



O.253

### Maternal immunity towards Porcine circovirus type 2 (PCV2)

Per Wallgren<sup>1</sup> Maria Persson<sup>1</sup> Gunilla Blomqvist<sup>1</sup> Caroline Fossum<sup>2</sup>

1. Dept of Animal Health and Antimicrobial Strategies, Uppsala, Sweden;

2. Swedish University of Agricultural Sciences, Uppsala, Sweden

#### Introduction

Postweaning Multisystemic Wasting Syndrome (PMWS) is associated to porcine circovirus type 2 (PCV2), and as neutralising antibodies prevent pigs from developing PMWS (1) transfer of maternal antibodies to PCV2 may protect the offspring from developing PMWS. We therefore scrutinised the maternal immunity to PCV2 in two herds diagnosed with PCV2, but not with PMWS.

#### Materials and Methods

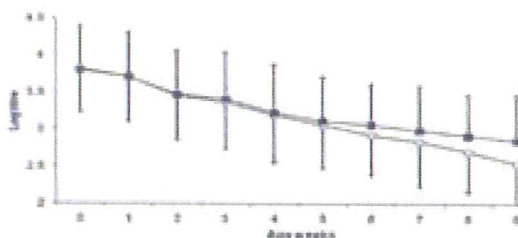
The studies were carried out in a nucleus herd with 180 Yorkshire sows, and in an SPF herd established by caesareans in 1988 with 170 Landrace, Yorkshire and hybrid sows. (2). The SPF herd was infected by PCV2a in 1993, certainly through semen. The herd experienced a reproduction disorders for around six months following the introduction of PCV2 (3), but has stayed healthy since then. PCV2a was demonstrated in both herds prior to initiating the studies. Neither sows nor piglets were vaccinated against PCV2, and PMWS had not been diagnosed in any pig.

Six piglets from 4 litters in each herd were given an identity. Blood was collected from these piglets weekly from birth to 9 weeks of age. Presence of serum antibodies to PCV2 was analysed in two-fold dilutions with an IPMA-technique. The amounts of antibodies are presented as Log<sub>10</sub>-values.

#### Results

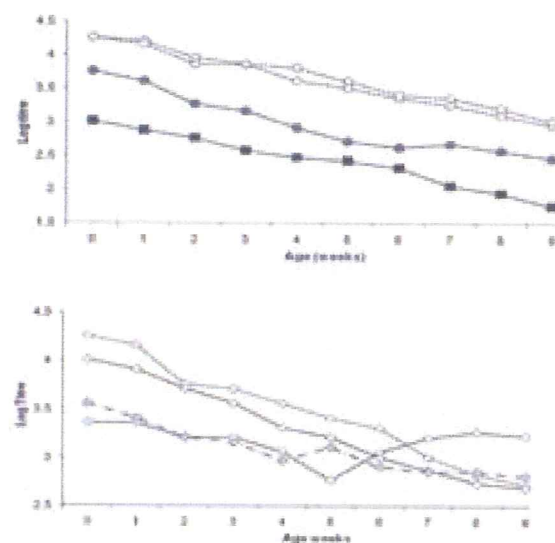
As seen in figure 1, piglets in both herds received maternal antibodies to PCV2 with a mean log<sub>10</sub>-titre of 3.8 ± 0.6. The titres decreased significantly in both herds, but the decrease was more pronounced in the SPF herd from 5 weeks and onwards. Four piglets from two litters in the conventional herd seroconverted at 6, 7, 7 and 9 weeks of age, whereas only one pig in the SPF herd seroconverted to PCV2 at the age of 9 weeks.

**Figure 1.** Mean antibody titres to PCV2 in 24 piglets from 4 litters in an SPF herd (○) and in a conventional herd (■).



The mean antibody titres never differed between the herds, but when the offspring to different sows were compared significant differences between litters were at hand within both herds (Fig 2).

**Figure 2.** Mean antibody titres to PCV2 in piglets from different litters in an SPF herd (upper part) and in a conventional herd (lower part). Breed of the dams; ○ = Landrace, ◇ = Yorkshire, □ = LY-hybrid. Open symbols = 1st parity, grey symbols = 3rd parity and black symbol = 7th parity. Dotted lines = pure bred litter, Full lines = crossbred litter.



#### Discussion and Conclusions

The results showed that all piglets received a maternal transfer of antibodies to PCV2, and that the level of antibodies not was influenced by the rearing system. Nor was any influence of breed indicated since maximal levels of antibodies (Log<sub>10</sub> = 4.3) was measured in offspring to both pure bred Landrace and Yorkshire sows.

In contrast, significant differences in antibody levels were recorded between litters belonging to the same farrowing batch within each herd, and the lowest levels of maternal antibodies was measured in the offspring to the 7th parity sow. In the conventional herd, piglets that received low amounts of antibodies commenced to seroconvert to PCV2 already at 6 weeks of age, indicating an active infection with PCV2. Such piglets may thus increase the PCV2 load concurrently to the decline of maternal immunity in other litters. Therefore sows that transfer a low maternal immunity may jeopardise the health status of a herd because piglets that receive low amounts of antibodies will meet the PCV2-infection at a younger age. The results obtained indicate that old sows may contribute to such risks, and vaccination of old sows may be of particular interest.

#### References

1. Meerts *et al.*, 2005. *Virology* 18:333-341.
2. Wallgren & Vallgård. 1993. *Sw Vet J.* 45:733-735.
3. Wattrang *et al.*, 2002. *Vet Microbiol.* 86:281-293.