CLINICAL AND VACCINE IMMUNOLOGY, Oct. 2011, p. 1644–1649 Vol. 18, No. 10
1556-6811/11/$12.00 doi:10.1128/CVI.05183-11

Vaccination of Dams Increases Antibody Titer and Improves Growth Parameters in Finisher Pigs Subclinically Infected with Porcine Circovirus Type 2[▽]

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Received 20 May 2011/Returned for modification 8 June 2011/Accepted 4 August 2011

Porcine circovirus type 2 (PCV2) is the obligate infectious agent in postweaning multisystemic wasting syndrome (PMWS) of pigs. To control PMWS, we vaccinated dams at 4 and 2 weeks before pregnancy and again in the 12th week of gestation with an inactivated PCV2 vaccine (Circovac). Two producer farms run under the control of Swiss Swine Health Organization were selected for the experiment. Previously, in one farm PMWS was diagnosed on pigs after weaning, whereas in the other farm, pigs wasted during the fattening period. For the experiments 113 dams were randomly vaccinated, and 111 dams were sham injected. Vaccination increased serum antibodies in dams 3- to 9-fold, accompanied by serum antibody titer increases in their offspring. In the sixth week of life, progeny from vaccinated dams had about the same IgG antibody titers as progeny of unvaccinated dams at the third day of life. In sera of vaccinated dams only low concentrations of PCV2 DNA were detected, and no progeny developed PMWS. Interestingly, at day 56 four progeny of unvaccinated dams tested positive for anti-PCV2 IgM antibodies, indicating a primary infection with PCV2. Of economic importance is the observation that progeny of vaccinated dams had a significantly higher daily weight gain in the fattening period (farm X, +51 g/day; farm Y, +30 g/day) and thus a shortened fattening period of about 6 days compared to progeny of controls. To our knowledge this is the first demonstration of subclinical circovirus infection and its effects on growth performance of fattening pigs by vaccination of dams.

Postweaning multisystemic wasting syndrome (PMWS) in pigs was first described in Canada (18) and has since been recognized as one of the economically most important swine diseases worldwide (2, 9, 19, 21, 24, 44). PMWS emerged as an epizootic disease in Switzerland in 2003 to 2004 even though cofactors described as important for PMWS development, including porcine reproductive and respiratory syndrome (PRRS), enzootic pneumonia (EP), actinobazillosis, and progressive atrophic rhinitis (pRA), were not present (54).

PMWS is an acute or chronic disease affecting animals at the age of 5 to 16 weeks (1, 11) or exceptionally until 30 weeks of age (37). Typical signs are wasting, profuse diarrhea, and dyspnea, and pigs may have gastric ulcers, enlarged lymph nodes, anemia, icterus, hemorrhages, vasculitis, or edema in various organs (1, 18, 39, 42, 43).

Various porcine circovirus type 2 (PCV2) genotype group members have the potential to be involved in the PMWS etiology (9, 19, 21, 24, 39, 44). Nevertheless, PCV2 can be detected in healthy pigs or isolated from various cells and organs, including peripheral blood, mononuclear cells, dendritic cells, and lymphocytes, and viral antigen is often found in defined lymphatic areas in lymph nodes, tonsils, spleen, and thymus (3, 4) or is scattered in their supporting reticular cells, associated with irregular tissue architecture and in macrophages (39, 49). In other cases, PCV2 was diagnosed in lung, liver, kidney, and the gastrointestinal tract and, in rare cases, in apoptotic vascular endothelial cells of the brain (55).

As PCV2 can replicate in multiple cells of various organs to measurable titers in clinically healthy or diseased animals, the virus may be present in serum or all other body fluids (1, 43) including semen (30, 41). Infection of naïve animals may occur by direct contact with infected animals and their secretions; airborne dissemination must be considered due to high viral loads in large farms (26). In addition, natural vertical transmission was diagnosed in field cases (20, 53) and could be induced experimentally (33, 40). Experimentally infected dams delivered dead and stillborn piglets. PCV2 infection in fetuses was verified and was associated with myocarditis, fibrosis, and degeneration of the myocardium as well as depletion of lymphocytes (32, 38). Recent evidence further suggests that intrauterine infection may have been underestimated at least in some herds (45).

In a retrospective epidemiological study, PCV2 could be traced back to 1979 in Switzerland (54). Nevertheless, the first PMWS case was not confirmed until 2001 (5). However, the epizooty started in late 2003 in areas with large swine populations (52).

PCV2 has been endemic worldwide since the mid-1990s and can be isolated from PMWS-diseased and clinically healthy animals. PCV2-specific antibodies are detected in almost all pigs (1, 16, 29, 36, 48, 51). Another issue is the observation that the profiles of PCV2 serum antibody titers of pigs from

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▽ Published ahead of print on 18 August 2011.