ANTIBODY RESPONSES IN LABORATORY ANIMALS INOCULATED WITH INACTIVATED PORCINE PARVOVIRUS VACCINE
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The etiologic role of porcine parvovirus (PPV) in reproductive failure in swine is well established (Donaldson-Wood et al. 1977, Gillick 1977, Mengeling 1978). Most often this is manifested as fetal death and resorption or mummification, although infertility, abortion, stillbirth, neonatal death and reduced viability may also be consequences of in utero PPV infection (Cartwright and Huck 1967, Johnson 1969, Kirkbridge and McAdaraugh 1978). PPV infections are now ubiquitous in swine populations throughout the world, and nearly all pigs over 12 months of age have been exposed to the virus (Johnson et al. 1976, Thacker et al. 1981). In the United States, reports indicate that PPV is 'the most common infectious agent associated with reproductive failure (Mengeling 1978, Thacker et al. 1981). In an attempt to control PPVinduced disease, both inactivated and modified live virus vaccine have been developed (Joo and Johnson 1977, Mengeling et al. 1979, Paul and Mengeling, 1980). Several of these vaccine preparations have been evaluated in controlled experiments with pregnant pigs (Fuzisaki et al. 1978, Mengeling et al. 1979, Cutler et al. 1980, Sorensen and Askaa 1981). The results of such studies, employing small numbers of animals, indicate that the vaccines can prevent PPV-induced reproductive failure. Since several PPV vaccines are available in the market, immunization of breeding stock with an effective vaccine is recommended to prevent PPV-induced diseases.

In testing efficacy of PPV vaccine, a number of PPV seronegative pregnant pigs are required. Unfortunately it is very difficult to find seronegative pigs and expensive to maintain such animals in an isolated environment. Laboratory animals are used for routine testing of a variety of vaccines. If PPV vaccine can be routinely tested in laboratory animals, testing will be easy, inexpensive, and the subsequent quality of vaccine can be readily controlled. To this date, no report is available on the antibody responses of PPV vaccines in laboratory animals. The present studies are designed to compare the antibody response of pigs to laboratory animals. Additional variables examined were the optimal virus concentration, ratio between virus and adjuvant and the time between initial and booster injections.

A local isolate of PPV was used for vaccine preparation and the virus was propagated in a continuous line of swine testicular (ST) cells. Beta-propiolactone (BPL) inactivated vaccine adjuvanted with aluminum hydroxide gel was prepared by the procedure previously described (Joo and Johnson 1977). Virus concentration was adjusted to contain various hemagglutination (HA) units. The aluminum hydroxide gel was prepared to contain no supernatant water after overnight sedimentation, and this was sterilized by autoclaving. Two volumes of the inactivated virus were mixed with 2 volumes of the gel (50%), I volume of the gel plus 1 volume of tissue culture media (25%) or 2 volumes of the media (0%). Several PPV vaccines were also obtained from commercial sources. Adult white rabbits (2.5 kg) and guinea pigs (200-500 gm) were purchased from a local commercial source. PPV seronegative finishing pigs were obtained from the University farm. All animals were pretested for serostatus of PPV and seronegative animals were housed in individual cages or pens in an isolation room. Rabbits, guinea pigs and pigs were inoculated intramuscularly with 1 ml, 0.2 - 0.5 ml and 5 ml of PPV vaccine, respectively, and hemagglutination inhibition (HI) antibody titers were measured at weekly intervals by the method described previously (Joo et al. 1975). Second

vaccination was made with the same dose at different intervals following the initial vaccination.

Results showed that antibody titers of rabbits and guinea pigs following vaccination with BPL inactivated gel vaccines were similar or identical when compared to those in pigs. In vaccinated animals, PPV HI antibody titers of >1:32 were first detected at 1 week post vaccination and the titers rose to 1:256 at 2 weeks post vaccination. A booster inoculation at 2 weeks following the initial vaccination caused an increasing HI antibody titer up to 4-fold, when measured at 2 weeks post-booster vaccination. From these results, guinea pigs were the choice of laboratory animals as they are sensitive and easy to handle. Subsequent investigations of antibody response to various vaccine preparations were carried out with guinea pigs.

Different concentrations of gel adjuvant in the vaccine caused significant variations in antibody preparation with guinea pigs. A comparison of gel concentration between 50, 25 and 0% showed mean HI titers of 32, <8 and <8 at 3 weeks after first vaccination and 512, 32 and 8 at 1 week after the second vaccination, respectively. The HI antibody titers found in guinea pigs were proportional to the concentrations of PPV hemagglutinin in the vaccines. A consistent antibody production with significant levels in guinea pigs was observed when the vaccine contained PPV at HA titers of >1:128/0.1 ml and adjuvanted with the gel at a 50% concentration. The booster effects were greater when second vaccination was carried out at 4 weeks than did at 2 weeks after the initial vaccination.

Reports suggest that a certain level of humoral antibody in pregnant dams prevents the development of viremia and subsequent transplacental PPV infection (Mengeling et al. 1979, Paul and Mengeling 1980). Present results demonstrated that immunological response of laboratory animals to PPV vaccination was equivalent to that in pigs. Consequently the use of laboratory animals would be greatly contributed in testing the potency of vaccine. Furthermore, the efficacy test in pregnant pigs may not be necessary, if a PPV vaccine produces a significant antibody titer in laboratory animals. However, a precise comparative study between immunogenicity in laboratory animals and protection in host animals required before the efficacy test in pregnant pigs is eliminated.

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