PORCINE PARVOVIRUS: VIRUS EXCRETION AND ANTIBODY DEVELOPMENT
AFTER EXPERIMENTAL INFECTION AND NATURAL TRANSMISSION.
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Porcine parvovirus (PPV) is a common cause of reproductive failure of swine characterized by embryonic and fetal death usually without maternal clinical signs. It is known, that gilts especially are at risk due to lack of immunological protection, when maternal antibodies have disappeared. Serological examinations in Danish herds, however, have shown that a variable number of sows in different herds also are negative for antibody to PPV and therefore susceptible to the infection. Two methods for immunoprophylaxis are available: 1) Vaccination of susceptible breeding animals, and 2) natural immunization by establishing an active infection of gilts before breeding. To obtain natural immunization an effective spread of the infection among gilts is necessary. As the spread apparently can be insufficient, an experiment was set up to obtain more knowledge about virus excretion and antibody development after infection with PPV.

Three gilts (no. 1, 2 & 3) without antibody to PPV and two gilts (no. 4 & 5) with passively acquired antibodies were infected by oral and nasal inoculation of PPV. Three PPV antibody negative gilts (no. 6, 7 & 8) were kept in direct contact with the infected gilts from the 3th day after infection. Sta-bilized blood samples for preparation of leucocyte cultures and stimulation of leucocyte cultures with concanavalin A (Con A) and non stabilized blood samples for serum preparation were taken requiarly from all gilts as were faecal and nasal swabs. Urine samples were collected from contact exposed gilts. The gilts were killed after 33-47 days and 10% tissue homogenates were prepared from spleen, liver, lung, small and large intestine, mesenteric lymph nodes and tonsils. Leucocytes from blood, tonsils, spleen and mesenteric lymph nodes were cultured and stimulated with Con A. All the samples were inoculated on primary

pig kidney cell cultures, which were examined for evidence of PPV replication by immunofluorescence microscopy after one subcultivation.

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Serumsamples were also examined for antibody to PPV by hemagglutination inhibition (HI) tests.

All the gilts developed an antibody response with high titers to PPV 7-10 days after exposure showing, that an active infection had occurred.

PPV was isolated from rectal- and nasal swabs of gilts no. 1, 2, 6 & 7 between 3 and 14 days after exposure, from a nasal swab of gilt no. 4 three days after exposure and from urine of gilt no. 6, 7 & 8 collected between 4 and 9 days after exposure. By titration of the PPV positive samples the concentration of PPV was estimated to be less than 10 TCID₅₀ per 0.5 ml sample. PPV was isolated from serum of gilt no. 1, 2, 3, 6, 7 & 8 between 3 and 11 days after exposure. Maximum concentration of PPV was estimated to about 10 TCID₅₀ per 0.5 ml serum. PPV was isolated from non stimulated leucocytes of gilt no. 1, 2, 3, 6, 7 & 8 and from Con A stimulated leucocytes of gilt no. 6, 7 & 8 between 3 and 11 days after exposure. From Con A stimulated leucocytes of gilt no. 1, 2 & 3 PPV was isolated up to 35 days after exposure. The samples collected after killing of the gilts were all negative for PPV.

Conclusions:

Excretion of PPV in nasal secretions, faeces and urine of gilts occurs within about 2 weeks after infection. This information, and the finding, that the concentration of PPV in the excretions is low, contribute to explain, why the spread of PPV infection within individual herds can be insufficient resulting in low herd immunity. Viremia occurs in PPV antibody negative gilts and the virus can be found in serum within about 11 days after infection. In leucocytes PPV can persist for some weeks after infection.

An antibody response with high titers to PPV

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