CONSEQUENCES OF MATERNAL EXPOSURE TO PORCINE PARVOVIRUS AT DIFFERENT TIMES DURING GESTATION

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Porcine parvovirus (PPV) is a major cause of maternal reproductive failure of swine. Although dams seldom have obvious clinical signs, transplacental infection often results in embryonic and fetal death. Initial studies indicated that the development of reproductive failure depended on the time of gestation at which dams were exposed to the virus. However, the full interval of maternal susceptibility was not defined. We have since conducted a series of experiments wherein dams were exposed oronasally to PPV at selected times during gestation and their litters were subsequently examined for evidence of virus-induced changes. The purpose of this report is to summarize several of these experiments.

Twenty gilts were exposed to PPV at the onset of gestation and killed 22 + 1 days later. Litters of 12 gilts were infected and PPV had caused death of 1 or more embryos in each of the infected litters. Most of the embryos were believed to have been infected transplacentally. However, a few may have been infected by intrauterine spread of virus from adjacent transplacentally infected littermates.

Two gilts were exposed to PPV at either the 7th or 14th day of gestation and killed 49 days later. Both litters were infected and were comprised of necrotic remnants of infected dead embryos, or early fetuses, as well as clinically normal noninfected fetuses. Circumstantial evidence indicated that the litter of the gilt exposed at 7 days of gestation may have been comprised of additional embryos which were completely resorbed before the litter was examined.

Twenty-three gilts were exposed to PPV at various times between 23 and 44 days of gestation and were killed between 28 and 45 days later. Litters of 20 gilts were infected and PPV had caused death of 1 or more fetuses in each of the infected litters. In some litters, all of the fetuses had died of infection.

Nine gilts were exposed to PPV at 56 days of gestation and either killed 53 or 54 days later (4 gilts) or allowed to farrow normally (5 gilts). Litters of 7 gilts were infected and PPV had caused death of fetuses in 5 of the infected litters.

Fifteen gilts were exposed to PPV at various times between 67 and 80 days of gestation and were killed between 28 and 40 days later. Litters of 11 of the gilts were infected, but PPV had caused neither fetal death nor any other pathologic sequelae. Tests of fetal serums for hemagglutination inhibition (HI) antibody for PPV revealed that most infected fetuses had responded immunologically to the virus. Most HI titers were 320 or greater and, therefore, similar to those found in serums of postnatal pigs.

Collectively the results of these experiments established 2 salient features of PPV-induced reproductive failure. First, none of the gilts became clinically ill or aborted following exposure to PPV. Second, reproductive failure developed only when gilts were exposed to PPV during about the first one-half of gestation, even though the incidence of transplacental infection was similar for gilts exposed before, at or after midgestation (Table).

Results of Oronasal Exposure of Gilts to Porcine Parvovirus Before, At, or After Midgestation

Stage of gestation exposed (days)	No. of gilts exposed	No. (%) of litters infected transplacentally	No. (%) of infected litters with PPV-induced reproductive failure
<56	45	34 (76)	34 (100)
56	9	7 (78)	5 (71)
≥67	15	11 (73)	0 (0)

Selected references: Redman, D. R., Bohl, E. H., and Ferguson, L. C.: Infect. Immun. 1974, 10:718; Bachmann, P. A., Sheffy, B. E., and Vaughan, J. T.: Infect. Immun. 1975, 12:455; Cutlip, R. C., and Mengeling, W. L.: Am. J. Vet. Res. 1975, 36:1751; Joo, H. S., Donaldson-Wood, C. R., and Johnson, R. H.: Arch. Virol. 1976, 51: 123; Mengeling, W. L., and Cutlip, R. C.: Am. J. Vet. Res. 1976, 37:1393; Mengeling, W. L., Cutlip, R. C., and Barnett, D.: Proc. 5th Intl. Pig Vet. Congr. 1978, K.A. 15; Mengeling, W. L.: Can. J. Comp. Med. 1979, 43:106; Mengeling, W. L., Paul, P. S., and Brown, T. T.: Arch. Virol. 1980, 65:55.