

Decay of Colostrum-Derived Antibodies to Porcine Reproductive and Respiratory Syndrome (PRRS) Virus in Neonatal Swine Nursing Immune Dams

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Summary and Implications

The decay of colostrum-derived antibodies specific for porcine reproductive and respiratory syndrome virus (PRRSV) was characterized in young pigs. Six pigs per litter were randomly selected from seven multiparous sows in a commercial sow herd. Prior to farrowing, all sows were confirmed seropositive to PRRSV. Colostrum and serum samples were collected from each sow at the time of farrowing. The pigs were weaned and transported to an isolated facility at 7–10 days of age. Sera were collected periodically from all pigs between 3 and 40 days of age and assayed for PRRSV-specific antibodies by the serum neutralization (SN) test and ELISA. Serum and colostrum samples also were assayed for the presence of PRRSV. No virus was isolated from any of the serum or colostrum samples. During the observation period 10 of 42 pigs (ELISA) and 17 of 42 pigs (SN) showed a normal decay of antibodies. The mean half-life for the decay of maternal antibodies was estimated to be 16.2 days (ELISA) and 8.14 days (SN) (i.e., a relatively short half-life). A number of pigs, however, seroconverted during the observation period. Because there was no apparent source of virus, this suggested that colostrum or milk could be a source of virus. Early weaning (7–10 days) appeared to be ineffective at eliminating PRRSV infections in pigs.

Introduction

Porcine reproductive and respiratory syndrome virus infection (PRRS) is endemic in most pork-producing areas of the world. Because the appearance of PRRSV in Canada in the late 1970s, the United States in the mid-1980s, and Europe in 1990, it has become the most economically important viral disease of swine in the United States (7). Because of its economic impact on pork production, one important area of focus has been the development of effective control strategies.

In general, passive immunity provides a way to control diseases in young animals. The presence of colostrum-

derived antibodies, however, can adversely impact vaccination success by either of two mechanisms. First, passively acquired maternal antibodies may interfere with the response to vaccination. This has been observed in several other disease and animal species, such as pseudorabies virus (4) and parvovirus (3). The second mechanism that might be operative with PRRSV is antibody-dependent enhancement (ADE) (6). The ADE of virus infection has been reported for several viral diseases, for example feline infectious peritonitis and dengue fever in humans (2). In ADE, serum antibodies fail to neutralize the virus and actually enhance the severity of infection.

Our initial objective in evaluating the influence of colostrum-derived antibodies on PRRSV infection in neonatal pigs was to characterize antibody decay.

Materials and Methods

Seven multiparous sows that farrowed during a 10-day period were selected from a commercial sow farm that had been confirmed to be seropositive to PRRSV. Colostrum and serum samples were collected from each sow within 6 hours of farrowing. Colostrum samples were collected manually from a minimum of 3 mammary glands following intravenous administration of 40 IU of oxytocin. Whole colostrum samples were centrifuged at 1,100 X g for 10 minutes at 22°C and the lipid layer removed prior to antibody determination.

At 7 to 10 days of age, 6 piglets were randomly selected from each litter and transported to an isolation facility. Each litter was housed as a group in a large plastic tub and serologically monitored over time. Serum samples were collected from all offspring at 3, 10, 17–19, 24–26, 31–33, and 37–40 days of age. All samples were stored at -70°C until tested.

Virus isolation (VI) was attempted on all serum and colostrum samples by using both the MARC-145 cells and pulmonary alveolar macrophages (PAM). In addition, all serum and colostrum samples were assayed for the presence of PRRSV-specific antibodies by the SN test and a commercial ELISA. All samples were completely randomized and renumbered prior to submitting the samples for testing to avoid bias.

The rate of antibody decay, measured as antibody half-life, was determined for all pigs exhibiting declining titers throughout the study period. A linear regression curve was fitted to the natural logarithms (ln) of the antibody titers. Antibody half-life estimate was obtained using the equation: $h = -(\ln 2) / b$, where h is the estimated half-life of antibody and b is the slope of the regression line (1). Half-life for each pig was determined separately and the mean half-life was

used as a point source estimate. Approximate 95% confidence intervals on the mean half-lives were obtained.

Results and Discussion

No virus was isolated from any serum or colostrum samples by using either the PAM or MARC-145 cells. The negative VI results may be attributed to either a lack of susceptibility of the cell lines or interference of antibody present in the serum or colostrum.

Antibodies in colostrum were concentrated compared with those levels observed in the serum of sows at the time of farrowing as summarized in Table 1. During the observation period, 10 of 42 pigs (ELISA) and 17 of 42 pigs (SN) showed normal decay of antibodies. The mean antibody half-life for these pigs was 16.20 days (ELISA) and 8.14 days (SN). The 95% confidence intervals for the mean half-life were 13.68–18.72 days and 6.29–9.99 days for ELISA and SN antibodies, respectively. The relatively short half-life of both PRRSV-specific ELISA and SN antibodies may result in the misinterpretation of antibody titers as passive or active in the young pig, especially if paired or serial sampling is not used.

Interestingly, many of the remaining pigs seroconverted during the study period with no apparent new source of virus, suggesting that colostrum or milk can be a source of virus. This possibility was demonstrated in a subsequent study in our laboratory (5). Therefore, early weaning (7–10 days) may not be as effective as anticipated at eliminating PRRSV infections in pigs. Further information regarding colostrum-derived antibodies and infection with PRRSV remain to be investigated.

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References

1. Bryan, M., Zimmerman, J.J., and Berry, W.J. 1990. The use of half-lives and associated confidence intervals in biological research. *Vet. Res. Commun.* 14:235–240.
2. Kurane, I., Mady, B.J., and Ennis, F.A. 1991. Antibody-dependent enhancement of dengue virus infection. *Rev. Med. Virol.* 1:211–221.
3. Paul, P.S., Mengeling, W.L., and Brown, T.T. 1980. Effect of vaccinal and passive immunity on experimental infection with porcine parvovirus. *Am. J. Vet. Res.* 41:1368–1372.
4. Van Oirschot, J.T. 1987. Intranasal vaccinating of pigs against Aujeszky's disease: comparison of one or two doses of attenuated vaccines in pigs with high maternal antibody titers. *Res. Vet. Sci.* 42:12–16.
5. Wagstrom, E.A., Chang, C.C., Yoon, K.-J., and Zimmerman, J.J. 1998. Evaluation of shedding of porcine reproductive and respiratory syndrome (PRRS)

virus in mammary secretions of sows in commercial swine units. *Proceedings, 15th International Pig Veterinary Society Congress*, 2:98.

6. Yoon, K.-J., Wu, L.-L., Zimmerman, J.J., Hill, H.T., and Platt, K.B. 1996. Antibody dependent enhancement (ADE) of porcine reproductive and respiratory syndrome virus (PRRSV) infection in pigs. *Viral Immun.* 9:51–63.
7. Zimmerman, J.J., Yoon, K.-J., Stevenson, G., and Dee, S.A. 1998. *The 1998 porcine reproductive and respiratory syndrome compendium*. Published by the National Pork Producers Council in cooperation with the National Pork Board, Des Moines, IA. 128 pp.

Table 1. Titer comparison of sow serum and colostrum at farrowing.

Sow	SN		ELISA	
	Serum	Colostrum	Serum	Colostrum
1	4	32	.15	.72
2	64	>256	1.83	1.71
3	8	8	.23	.43
4	<2	16	.78	1.25
5	64	>256	1.15	2.71
6	8	32	.53	.69
7	2	8	.38	.59