used as donor sources of virus and d) inoculation of negative replacement animals with serum collected from viremic pigs from the same farm (Dee, 2003; Vashisht et al., 2008). The use of serum containing viable field virus has recently increased due to the increased genetic heterogeneity among PRRSV strains and the perception that commercial vaccines do not induce enough protective cross-protection against newly identified PRRSV strains. This method has inherent risks and requires thoughtful application and high quality control standards (Zimmerman et al., 2006). Consistent acclimatization of incoming breeding stock to PRRSV results in: a) the stabilization of clinical signs, b) the production of PRRSV negative piglets at weaning, c) the prevention of PRRSV outbreaks associated with endemic PRRSV strains, d) the development of specific immunity to the homologous herd strains, and e) the improvement of production parameters (Pesente et al., 2006; Vashisht et al., 2008). It is generally accepted that early exposure (2–4 months of age) can result in the protection of the exposed animals and the introduction of the replacement animals at a time when shedding has stopped. Therefore, it is recommended that gilts can be purchased and acclimatized at an early age (Vashisht et al., 2008).

In a closed-herd system, replacements are produced internally and are introduced into the sow herd directly from the grower or finisher stage, regardless their PRRSV infection status. Generally, closed herd systems do not eliminate PRRSV infection, because replacement gilts usually have previous exposure to PRRSV strains circulating in the herd. For this reason, gilt developer facilities are very important for introduction of gilts into an infected farm. Gilt developer facilities should be located in the sow site or preferably, in an alternative location far from the other unites. Gilts may be introduced at the age of weaning piglets or 2 to 5 months of age, under AIAO pig flow practices.

A field study in a commercial farrow-to-finish farm with 1,100 sows, where the gilts were housed with finishing pigs, indicated that even if the breeding stock were vaccinated with a PRRSV inactivated vaccine for a period of 24 months, the non-vaccinated gilts show high (93,3 - 100%) seroprevalence (Figure 1). This was an indication of high virus circulation in fattening buildings, but also was important evidence that some infected gilts re-introduced PRRSV into the breeding stock (Papatsiros, 2006).

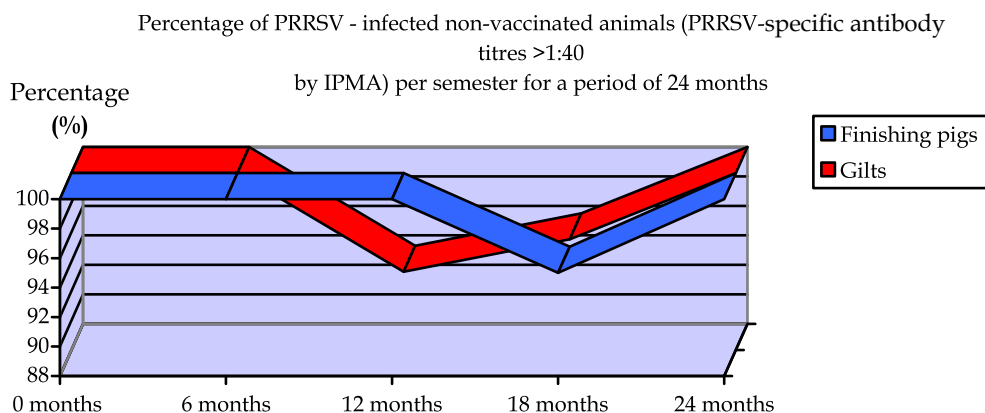


Fig. 1.

Finally, the strategy of “herd closure” could also be applied in order to eliminate the virus circulation by the reduction of viral shedding and elimination of carrier pigs. The basis of herd closure is the cessation of replacement gilt introduction for an extended period (4 to 8 months), depending on the herd health status and pig flow. During the period of herd closure, the introduction of replacement gilts and boars is discontinued, and only the introduction of semen for artificial insemination is allowed. In addition, the vaccination of the breeding herd could be applied at this period. However, herd closure can result in the production of an improper parity distribution and the development of a PRRSV-negative breeding population over time. These effects can be minimized through the use of off-site breeding projects for replacement gilts (Cho & Dee, 2006; Zimmerman et al., 2006).

2.3.3 Other management practices

McCaw (1995) proposed the McREBEL (Management Changes to Reduce Exposure to Bacteria to Eliminate Losses) system for PRRS control. The aim of McREBEL is to maximize the number of piglets remaining with their birth mother and, secondly, to maximize the number of piglets remaining with the colostrum mother. According to the author, the McREBEL system reduces considerably the economic losses, without eliminate successfully the virus circulation in the farm. This system is recommended to be applied in farms with separate premises for the different ages both at farrowing and in newly weaned piglets, using the AIAO system as following:

- i. Limitation or interruption of the cross-fostering of newborn piglets for resizing or saving sick pigs, fall-behinds, and runts. The cross-fostering of piglets should be performed only within the first 24 hours of age.
- ii. Piglets should be moved within farrowing rooms at birth and not between different rooms to "nurse sows" (especially poor-doing or possibly diseased piglets to younger age groups attempting to save them). In addition, sows or piglets should not be moved between rooms.
- iii. Interruption of using "nurse sows" for weak-born PRRSV-infected pigs, fall-behinds, and runts.
- iv. Minimization of piglets' handling, especially routine antibiotic treatments. The effect of each handling or treatment procedure should be evaluated on clinical disease levels.
- v. Immediate euthanization of all very sick and debilitated piglets those are non-responsive to therapy.
- vi. Sick or lightweight piglets should not be retained with or mixed with younger piglets. Euthanization of small piglets with poor body condition at weaning.
- vii. Suckling and weaning piglets should only be moved AIAO, by room. 2-3 days should be allowed between batches for cleaning and disinfection and drying.
- viii. Weaning piglets should be loaded at a time with earlier weaning of a few of the oldest and biggest litters.

Co-infections of PRRS by several other respiratory pathogens have the ability to increase both the severity and duration of PRRS associated disease. For this reason, a porcine herd health management for the prevention and control of PRRSV infection should also include measures for the control of bacterial co-infections (e.g. *Haemophilus parasuis*, *Streptococcus suis*, *Actinobacillus pleuropneumoniae*, *Pastereula multocida*) and viral diseases such as swine influenza, Aujeszky's disease and PCV2 associated diseases. Appropriate medication and vaccination protocols need to be applied for the individual infections. Water soluble antimicrobials or feed medications can be selected based on the sensitivity pattern of the secondary bacteria. Oxytetracycline, chorotetracycline, trimethoprim/sulpha, or synthetic penicillins are the medicines of choice, but tiamulin, tylosin or lincomycin may be also used. If *Actinobacillus pleuropneumoniae* is active, ceftiofur or florfenicol could be medicines of choice for individual treatments. Feed medications (e.g. 500 to 800g of tetracycline or 400g of trimethoprim/sulpha per tonne in-feed) must be provided at the earliest possible post infection time in order to maximise efficacy. Anti-fever drugs may be used to decrease the negative effects of fever, such as it depresses appetite, abortion in sows, and decreases milk production. Finally, vaccination protocols should be applied, including vaccines against viral pathogens such as PCV2, swine influenza virus and Aujeszky's disease virus, as well as bacterial pathogens such as *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae* and *Haemophilus parasuis*. The most important vaccinations of weaning pigs in farms suffering from PRDC are considered those against *Mycoplasma hyopneumoniae* and PCV2 (Alexopoulos et al., 2004).

3. Monitoring serology

The preliminary diagnosis of PRRSV infection is most often based on a review of herd history and clinical signs. Gross post mortem findings can help to confirm the suspicions

and finally serological, histological, immunohistochemistry and PCR examinations can confirm the diagnosis. Because of the PRRSV genetic diversity, it is preferable to isolate and characterize the farm specific PRRSV strains, using genetic typing such as restriction fragment length polymorphism (RFLP) and gene sequencing. This allows also determining any new PRRSV strain (Zimmerman et al., 2006).

Before starting a vaccination programme, it is essential to investigate when the PRRSV infection took place. Routine serological monitoring tests for PRRSV serum antibodies are very useful diagnostic tools for the determination of PRRSV herd exposure status. Therefore, the vaccination programme can be applied prior to the usual infection time in order to provide efficient protection in vaccinated animals. The serological diagnostics of PRRS include enzyme-linked immunosorbent assay (ELISA), blocking ELISA, serum virus neutralization (SVN), indirect fluorescent antibody (IFA) and immunoperoxidase monolayer assay (IPMA). The IFA has high specificity (99.5%), but unknown sensitivity for individual animals, while the IPMA is also considered to be a highly specific and sensitive test (Wensvoort et al., 1991). The ELISA is also reported to be sensitive and specific (O'Connor et al., 2002). The specificity of a commercial PRRS ELISA (HerdChek® PRRS ELISA, IDEXX Laboratories Inc., Westbrook, Maine), has been estimated to be between 99.3 and 99.5 % (O'Connor et al., 2002). The ELISA is well-suited for monitoring since it is sensitive, specific, standardized, simple to perform, relatively inexpensive, easy to implement, able to detect both American and European strains, and potential for rapid analysis of numerous serum samples. Antibodies can be detected as early as 9 DPI (days post infection), peak at 30-50 DPI and then decline to negative levels 4-12 months after infection. ELISA results are interpreted as positive when a sample-to - positive (S/P) ratio ≥ 0.4 (indicates presence of antibody to PRRSV) or negative (S/P < 0.4). The demonstration of seroconversion (negative to positive), using acute and convalescent serum samples, is the most definitive method to diagnose PRRSV infection serologically. Increasing titers of PRRSV specific antibody demonstrated by rising ELISA S/P ratios in a group of infected animals can also indicate PRRSV infection. Finally, sequencing of PRRSV isolates followed by phylogenetic analysis is a powerful tool to monitor the spread of virus intra and inter farms and allows a fuller understanding of the success or failure of the control programme (Pesente et al., 2006). The ELISA in all of its current forms is the best practical diagnostic method to detect exposure events and confirm vaccinations, but it is not a reliable indicator of protection against PRRSV.

The number of serum blood samples needed to identify an infected herd depends on the seroprevalence. Shortly after an acute outbreak, the prevalence of seropositive pigs in infected herds is high. For this reason, it is possible to identify infected herds by testing only a small number of samples. In case of a herd with unknown serostatus, more samples from breeding herd than from a finishing herd should be tested, because seroprevalence in breeding herd is usually lower compared to seroprevalence in finishing herd. A porcine herd health management programme for the control of PRRS should include a herd serological monitoring, once or twice per year. The herd serostatus is predicted by testing a representative number of animals. The number and ages of animals to be tested is determined by the assumed seroprevalence in each age group. Seroprevalence to PRRSV in the breeding herd can vary widely depending on how long the herd has been infected.

Herds with a recent history of reproductive failure have often been infected less than 1 year and tend to have a high seroprevalence (approximately 50%) in breeding animals. To detect seropositive animals with 95% confidence (assume 50% seroprevalence in the breeding herd) at least 10-20 blood samples from breeding stock is recommended. In endemic PRRSV-infected herds, which have probably been infected more than 1 year, the seroprevalence is usually low in the breeding herd (20% or less) and high in the finishing pigs (50% or greater) (Stevenson et al., 1993). For example, in a breeding herd of 100 animals, to detect seropositive animals with 95% confidence (assume 10% seroprevalence in breeders and 50% seroprevalence in finishing pigs) at least 25 blood samples from breeding stock and 7 from finishing pigs are recommended. However, herd serology only demonstrates previous exposure to the virus and does not provide a definitive diagnosis of PRRSV as a cause for the clinical problems on the farm. It is generally recommended that young pigs (10-15 blood samples from pigs at 3, 5, 7, 9 weeks and 16 of age), rather than breeding stock, should be tested to determine PRRSV circulation and infection status in a herd. In single-site, farrow-to-finish herds, the seroprevalence of PRRSV infection is usually considered to be highest in the grow-finish unit. Moreover, in herds without clinical signs of PRRS, testing 12 blood samples in both PCR and ELISA, (6 samples in pigs 9 weeks of age and 6 samples in pigs 16 weeks of age), is reported as a cost-efficient first evaluation of the PRRSV infection-status (Duinhof et al., 2011). In order to detect an increasing seroprevalence over time is preferable to perform serology in nursery pigs, including ear tagging and bleeding 10-20 pigs at 3-4 weeks of age and bleeding the same pigs at 7-8 weeks of age (Van Alstine et al., 1993). A greater percentage of seropositive pigs at the second bleeding would indicate that the virus is actively spreading among pigs on the farm. This may be confounded by passively acquired maternal antibody that usually lasts for 6-8 weeks, but can last up to 16 weeks of age.

4. Conclusion

PRRS remains to have a great negative economic impact on the the global swine industry. The control of PRRS economic losses in commercial swine farms is a challenge for farmers and swine veterinarians. Many factors including management practices and pig flow, the level of risk associated with local pig density, and the inherent characteristics of the specific PRRSV strains on farm level contribute to a successful a porcine herd health management programme development. The goal of a porcine herd health management to control PRRSV infection is based mainly on: a) minimizing the virus transmission and circulation in the farm, b) maximizing the herd immunity against PRRSV and c) limiting the losses due to secondary co-infections. A good knowledge of PRRS disease at the farm level, the establishment of strict biosecurity measures, acclimatization of replacement gilts by exposure to the specific PRRSV circulating strains in farm as well as the application of appropriate vaccination programme are commonly considered the most sound strategies to control PRRS. Furthermore, a completed herd history, assessment of clinical signs and proper diagnostic tests are very important tools for a swine veterinarian practitioner to control successfully PRRSV infections. Among the diagnostic tests, ELISA is the best practical diagnostic method to detect exposure events and confirm vaccinations, but it is not a reliable indicator of protection against PRRSV.

Finally, the establishment of a porcine herd health management programme to control PRRS is an excellent investment for swine farmers in order to improve the herd health status and reduce the cost of pork meat production.

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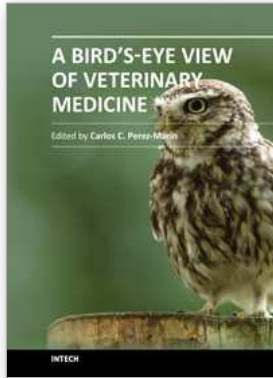
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Veterinary medicine is advancing at a very rapid pace, particularly given the breadth of the discipline. This book examines new developments covering a wide range of issues from health and welfare in livestock, pets, and wild animals to public health supervision and biomedical research. As well as containing reviews offering fresh insight into specific issues, this book includes a selection of scientific articles which help to chart the advance of this science. The book is divided into several sections. The opening chapters cover the veterinary profession and veterinary science in general, while later chapters look at specific aspects of applied veterinary medicine in pets and in livestock. Finally, research papers are grouped by specialisms with a view to exploring progress in areas such as organ transplantation, therapeutic use of natural substances, and the use of new diagnostic techniques for disease control. This book was produced during World Veterinary Year 2011, which marked the 250th anniversary of the veterinary profession. It provides a fittingly concise and enjoyable overview of the whole science of veterinary medicine.

How to reference

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