Due to the similarity of clinical signs of PRRS virus infection and those caused by other viral and bacterial pathogens, laboratory tests are required to definitively diagnose PRRS.

Several kinds of serologic tests are available include enzyme-linked immunosorbent assay (ELISA), serum virus neutralization (SVN), and indirect fluorescent antibody (IFA). These tests only indicate that a pig has been exposed to the virus naturally or through vaccination but cannot tell if the pig remains infected.

Additional tests are available to actually determine the presence of the virus. These tests include immunohistochemistry staining (IHC), fluorescent antibody staining (FA), polymerase chain reaction (PCR), and virus isolation (VI). While a positive result on these tests indicates the virus is present in the sample, a negative test does not necessarily indicate that the pig is free of the virus. Correct sample selection, sample handling, and the sensitivity of the test all interact to provide a reliable result.

No serologic test can differentiate whether a pig has been infected with a field strain of PRRS virus or has only been vaccinated.

Genomic sequencing of the virus can predict with some accuracy the relatedness between two strains of PRRS virus as well as how closely related they are to vaccine strains. Genomic sequencing will not predict the likelihood of a vaccine to successfully prevent the disease.

Restriction fragment length polymorphism (RFLP or “cut pattern”) has limited value as a PRRS virus diagnostic test.

Pigs born to seropositive dams will remain positive on serologic tests until 3 to 16 weeks of age. The exact timing will vary according to the level of positivity in the dam and what serologic test is being used.

Since antibodies do not persist for the lifetime of an animal, it is generally recommended that young pigs, rather than breeding stock, be tested to determine a herd’s PRRS virus infection status.
Diagnosis of PRRS Virus
K-J Yoon, J Christopher-Hennings, EA Nelson

Introduction

A tentative diagnosis of PRRS virus infection is suggested by clinical signs; reproductive problems in breeding stock or respiratory disease in pigs of any age. Reproductive problems associated with PRRS include poor conception rates, late-term abortions, and an increase in the rate of stillborn pigs, mummified fetuses, and weak, non-viable piglets. Infection with PRRS virus often does not induce unique gross or microscopic lesions, although interstitial pneumonia is a common finding in the respiratory form. In addition, the gross lesions caused by PRRS virus infection may resemble or be obscured by lesions caused by other infectious agents.

Due to the similarity of clinical signs of PRRS virus infection to those caused by other viral and bacterial pathogens and the lack of PRRS virus-specific lesions, differential tests are required for a definitive diagnosis. The differential diagnosis includes infection with porcine parvovirus, pseudorabies virus, hemagglutinating encephalomyelitis virus, porcine circovirus type 2, porcine enterovirus, swine influenza virus, classical swine fever (hog cholera) virus, porcine cytomegalovirus, and leptospirosis (Allan and Ellis, 2000; Keffaber, 1989; Halbur et al., 1993, 1995a; Mengeling et al., 1993; Paton et al., 1992b; Yoon et al., 1996a). Therefore, when the clinical history and pathology is suggestive of PRRS, detection of viral antigens, viral genomic material, or isolation of virus from clinical specimens must be used to confirm the tentative diagnosis. Alternatively, documentation of rising serum antibodies in a group of affected animals can also be used for diagnosis of PRRS virus infection. One must exercise caution when interpreting serology in herds already known to be infected or vaccinated. This is due to the fact that none of the available serologic tests can differentiate positive results due to infection from positive results due to vaccination. Additionally, serology can not be relied upon to determine how long a pig has been infected. Serologic testing coupled with an

General Guidelines for PRRS Diagnosis

Diagnosis is based on obtaining evidence of PRRS virus infection in animals suspected of harboring the infection. Such evidence may be obtained by isolating PRRS virus or detecting PRRS viral antigens or nucleic acid in the animals. In clinically affected animals, demonstration of suggestive pathological lesions supports the diagnosis of PRRS in conjunction with laboratory testing. It should be kept in mind that PRRS virus or PRRS viral RNA can be detected in clinically normal animals vaccinated for PRRS or animals persistently infected. Timing after vaccination or history of previous exposure should be taken into consideration when conducting a diagnostic investigation for PRRS.

Successful isolation and/or detection of viruses in clinical materials are highly dependent on proper collection and handling of specimens. In general, specimens intended for virus assays should be collected as early as possible in the course of the disease, i.e., within the first 7 to 10 days after the onset of illness. Samples collected during the acute phase of viral infection usually contain adequate amounts of virus for detection in available assays. Samples collected later in the course of infection usually require more laboratory time and often yield poor or negative results. It is important to choose not only the most appropriate specimen, but also to collect an adequate amount of specimen for virological testing. Insufficient amounts of sample are a potential cause of inconclusive diagnosis or false-negative result.

For best results in isolation and detection of viruses, clinical specimens should be aseptically collected, kept fresh, and transported immediately to the laboratory. If delays are unavoidable or any detrimental affects on virus in samples are anticipated during transport, samples should be refrigerated at 40° F (4° C) for no more than 2 days. For longer storage periods, freeze samples at minus (−)70° C, but NEVER at minus (−)20° C. Self-defrosting freezers in conventional refrigerators are not appropriate for storage. Ideally, frozen samples should be submitted on dry ice, but commercial refrigerant packs can be used if necessary.

Demonstration of seroconversion or increasing titers of PRRS virus-specific antibody in a group of affected animals can also be used for diagnosis of PRRS virus infection. One must exercise caution when interpreting serology in herds already known to be infected or vaccinated. This is due to the fact that none of the available serologic tests can differentiate positive results due to infection from positive results due to vaccination. Additionally, serology can not be relied upon to determine how long a pig has been infected. Serologic testing coupled with an
epidemiological approach, such as case-control or longitudinal studies, strengthens the use of serology for PRRS diagnosis. When herd monitoring is desired, utilizing other tests such as polymerase chain reaction (PCR) or virus isolation (VI) in combination with serology will give the producer and veterinarian a clearer picture of virus transmission patterns.

Assays for Detection of PRRS Virus

Isolation of live virus
PRRS virus is known to replicate in only two types of cells: porcine alveolar macrophages (PAMs) and certain African monkey kidney cell lines (Bautista et al., 1993b; Dea et al., 1992; Paton et al., 1992a, 1992b; Wensvoort et al., 1991; Yoon et al., 1992b; Zeman et al., 1993). However, all PRRS virus isolates do not replicate in both cell types. This suggests that at least two cell types should be used for virus isolation whenever possible. Use of PAMs may be required when attempts to isolate European-like PRRS virus are made (Dewey et al 2000; Wensvoort et al., 1991; M. Murtaugh personal communication).

PRRS virus has been isolated from a variety of clinical specimens (Done et al., 1996; Goyal 1993; Paton et al., 1992b; Rossow, 1998), including serum, plasma, peripheral blood mononuclear cells (i.e., buffy coat layer), bone marrow, tonsil, lungs, lymph nodes, thymus, spleen, heart, brain, liver, testis, epididymus, ductus deferens, bulbourethral gland, penile tissue, oropharyngeal scraping, nasal turbinante, nasal swabs, placenta, saliva, urine, feces, and semen (Baron et al., 1992; Benfield et al., 1994a, 1994b; Christianson et al., 1992, 1993; Christopher-Hennings et al., 1995b, 1998, 2001; Dea et al., 1992; Done et al., 1992; Goyal and Collins, 1992; Horter et al., 2002; Joo, 1993; Keffaber et al., 1992; Mendez-Trigo, 1993; Ohlinger et al., 1992; Paton et al., 1992a, 1992b; Rossow et al., 1994, 1999; Rowland et al., 1996; Swenson et al., 1994; Thacker, 1992; Van Alstine et al., 1993a, 1993b; Wills et al., 1997a, 1997b; Yoon et al., 1993). Among all samples described above, fluid collected from deep with the lungs (bronchoalveolar fluid) and serum are considered by some as the preferred specimens for virus isolation when an acute PRRS outbreak occurs, regardless of the age of the affected animals. PRRS virus is more stable in serum than in tissue (Mengeling et al., 1995; Rossow, 1998; Van Alstine et al., 1993a). In older animals, viremia is of short duration and PRRS virus may be found in tissues longer than in serum (Christopher-Hennings et. al., 2001). If tissues are submitted, they should include lung, tonsil, and lymph nodes (Joo, 1993). Tissue must be fresh if virus isolation is to be successful, regardless of which tissues are submitted for diagnostic investigation.

Sample selection may also depend on the stage of infection (i.e., acute, convalescent, or persistent). Serum, lung, and bronchoalveolar lavage fluid are samples of choice for isolation of PRRS virus in acutely infected animals (Joo, 1993; Mengeling et al., 1995, 1996a, 1996b). For virus isolation from persistently infected animals, tonsil, oropharyngeal scraping, and bronchoalveolar lavage fluid are better samples than serum and lung (Christopher-Hennings et al., 2001; Horter et al., 2002; Mengeling et al., 1995, 1996a, 1996b; Wills et al., 1997a). In cases of late-term abortion and early farrowing, samples should be collected from weakborn, pre-suckle pigs, rather than mummies, aborted, or stillborn pigs (Done et al., 1992; Joo 1993; Van Alstine et al., 1993a; Zeman et al., 1993). Within affected litters, weakborn pigs are the most likely to be viremic, but the presence of high levels of maternal antibody to PRRS virus may hinder attempts at virus isolation.

The survivability of PRRS virus in diagnostic specimens exposed to different environmental temperatures has been evaluated in clinical specimens (lung, spleen, thymus, and serum). The current recommendation is that tissues and clinical specimens for virus isolation be kept at 4º C (40º F) or lower following collection and during shipment to a diagnostic laboratory in order to enhance the likelihood of isolating virus (Van Alstine et al., 1993a, 1993b). If long-term storage of samples is necessary, specimens should be kept frozen at minus (~)70º C without repeated cycles of freeze-thawing.

Detection of virus or antigen (live or dead)
The frozen tissue section fluorescent antibody test (FA) and immunohistochemistry (IHC) test may be used for detecting PRRS virus antigen in tissues. The direct FA test on frozen tissue sections is inexpensive and rapid. The test is specific (has few false positive results), but is not always very sensitive (tends to result in a few false negative results). Sample quality greatly affects FA test results. Tissue should be collected from recently dead or euthanized pigs and promptly refrigerated or frozen. In contrast, IHC is useful for detecting virus in formalin-fixed tissues. IHC is more sensitive than direct FA examination of frozen tissues, but takes more time and is more expensive than the FA test. A definitive diagnosis can be accomplished by detection of microscopic
lesions characteristic of PRRS virus in conjunction with IHC or FA tests.

For direct FA examination, fresh or frozen tissues should be submitted. For IHC examination, tissues should be fixed in 10 percent neutral buffered formalin. Preferred tissues for these tests are heart, kidney, lung, lymph nodes, spleen, thymus, and tonsil. PRRS viral antigens may also be detected in the adrenal gland, intestine, liver, and occasionally in the brain (Halbur et al., 1996; Rossow et al., 1999). When performing antigen detection tests, such as FA and IHC, laboratories must choose whether to test for the US or European PRRS virus. Veterinarians should familiarize themselves with their local laboratory to understand which test should be run.

**Genetic-based testing for PRRS virus**

Polymerase chain reaction (PCR)-based tests have been developed for detecting PRRS virus RNA in clinical specimens. Since the virus does not need to be isolated in cell culture to detect the viral RNA, PCR can be performed in a shorter amount of time than virus isolation. As a general principle, PCR-based assays are believed to be highly sensitive and highly specific (Benson et al., 2002; Horter et al. 2002).

Several types of PCR-based assays have been developed, most being designed to detect regions of the virus called ORF7, ORF6 or ORF1b and can be run directly on diagnostic specimens (Chen and Plagemann 1995; Christopher-Hennings et al., 1995a; Cook and Spatz, 1998; Egli et al., 2001; Gilbert et al., 1997; Legeay et al., 1997; Mardassi et al., 1994; Oleksiewicz et al., 1998, Shin et al., 1997; Spagnuolo-Weaver et al., 1998; Suarez et al., 1994; Van Wonsel et al., 1994). Some PCR assays use a “nested” procedure (RT-nPCR) for added sensitivity. More recently, automated fluorogenic PCR-based tests, such as the TaqMan™ PCR (Egli et al., 2001; Spagnuolo-Weaver et al., 2000) or “Molecular Beacon” PCR (Carlson et al., 2002) have been developed for detecting PRRS virus in clinical specimens. These PCRs are believed to improve the reliability and consistency of conventional PCR tests for PRRS virus detection.

Historically, the primary diagnostic application of PRRS RT-PCR has been for the detection of virus in boar semen (Christopher-Hennings et al., 1995a, 1995b, 1996; Shin et al., 1997). PCR is particularly useful for the detection of virus in samples like semen or feces because these samples are difficult to analyze by traditional methods. It has also been found useful for detection of PRRS virus in fetal tissues and thoracic fluids (Benson et al., 2002). The use of PCR assays has become more common both for the diagnosis of PRRS and to aid in herd monitoring, i.e., screening of replacement animals, detection of persistently infected animals, and test and removal programs (Bierk et al., 2001; Dee et al., 2001a; Horter et al., 2002; Kleiboeker et al., 2002). The performance of PCR testing among different laboratories may vary depending upon sample condition, sample processing, laboratory technique, and the skills and experience of the technician performing the assay. Therefore, it is important for laboratories performing PCR to validate their assays and provide this information to producers and veterinarians using their specific PCR test.

**Assays for Detection of Serum Antibodies (Seroology)**

The indirect fluorescent antibody (IFA) test, serum virus neutralization (SVN) test, immunoperoxidase monolayer assay (IPMA), and enzyme-linked immunosorbent assay (ELISA) have all been used for the detection of antibodies specific for PRRS virus. The IFA, SVN, and ELISA are currently available in most North American veterinary diagnostic laboratories, while IPMA has been extensively used in Europe.

The IFA is thought to have high specificity (99.5%) but unknown sensitivity for individual animals (Yoon et al., 1992a). An advantage of the IFA test compared to ELISA is that the magnitude of the antibody titer can be determined. An antibody titer of 16 or 20, depending upon the initial serum dilution for the test, is considered positive. The IFA reliably detects specific antibodies for 2 to 3 months after infection (Frey et al., 1992; Yoon et al., 1995b). Test results or endpoint antibody titers will vary depending on the degree to which the PRRS virus strain used in the assay differs from the isolate that infected the pig (Bautista et al., 1993a).

The IPMA is also considered to be a highly specific and sensitive test (Wensvoort et al., 1991). In one comparative study, the sensitivity of IPMA was better than that of a commercial ELISA (Drew, 1995). Antibodies to PRRS virus are usually detected by IPMA between 7 and 15 days after infection (Ohlinger et al., 1992; Wensvoort et al., 1991). Like the IFA, IPMA also reliably detects specific antibodies for 2 to 3 months after infection (Frey et al., 1992; Ohlinger et al., 1992; Yoon et al., 1995a). The relatedness of the virus strain used in the assay and the virus strain infecting the pig will
likely affect the performance of the IPMA test (Wensvoort et al., 1992).

The ELISA is also reported to be sensitive and specific (Albina et al., 1992, Edwards et al., 1994; Nodelijk et al., 1996; O’Connor et al., 2002; Takikawa et al., 1996). One disadvantage of the ELISA format reported during the developmental stage was an unacceptable number of false positive results (Edwards et al., 1994; Paton et al., 1992b). Several ELISA formats have been described: an indirect ELISA using a sample to positive (S/P) ratio system (Yoon et al., 1995a), an indirect ELISA using direct OD values (Albina et al., 1992; Cho et al., 1996; Takikawa et al., 1996), and a blocking ELISA (Ferrin et al., 2002; Houben et al., 1995a; Sorensen et al., 1997; Zhou et al 2001). In the commercial ELISA kit (HerdChek® PRRS ELISA, IDEXX Laboratories Inc., Westbrook, Maine), a S/P ratio ≥0.4 is considered positive. Using the S/P ratio of 0.4 as a cut off, PRRS virus-specific antibody is detected in young pigs between 10 to 14 days post inoculation under experimental conditions and peaks at 2 to 3 months (Yoon et al., 1995a). The specificity of the HerdChek® PRRS ELISA has been estimated to be between 99.3 and 99.5 percent (O’Connor et al., 2002; Nodelijk et al., 1996). Recently a new version of the assay (HerdChek® 2XR) has been made available to veterinary diagnostic laboratories and practitioners. No published information on its performance or comparison with the previous assay is available at present. Uniformity in the manufacturing of the kit and a high degree of automation in performing the test in the diagnostic laboratory result in less variation in the results from the commercial ELISA compared to other assays. Other advantages of the commercial ELISA include: detection of antibody against North American and European PRRS virus strains, fast turnaround time, and licensure by USDA and AgCanada.

The SVN test is also considered a specific test, but previous studies have suggested that SVN is less sensitive than IFA and ELISA (Benfield et al., 1992b; Morrison et al., 1992). The low sensitivity of the test is primarily due to the fact that neutralizing antibodies (the type of antibody detected by the SVN test) against PRRS virus develop as late as 1 to 2 months after infection (Frey et al., 1992; Goyal and Collins, 1992; Minehart et al., 1992; Morrison et al., 1992; Nelson et al., 1994; Yoon et al., 1995a). Currently, a titer of ≥4 is considered positive. The SVN test is best considered a research tool rather than a routine diagnostic test because of its laborious nature. As with IFA and IPMA, test results are greatly influenced by the degree of relatedness between the isolate employed in the test and the isolate infecting pigs (Wensvoort et al., 1992; Yoon et al., 1997).

Interpretation of Serologic Results

Antibodies specific for PRRS virus often do not persist for the lifetime of an animal. In pigs exposed to PRRS virus under experimental conditions, virus-specific antibodies were initially detected by the IgG-IFA, IPMA, ELISA, and the SVN test at 5 to 9, 9 to 11, 9 to 13, and 9 to 28 days post inoculation, respectively. PRRS virus-specific IgM antibodies are detected within 5 days post inoculation and persist 21 to 28 days post inoculation (Park et al., 1995). Depending on the assay, antibody levels reach their peak values by 30 to 50 (IFA), 35 to 50 (IPMA), 30 to 50 (ELISA), and 60 to 90 days post inoculation (SVN) (Yoon et al., 1995a), after which they begin to decline. Yoon et al. (1995a) estimated from experimental and field observations that antibody titers approach undetectable levels by 4 to 5 months (IFA), 4 to ≥10 months (ELISA), 11 to 12 months (IPMA), and 12 months (SVN) post infection. The same time frame would be expected in those pigs that have been vaccinated with MLV vaccine (but have not been previously exposed to wild PRRS virus).

Several problems or limitations should be taken into account when interpreting PRRS serology. Results from a single blood sample collected from an individual pig showing clinical signs consistent with PRRS, is generally insufficient for confirming a diagnosis of PRRS (Van Alstine et al., 1993b). Positive results may or may not mean that PRRS virus caused clinical disease (Henry, 1994). The possible presence of maternal antibody should be considered when interpreting serology results. Albina et al. (1994) reported that maternal antibody was detected in the serum of piglets tested 4 days after birth and disappeared by 3 weeks of age. Alternatively, maternal antibody specific for PRRS virus has been reported to persist as long as 4 to 10 weeks of age (Goyal, 1993; Houben et al., 1995b; Nodelijk et al., 1997) and occasionally up to 16 weeks of age in pigs nursing immune dams (Van Alstine et al., 1993b).

Since antibodies often do not persist for the lifetime of an animal and because of the relatively short duration of IFA and/or ELISA antibodies, it is generally recommended that young pigs, rather than breeding stock, be tested to determine a herd’s PRRS virus infection status. In single-site, farrow-to-finish swine herds, the seroprevalence of PRRS virus
infection is usually considered to be highest in the grow-finish unit.

Negative PRRS serology on samples at one point in time can have several possible interpretations (AASP Subcommittee on PRRS, 1996):

1. The pigs are not infected with virus.
2. The pigs were recently infected with virus but have not yet had time to seroconvert.
3. The pigs were infected with virus some time ago, but have since become seronegative.
4. The result was falsely negative due to poor sensitivity of the test or a laboratory error.

Therefore, if using single point-in-time samples, PRRS serology must be used in conjunction with valid population sampling methods and knowledge of a herd’s history to determine if the herd has been exposed to PRRS virus. Current serologic tests are better suited for determining the status of a population, not necessarily the status of individual animals.

Diagnosis of PRRS virus infection as the cause of reproductive failure or respiratory disease can be achieved by showing a change in antibody titer (i.e., rising antibody titer) in paired serum samples. However, a definitive diagnostic evaluation of PRRS with respect to clinical disease requires that serological information be interpreted in combination with results from virus isolation and/or detection of antigenic or genomic material (Christianson and Joo, 1994; Goyal, 1993). It is important to bear in mind that the current serologic assays used in the diagnostic setting cannot routinely differentiate vaccine-derived antibodies from field isolate-derived antibodies.

The occurrence of false positive serologic results on the commercial ELISA has been a concern for diagnosticians and practitioners, particularly in expected-negative herds or groups (Keay et al., 2002; O’Connor et al., 2002; Torremorell et al., 2002). Field observations in "expected-negative" herds have suggested that false positive animals (“singleton reactors”) occur at a rate of 0.5 to 2 percent. Most suspected false positive animals have ELISA S/P values around the 0.4 cut off value, but S/P ratios can occasionally exceed 1.0 in these animals. At present, no information regarding the occurrence of false positives is available on the recently released 2XR version of the HerdChek® ELISA. Current recommendations for evaluating suspected false positive animals include repeating the test, testing by other serological methods in conjunction with PCR, re-sampling the suspected false positive animal and re-testing, or occasionally, sacrificing the animal and conducting a complete diagnostic work-up. A few diagnostic laboratories have developed in-house ELISAs for more specific detection of positive animals (Ferrin et al., 2002; Zhou et al 2001). Such tests are available on an experimental basis at present.

**Differential Testing**

Serologic assays cannot routinely differentiate antibodies to field isolates from vaccine-derived antibodies. However, characterization of virus isolates is possible by several methods. Panels of monoclonal antibodies can easily differentiate European isolates from North American isolates and vice versa (Dea et al., 1996; Drew et al., 1995; Nelson et al., 1993, 1996; Yang et al., 1999, 2000). Using this technique, Nelson et al. (1996) could find no evidence of the Lelystad virus (LV) or LV-like PRRS viruses in Midwestern US swine herds after evaluating 306 field isolates collected before 1995. However, recent reports of “EuroPRRS” virus, i.e., Lelystad-like virus or European PRRS virus in North America (Dewey et al., 2000) suggest that monoclonal antibody analysis of isolates may be useful for differentiation of isolates. Monoclonal antibody analysis can also be used to differentiate commercial modified-live vaccine virus from field isolates (Yang et al 2000).

Molecular biology has also made it possible to characterize PRRS virus isolates using PCR, a restriction fragment length polymorphism (RFLP) assay (Wesley et al., 1998a; Umthun et al. 1999), and direct DNA sequencing (Kapur et al., 1996; Yoon et al 2001). A PCR-based technique has been developed to differentiate North American from European isolates (Christopher-Hennings et al., 1995a; Egli et al., 2001; Gilbert et al., 1997; Mardassi et al., 1994). Although the investigators demonstrated its usefulness, PCR was not routinely used for that purpose in North American until the recent report of the presence of European-like PRRS virus in North America (Dewey et al., 2000). Currently, several diagnostic laboratories conduct differential PCR on suspect cases as per request or at the diagnostician’s discretion. More recently, a heteroduplex mobility assay (HMA) has been developed for a rapid identification and differentiation of vaccine-like virus from field viruses (Key et al., 2002a) but has only limited availability.

The RFLP assay is a crude technique for differentiating one PRRS virus isolate from another.
It was developed early on in the genesis of PRRS virus diagnostic tools but has recently fallen out of favor. The RFLP technique involves virus isolation followed by restriction endonuclease digestion. Restriction endonucleases are enzymes that make cuts at specific places in a genomic sequence. Different PRRS viruses differ in their genomic sequences, so fragments of different sizes are produced. The lengths of these fragments are then assigned a 3-digit number, e.g., 2-5-2 or 1-4-2. The RFLP pattern of a virus is a symbolic code. That is, RFLP patterns have no known association with specific viral characteristics, such as virulence or antigenic relatedness, and in that sense, the results have limited usefulness. Also, the RFLP cut pattern is representative of only a small percentage of the entire genomic sequence.

In contrast to RFLP, direct genomic sequence analysis of PRRS virus detects differences and similarities between isolates, enabling investigators to differentiate viruses at the genetic level. Sequencing provides investigators with the exact and complete sequence of the desired part of the genome. The sequencing is able to show nucleotide mutations, additions, and deletions that RFLP analysis may miss. Sequence analysis and comparison can be a useful tool for veterinarians or investigators who are monitoring virus spread through pig flow within a production system over time. With sequence analysis it is possible to differentiate vaccine from field isolates, analyze changes to the PRRS virus that occur over time, and describe the “family tree” among various PRRS virus isolates both within and between populations of animals (Andreyev et al., 1997; Dee et al., 2001b; Key et al., 2001; Meng, 2000; Murtaugh et al., 1995; Nelsen et al., 1999; Pirzadeh et al. 1998; Rowland et al., 1999).

Numerous field-based descriptive studies have revealed remarkable genetic variability among PRRS viruses (Andreyev et al. 1997; Kapur et al., 1996; Key et al., 2001b; Meng 2000), suggesting that PRRS virus is rapidly evolving over time. However, experiments examining PRRS virus mutation have not explained all of the observed variability seen among field isolates. Under experimental conditions, the divergence between the parent strain and the mutated viruses arising from the parent strain resulted in sequence differences of less than one percent (Chang et al., 2002; Rowland et al., 1999). This is in contrast to sequence differences of >15% commonly seen in the field. On this basis of the experimental evidence, diagnostic laboratories providing PRRS sequencing results consider sequence changes greater than 0.5 to 1.0 percent to be suggestive of an interpretation that two viruses are not closely related (Christopher-Hennings et al., 2002). Although sequencing is the best tool to assess the relatedness of strains, more definitive statements generally cannot be provided. That is, the origin, disease causing potential, or biological properties of a field isolate cannot yet be predicted on the basis of genomic sequence information. Likewise, similarity of the genomic sequence between field isolates and vaccines may not be an accurate predictor of efficacy and should not be used in selecting a vaccine.

References


Bautista EM, Goyal SM, Yoon IJ, et al. 1993b. Comparison of porcine alveolar macrophages and CL2621 for the


