Detection of anti-PCV2 maternal antibody and PCV2 dsDNA Replicative Form as relative measures of the anti-PCV2 immune status in piglets.

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Key words
Porcine circovirus, immunosupressive and persisting viruses, maternal immunity

Aim of the study
One main aim of this project was to develop tests for analyzing piglet humoral maternal immunity to understand the relationship between antibody levels, viral load and health status. This was to identify piglets at greater risk for development of PCVD. A second aim was to further characterize the immunosuppressive nature of PCV2 and how this can be modulated. For comparison, we extended these analyses to a second viral pathogen associated with immunosuppression and viral persistence, PRRSV.

Material and methods
We employed serology using an in-house established cell-based ELISA system to analyze a large panel of sera obtained from piglets between 0.5 and 19 weeks. This enabled to relate antibody titer and avidity to disease incidence. The potential immunosuppressive characteristics of PCV2 was related to free non-encapsulated dsDNA detected by gel electrophoresis and characterized using dendritic cell cultures

Results and significance
Our serology work demonstrated that while high titers and avidities at 0.5 weeks related to absence of disease, lower titers and avidities did not always reflect a higher incidence of disease. At 3.5 weeks of age, there was no correlation between antibody titer and disease incidence. Our data and that of others show that maternal antibody ensures resistance to incidence of PCV2-disease early in life, but cannot prevent infection. However, lower titers and avidities do not reflect an immune status, which will always results in susceptibility to PCV2-disease. By 3.5 weeks, the titers are no longer indicative of disease susceptibility, but the avidities continued to reflect animals which would resist. It now becomes evident from our work and that of others that PCV2 persists in sows despite antibody presence. The virus is then transplacentally transmitted to piglets and this cannot be prevented by vaccination. In piglets, PCV2 will also persists despite the presence of humoral immunity, passively or actively acquired. Nevertheless, both sow and piglet vaccination is necessary to control PCV2 viral DNA loads at low levels to avoid PCVD development. Our work on dendritic cells support a role for such high viral DNA loads in disease pathogenesis. In the frame of this project we have also performed comparative experiments with PRRSV, which did not demonstrate such suppressive characteristics.

Publications, posters and presentations (Formatvorlage Überschrift 2)
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Project *BVET Grant 1.10.10.*

Project duration *August 2010 to September 2013*