RESEARCH ABSTRACT

SWINE HEALTH

Title: Is humoral immunity defective in PCV-2 infected piglets?- NPB #07-201

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SCIENTIFIC ABSTRACT

A total of 64 germfree (GF) isolator piglets were studied in three different experiments that compared the effect of infection with PCV-2, SIV and PRRSV on: (1) the ability of piglets to resolve the viral infection, (2) changes in total IgG, IgM and IgG levels in blood and BAL, (3) phenotypic differences in blood NK, T and B cells and at the site of infection, (4) the ability to make antibody responses to the virus, (5) the ability to respond to irrelevant thymus dependent (TD) and thymus independent (TI-2) immunogens and (6) the clonotypic (spectratypic) pattern of B cells in the target tissues.

Our studies reveal that while GF piglets inoculated with SIV can resolve the infection in ~ 21 days, PCV-2 and PRRSV remain persistent infections. All SIV infected piglets had antibodies to the virus as did piglets infected with PRRSV but few PCV-2 infected piglets made anti-viral antibodies. In the case of PRRSV, the anti-viral antibodies may be simply the result of polyclonal B cell activation and hypergammaglobulinemia since there is no difference between immunized and PRRSV-infected piglets in their ability to make antibodies to an irrelevant control antigen, e.g. TNP. This would agree with our previous studies (Lemke et al 2004; Butler et al 2007; 2008). Most notable was the inability of either PCV-2 or PRRSV infected piglets to develop activated cytotoxic or helper T cells at the site of infection; perhaps this is the reason that the viral infections persist. Furthermore in PRRS, B cells are driven to end stage plasma cells which may interfere with their role as APCs and thus the generation of cytotoxic T cells and antigen driven anti-viral antibodies. PCV-2 infected piglets demonstrated a gradual increase in serum Ig levels of all isotypes similar to SIV-infected piglets indicating that PCV-2 does not suppress total B cell activity. However, PCV-2 infected piglets failed to develop antibodies to irrelevant, define immunogens as well as the virus which is consistent with their lack of activated helper T cells. It is our opinion that the defect in PCV-2 infections may be related to the lack of a strong adjuvant effect of the infection. This would suggest further studies using polyvalent vaccines or vaccines given together with strong adjuvants.