PCV2 AND PMWS IN FATTENING HERDS

P. Wallgren1*, I.M. Brunborg2, G. Blomqvist1, B. Lium3, F. Wikstrøm4, A. Jörgensen5,
E. Merlo5-6, L Eliasson-Selling6, C. Fossum5 and C. Monceyron Jonassen2

The National Veterinary Institutes in; 1 Uppsala, Sweden and 2 Oslo, Norway, The Animal Health Services in; 3 Oslo, Norway and
4 Uppsala, Sweden, 5 Div of Immunology, SLU, Uppsala, Sweden, 6 INRA, St Gilles, France
E-mail address: Per.Wallgren@sva.se

Introduction

PMWS mainly affects pigs aged 8-16 week, but so far focus has been on weaners. This study aimed to elucidate the situation in fattening enterprises.

Materials & Methods

PMWS has not been diagnosed in Norway since 2003, while PMWS now is regarded endemic in Sweden. A Swedish fattening herd with PMWS (A) was compared with a healthy herd (B) receiving pigs from the same source. As a true control a recently established Norwegian fattening herd with similar management was included (C). All herds documented their productivity (Table 1).

40 pigs in each herd were randomly selected. Their chest perimeter was measured and blood was collected week 1-5 after arrival. The log 10 level of PCV2-virus per ml serum was measured by a real time PCR (2). The amounts of serum antibodies to PCV2 was analysed in two-fold dilutions by IPMA and presented as log titres (1). At week 5, saliva was collected at 11.00 am in all herds for cortisol analysis.

Table 1. Productivity data. For herd A, the results are presented as mean values pre and post diagnose of PMWS

<table>
<thead>
<tr>
<th>Herd A Healthy</th>
<th>Herd A PMWS</th>
<th>Herd B Healthy</th>
<th>Herd C Healthy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Batches</td>
<td>15</td>
<td>6</td>
<td>15</td>
</tr>
<tr>
<td>Pigs/bat</td>
<td>385±1</td>
<td>383±2</td>
<td>389±17</td>
</tr>
<tr>
<td>Arr-age</td>
<td>88±2</td>
<td>97±3</td>
<td>94±5</td>
</tr>
<tr>
<td>Arr-kg</td>
<td>32.2±3.1</td>
<td>32.3±4.1</td>
<td>31.1±1.8</td>
</tr>
<tr>
<td>Mortal, %</td>
<td>2.0±0.8</td>
<td>2.2±0.9</td>
<td>0.5±0.6</td>
</tr>
<tr>
<td>Conde, g/kg</td>
<td>0.7±0.6</td>
<td>0.2±0.4</td>
<td>0.2±0.2</td>
</tr>
<tr>
<td>DWG, kg</td>
<td>906±28</td>
<td>917±41</td>
<td>886±21</td>
</tr>
</tbody>
</table>

Results

From week 1 to week 5 the chest perimeters increased with 16.1±5.4, 14.3±2.7 and 12.7±1.8 in herd A, B and C, respectively (A and B vs C; p<0.01, A vs B; p=0.07, t-test). In contrast, cortisol levels in saliva was lowest in herd C, 1.06±0.14 ng/ml vs 1.80±0.24 and 1.87±0.19 ng/m (A and B; p<0.05, t-test). However, the level was within the normal range in all herds. Figure 1 shows the mean levels of PCV2 and antibodies to PCV2 in serum.

Two of the 40 pigs in herd A developed PMWS. The diagnose was confirmed by necropsy and preceded by a reduced chest perimeter (from 67 to 58 cm in pig 13 and from 65 to 61 cm in pig 6 during the last week). The viral load in both these pigs increased to log 10 at the sampling the week before diagnose of PMWS. Still both pigs were seronegative to PCV2 in the IPMA test (log titre <1.6) at the time of diagnose.

Figure 1. Mean log levels of PCV2 in serum (left) and log titre of antibodies to PCV2 (right) in herds A (♦), B (□) and C (○).

Discussion

The most remarkable difference between the healthy and the PMWS-affected Swedish herds was the levels of antibodies on arrival. In herd A the levels of antibodies increased during the first four weeks (p<0.001 t-test) in all pigs except the two pigs that developed PMWS. The lower level of ab in herd A on arrival paved the way for an active PCV2 infection indicated by the increasing levels of virus and antibodies. Also the Norwegian pigs were sensitive for PCV2 on arrival, but in the low viral load these pigs in general remained seronegative for at least 5 weeks, thereby being old at seroconversion.

Development of PMWS was only seen in two pigs in herd A - one constantly seronegative and the other with a low and fading antibody titre to PCV2. Increasing levels of PCV2 virus and a dramatic wasting was seen. The other pigs with initially low levels of antibodies to PCV2 seroconverted and did not develop PMWS, despite occasionally high serum levels of virus.

Further, it is notable that the productivity in herd A was basically not affected by PMWS. It is striking that logistics had forced that herd to reduce time between batches to 0 instead of normally 7 days for 6 times prior to PMWS. Although the all in-all out concept was kept, no time for cleaning and disinfection, nor a natural decline in microbial load, was then at hand.

References