
Sponsors

University of Minnesota

College of Veterinary Medicine

College of Agricultural, Food and Environmental Sciences

Extension Service

Swine Center

Editors

W. Christopher Scruton

Stephen Claas

Layout

David Brown

Logo Design

Ruth Cronje, and Jan Swanson;

based on the original design by Dr. Robert Dunlop

Cover Design

Sarah Summerbell

The University of Minnesota is committed to the policy that all persons shall have equal access to its programs, facilities, and employment without regard to race, color, creed, religion, national origin, sex, age, marital status, disability, public assistance status, or sexual orientation.

Porcine parvovirus: A field investigation into vaccine failure

Brett Bower DVM

PIC USA, Franklin, KY

Introduction

Porcine parvovirus (PPV) is a tiny, single-stranded DNA virus that may persist in the environment for months. The primary sources of viral dissemination are oral secretions and feces from infected pigs. The virus is enzootic in most swine herds and sows are often immune. Owing to the long duration of maternal immunity, gilts present the greatest risk of infection. If infection takes place prior to pregnancy, clinical signs are not observed. However, if immunity has not developed before conception, early embryonic and fetal death may result. The clinical outcome is dependent upon the stage of gestation in which exposure occurs. If the conceptus is infected around 35 days of gestation or earlier, death and complete absorption result and the dam will likely return to estrus¹. Infection up to day 70 of gestation will produce fetal death and mummification². Because the rate of PPV spread within the uterus varies, the mummies produced will not be consistent in size. After day 70 of gestation, fetuses become immunocompetent and are able to fight off infection^{1,8}. Infection outside of gestation is not clinically or economically significant.

With PPV-induced reproductive failure, maternal illness is not seen during gestation and abortions are uncommon. Gilts are primarily affected and mummified piglets of various sizes are observed. Small litters associated with embryo loss, decreased conception rates, and increased numbers of stillborn piglets may be seen. Therefore, production records can assist in the diagnosis of this disease.

In the past several years, there has been a perceived increase in the number of PPV cases in herds using commercially available parvovirus vaccines. This increase has been associated with high health status farms and farms involved in PRRS-elimination projects in which gilt acclimatization is very limited. Multiple-site production, strict all-in—all-out management, and segregation of age groups within a herd have made it increasingly challenging to ensure gilt exposure to PPV prior to mating. Traditionally, environmental contamination has been the primary reservoir of PPV infection^{3,4}. However, intensive cleaning and disinfection practices currently employed in many production systems may be reducing the poten-

tial for exposure. Thus, susceptible gilt populations that have not been exposed to PPV are being created.

In the past, exposure to PPV was achieved during acclimatization by introducing replacement gilts to potentially contaminated areas, mixing gilts with older sows, and feeding gilts mummified fetuses and feces from the sow herd. Due to the variable immunity produced by these methods of natural exposure, inactivated PPV vaccine has been widely used as a control measure as well. Because of the long duration of maternal immunity, proper timing of vaccination is critical and difficult to determine. Passively acquired PPV antibodies may persist until 24 weeks of age⁵. Passive immunity may interfere with the development of active immunity post-vaccination. As soon as one week after becoming seronegative, pigs were shown to be susceptible to experimental PPV infection⁵. Gilts are often initially bred at 7 months of age and vaccinated at least 2 weeks before breeding. Therefore, vaccination timing is crucial in order to ensure that gilts do not have passively acquired antibodies that may reduce vaccine efficacy.

Inactivated PPV vaccines are widely used and can be found as monovalent vaccines or in combination with leptospirosis and erysipelas. The literature suggests that vaccination targeted at increasing humoral immunity is effective in preventing viremia³. High levels of circulating antibodies are needed for protection against PPV infection. Inactivated vaccine titers between 1:2 and 1:160 appear to be protective¹. However, current field observations may indicate that either commercial vaccine products are not stimulating protective titers or vaccine administration is not being properly performed.

Parvovirus diagnostic testing is routinely performed at diagnostic laboratories. Diagnostics are based upon serology and antigen detection tests. Serological procedures should be relied upon for diagnosis only when mummified fetuses are not available for testing. Diagnosis of PPV antigen is performed by immunofluorescence microscopy on fetal tissues. Only mummies less than 16cm crown to rump should be submitted for antigen testing as larger fetal tissues contain antibodies that interfere with the test⁶. Virus isolation is generally less rewarding than IF microscopy.

Hemagglutination inhibition (HI) is the most widely used assay for PPV antibody titers. HI titers are expressed as the reciprocal of the highest serum dilution that inhibits hemagglutination. In unvaccinated gilts, titers of <1:10 are considered to be negative and titers between 1:10 and 1:160 indicate the presence of maternal antibodies. Titers greater than or equal to 1:160 represent active infection⁷. Once a pig is naturally exposed to PPV, lifelong immunity is thought to result¹. In contrast, vaccination induces protection for a limited time period and must be periodically repeated. Serology testing performed on mummies infected after 70 days of gestation and neonatal piglets that have not yet nursed can be used to diagnose in utero infection. Prior to breeding, gilts need to be naturally infected with PPV or vaccinated in order to develop active immunity before conception.

The purpose of the following field investigation is to explore a case of reproductive failure suggestive of porcine parvovirus infection in a production system utilizing parvovirus vaccine. Additional cases will be presented at the meeting.

Field case

In early 1999, a 2500 sow farm in Nebraska was stocked using a modified-medicated early weaning protocol. Gilts were vaccinated twice prior to breeding for parvovirus, leptospirosis, erysipelas, SIV, and mycoplasma using commercially available products. Gilts began farrowing the third week in April of 1999. During the first six week farrowing period, total born per litter averaged 10.4 with born alive per litter averaging 6.6. An average of 3 mummies per litter was reported. Mummies recorded as a percent of the total pigs born ranged from 17.1% to 38.5%. The mummies seen were of varying sizes. The percent-

age of repeat services was also elevated but these results were difficult to interpret because the farm was newly stocked and in the early phases of production (please refer to **Table 1**). The farm was routinely monitored by monthly serology for pseudorabies, brucellosis, and PRRS and remained negative to these agents.

A random sample of gilts (250lbs.) located in the grower were bled for PPV shortly before vaccination. On the HI test, most of the animals were negative or showed very low titers (1:8, 1:16) with the highest titer reported to be 1:32 in one animal. These gilts tested negative to pseudorabies, brucellosis, and PRRS at this time as well.

As a result of the high incidence of mummies during the first six weeks of farrowing, an investigation of parvovirus infection was initiated. Mummified fetuses from six different sows were submitted to the diagnostic lab on two separate occasions. Fetuses ranged in size from 1.5cm to 23cm in crown to rump length. The fetuses did not display any histopathological lesions. Bacteriology testing failed to isolate any pathogens from the fetal tissues. PCR testing on pooled tissues was negative for PRRS as well. Fluorescent antibody examination of fetal lungs was negative for PPV. However, a negative fluorescent antibody test does not rule out PPV infection and the presence of significant numbers of mummified fetuses varying in size is strongly suggestive.

As a next step, vaccination protocols were reviewed with farm employees. The importance of proper vaccine use and storage was emphasized. A protocol was reinforced wherein two doses of inactivated parvovirus vaccine were administered twice prior to breeding and 7–10 days post farrowing. Production numbers began to improve in late May of 1999. An abrupt decrease in the number of mummies occurred and 0.77 mummies per litter during the

Table 1. Production data

Period Start	04/17/99	04/24/99	05/01/99	05/08/99	05/15/99	05/22/99	6 week average
Period End	04/23/99	04/30/99	05/07/99	05/14/99	05/21/99	05/28/99	
Farrowings	2	38	54	53	82	59	48
% Repeat Services	23.7%	23.6%	12.2%	17.9%	27.7%	30.7%	22.6%
Total Born	26	378	514	497	822	631	478
Live Born	14	222	288	339	602	464	322
Total Born/Litter	13	9.9	9.5	9.4	10	10.7	10.4
Live Born/Litter	7.0	5.8	5.3	6.4	7.3	7.9	6.6
Stillborn	2	11	54	31	52	59	34.8
SB% Total	7.7%	2.9%	10.5%	6.2%	6.3%	9.4%	7.2%
Mummies	10	145	172	127	168	108	122
Mummies% Total	38.5%	38.4%	33.5%	25.6%	20.4%	17.1%	28.9%
Mummies/Litter	5.0	3.8	3.2	2.4	2.0	1.8	3.0

next 6 weeks of production were reported. The percentage of mummies dropped from an average of 28.9% to an average of 7.0% during this time period. Excluding the first six weeks of poor production, the yearly total born per litter averaged 10.5 and born alive per litter averaged 9.2. Mummies were reported at a rate of 0.45 per litter per year or an average of 4.5% of the total born population.

Conclusion

In summary, PPV is a significant cause of reproductive failure. Infection during gestation is characterized by increased returns to service and elevated numbers of various sized mummies. The virus is endemic in many swine units but may present a problem in high health farms if natural exposure has not been achieved. The effectiveness of commercially available parvovirus vaccines strongly depends upon the timing of vaccination since maternal antibodies may persist for up to six months and interfere with the development of active immunity.

References

1. Muirhead, M. R., and Alexander, T. J. L. *Managing Pig Health and the Treatment of Disease*. Sheffield, UK: Enterprises Ltd; 1997-170-173.
2. Pye, D., Bates, J., Edwards, S. J., and Hollingworth, J. Development of a vaccine preventing parvovirus-induced reproductive failure in pigs. *Aust Vet J*. 1990; 67: 179-182.
3. Paul, P. S., Mengeling, W. L., and Brown, Jr., T. T., Effect of vaccinal and passive immunity on experimental infection of pigs with porcine parvovirus. *Am J Vet Res*. 1980; 41:1368-1371.
4. Mengeling, W. L., and Paul, P. S., Interepizootic survival of porcine parvovirus. *J Am Vet Med Assoc*. 1986; 188:1293-1295.
5. Paul, P. S., Mengeling, W. L., and Pirtle, E. C., Duration and biological half-life of passively acquired colostral antibodies to porcine parvovirus. *Am J Vet Res*. 1982; 43:1376-1379.
6. Mengeling, W. L. Porcine parvovirus. In: *Diseases of Swine*. 8th ed. Ames, IA: Iowa State University Press; 1999-187-197.
7. Wrathall, A. E., Field trials of an inactivated, oil-emulsion porcine parvovirus vaccine in British pig herds. *Vet Rec*. 122:411-418.
8. Goyal, S. M., Porcine parvovirus serology. *Allen D. Leman Swine Conference*. 1994; 63-64.
9. Joo, H. S., Johnson, R. H., and Watson, D. L. Serological procedures to determine time of infection of pigs with porcine parvovirus. *Aust Vet J*. 1978; 54:125-127.
10. Joo, H. S., and Johnson, R. H., Serological responses in pigs vaccinated with inactivated porcine parvovirus. *Aust Vet J*. 1977; 53: 550-552.

