Post-weaning multisystemic wasting syndrome (PMWS) in pigs in France: clinical observations from follow-up studies on affected farms

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Abstract

A disease affecting weaned pigs and known as Postweaning Multisystemic Wasting Syndrome (PMWS) is described on French farms. Follow-up studies were designed on cohorts of pigs on a group of 12 severely affected farrow-to-finish operations. Three of them were free of Porcine Reproductive and Respiratory Syndrome Virus (PRRSV) infection. Three thousand and seventy eight pigs were included in the study. Mortality, from weaning to slaughter, was 11% and weeks 11–13 were the most critical. The first clinical sign reported was unthriftiness, then pallor and often fever with associated respiratory or digestive disorders. Wasting could follow rapidly and when clearly established in an individual the prognosis was grave especially when the sick pigs were kept with their penmates. Antibiotics were administered without real efficacy. The disease did not show a collective impact. From preliminary epidemiological investigations, a strong litter effect on disease susceptibility was suspected. PRRS virus was excluded as a major agent. On the other hand, a porcine circovirus (PCV2) was found associated to the lesions. The environment was suspected as an important determining factor for the effect of PMWS in the herds.

Résumé

Les manifestations pathologiques associées au syndrome du dépérissement du porcelet (PMWS) aussi appelé Maladie de l’Amaigrissement du Porcelet (MAP) sont exposées. Pour cela des suivis de cohortes de porcs ont été mis en place dans un groupe de 12 élevages. Il s’agissait d’élevages naisseurs-engraisseurs sévèrement affectés par la maladie. Trois d’entre eux étaient indemnes de SDRP (Syndrome Dysgénésique et Respiratoire Porcin). Trois mille soixante dix huit porcs sont inclus dans l’étude. Le taux de mortalité du sevrage à l’abattage a atteint 11 % et les semaines 11 à 13 se sont avérées être les plus critiques à cet égard. Le premier signe perceptible est la perte de vigueur puis la pâleur et souvent la fièvre. Les manifestations respiratoires (toux, dyspnée) et digestives (diarrhée) sont fréquentes. Il s’en suit un amaigrissement très rapide (2–3 jours). Lorsque l’amaigrissement est clairement exprimé le pronostic est sombre notamment lorsque les animaux

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atteints ne sont pas retirés du groupe. Les médicaments s’avèrent inefficaces. Les lésions aident différents organes: les poumons, la rate, les reins et surtout les ganglions lymphatiques où on observe une sévère dépletion lymphoïde. La maladie ne possède pas d’expression collective. Les premières données épidémiologiques mettent en relief une susceptibilité accrue de certaines portées. Le virus du SDRP est exclu comme agent causal au bénéfice d’un circovirus porcin (PCV2). Les conditions d’environnement offertes aux animaux sont suspectées décisives dans la sévérité de l’impact de la maladie dans les élevages. © 2000 Elsevier Science B.V. All rights reserved.

Keywords: Wasting syndrome; Pig; Postweaning; Epidemiology; PMWS

1. Introduction

Late in Spring 1996, two farmers complained about unexpected mortality occurring in growing pigs in Brittany (France). The pigs were two to three months old and no obvious other health disturbance could be detected in the farms. In a preliminary study, piglets showing the first clinical signs of wasting were transferred to our experimental facilities and SPF pigs of the same age were placed in contact. Surprisingly, the wasting piglets progressively recovered without any medication, whilst the contact SPF pigs became severely ill. They developed typical Glasser’s disease. At that time the situation was rather confusing. Porcine Respiratory and Reproductive Syndrome (PRRS) was detected on those farms affected by the piglet wasting problem as well as several other enzootic agents including M. hyopneumoniae, P. multocida, H. parasuis, S. suis and enterotoxigenic E. coli. During the second half of 1996 we tried to determine the presence or absence of the specific new syndrome. Field case observations, often associated with experimental trials, were designed. By the end of 1996 we came to the conclusion that since the condition could be transmitted to naive pigs in our facilities (Le Cann et al., 1998), it was infectious and transmissible. Finally, the syndrome (clinical signs, macroscopic lesions, histopathology) was recognised as being similar to the descriptions made in North America and called “Postweaning Multisystemic Wasting Syndrome” (Harding, 1996, Clark, 1996). Since then, an extensive research programme has been performed. The present paper reports a series of field surveys and laboratory investigations.

2. Material and methods

2.1. The farms

Farms were selected by field veterinarians. The latter, in turn, had been called to these farms because of high and persistent mortality in weaned pigs despite medication. The authors visited the farms and their selection was based on the willingness of the farmer to co-operate and also on the availability of information. Twelve farms in the western part of France working with six different farm organisations (co-operatives) were included in the survey. They were all farrow-to-finish operations like the majority of farms in the area. On 11 farms the replacement gilts came from a multiplier. On the other the replacement stock was home-produced. Artificial insemination from bought-in specialised AI centres was widely used. The average herd size was 343 sows (range 105–600). Eleven farms had intensive confined production (slatted floor, fan ventilated rooms) and on one (Farm 2, Table 1) the sows were kept outdoors. The productivity of the herds was good (24.4 piglets weaned/sow/year). Age at weaning was either 26–28 days (n = 7 herds) or 21 days on average (n = 5 herds). They were free of Pseudorabies but 9 of them were PRRS positive. Herds No. 2, 8 and 10 were PRRS free.

2.2. The protocol on the farms

The protocol applied across the 12 farms was designed to achieve two objectives. Firstly to characterise PMWS which was new to France. No serological test was available so we focused on other
Table 1
Postweaning Multisystemic Wasting Syndrome: impact in 12 farms in France

<table>
<thead>
<tr>
<th>Farm</th>
<th>Size(^a) (Nb sows)</th>
<th>Productivity (piglets weaned/year)</th>
<th>Age at weaning (days)</th>
<th>% mortality(^b) before wasting disease 3 month period after wasting disease occurred</th>
<th>Predominant overall clinical signs associated with wasting(^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>500</td>
<td>24</td>
<td>27.3</td>
<td>6.5</td>
<td>Diarrhoea + respiratory disorders</td>
</tr>
<tr>
<td>2</td>
<td>120</td>
<td>26.5</td>
<td>27.2</td>
<td>4</td>
<td>Diarrhoea + respiratory disorders</td>
</tr>
<tr>
<td>3</td>
<td>250</td>
<td>3.8</td>
<td>26.8</td>
<td>4.3</td>
<td>Respiratory</td>
</tr>
<tr>
<td>4</td>
<td>600</td>
<td>26.2</td>
<td>20.3</td>
<td>4.8</td>
<td>Not clear</td>
</tr>
<tr>
<td>5</td>
<td>250</td>
<td>24.3</td>
<td>21</td>
<td>5.1</td>
<td>Diarrhoea + respiratory</td>
</tr>
<tr>
<td>6</td>
<td>360</td>
<td>23.5</td>
<td>26.9</td>
<td>4.7</td>
<td>Diarrhoea + respiratory</td>
</tr>
<tr>
<td>7</td>
<td>300</td>
<td>24.3</td>
<td>28</td>
<td>3.8</td>
<td>Respiratory</td>
</tr>
<tr>
<td>8</td>
<td>105</td>
<td>24.8</td>
<td>26.9</td>
<td>3.7</td>
<td>Diarrhoea + respiratory</td>
</tr>
<tr>
<td>9</td>
<td>500</td>
<td>23.5</td>
<td>21</td>
<td>5.5</td>
<td>Diarrhoea + respiratory</td>
</tr>
<tr>
<td>10</td>
<td>220</td>
<td>24.5</td>
<td>27</td>
<td>3.2</td>
<td>Respiratory</td>
</tr>
<tr>
<td>11</td>
<td>600</td>
<td>25.2</td>
<td>21.1</td>
<td>5.6</td>
<td>Diarrhoea + respiratory</td>
</tr>
<tr>
<td>12</td>
<td>320</td>
<td>24.2</td>
<td>27</td>
<td>3.9</td>
<td>Not clear</td>
</tr>
<tr>
<td>TOTAL</td>
<td>343</td>
<td>24.4</td>
<td>24.5</td>
<td>4.6</td>
<td>14.6</td>
</tr>
</tbody>
</table>

\(^a\) Farrow-to-finish farms.
\(^b\) Mortality (for any reason) from weaning to slaughter.
\(^c\) Individual signs: unthriftiness, pallor, etc. . . . were present but not indicated here
Histopathology was undertaken with special emphasis on lymphoid tissues associated with lung and intestine. Viral inclusions were looked for following the protocol of Ellis et al. (1998).

Lastly, an in situ hybridisation specific for circovirus Type II, was performed in cases where there was microscopic damage.

3. Results

3.1. Impact of PMWS in the herds

The first warning signs usually came post-weaning when the pigs were around two months old. They were weaned at four to five weeks but in most cases, the critical phase for disease occurrence ended at around three months of age (13 weeks). Clear signs were still observed after the third month especially when the disorders occurred in week twelve. In a given batch of pigs only some individuals clearly showed clinical signs (i.e. low morbidity). A wide range of drugs was used without any significant positive effect. There was no detrimental effect on reproductive function. Prolificacy and farrowing rates were maintained at the previous level. Abortions, stillbirth or mummifications were not noticed. Muscle wasting in the piglets was in every case the major and most typical sign, but was associated with other manifestations (Table 1). The mortality rate was rather high, both post-weaning and in the finishing stage and depended on the age at which the pigs were moved to the finishing house. On these severely affected farms, mortality level reached 14.6% on average for the three months preceding our intervention. The corresponding figures before PMWS onset are also shown in Table 1. In every case, when PMWS signs appeared, a sudden increase in mortality was noticed by the farmers and confirmed by the recordings. In a given herd, considerable differences could be seen in mortality impact between the subsequent batches. However, a baseline was observed. This was around 10–15% (from weaning until slaughter) for the majority of the farms (8 farms), but it was closer to 20% for four farms. On three of the farms the problem remained acute for nearly two years (farms 1, 2 and 3).

3.2. The overall results of the cohort studies

The total number of pigs considered was 3078. Three hundred and thirty eight (11%) died or were sacrificed principally for animal welfare reasons. Fig. 1 shows the mortality according to their age. The twelfth week was the most critical and 68% of the losses occurred during weeks 11–13. Signs of illness were noticed in all the piglets before death. When recorded, rectal temperature was found to be 40.5°C or above, on at least one day in all these fatal cases. The first sign reported to us by the farmers was unthriftiness, then most of the pigs became pale and their flanks looked empty since their feed intake had been reduced. This was confirmed later by opening the stomach at necropsy. Wasting was established rapidly and the back bone soon became apparent. Cough and diarrhoea were frequent but not always found. Dermatitic skin lesions especially on the rear of the body were noticed in the cohorts on six farms ($n = 15$ pigs) and transient cyanosis (especially on the ears, and in the perineal area) on four farms ($n = 16$ pigs). Icterus was detected in only six pigs.

When the wasting process was clearly visible, despite a wide range of treatments, the prognosis was grave. When these pigs were removed from the pens and placed in hospital accommodation, about half of them survived, but only half later showed a correct weight gain. One hundred and eight pigs from 12 farms were sent to the laboratory. They were 81 days old on average ($\pm 12$ days). Table 2 gives the results of necropsy. Most of the pigs were in poor body condition when submitted to the laboratory. It is remarkable that a high percentage of the pigs were affected by pneumonia (67.5%). The lesions were often of severe bronchopneumonia and extended to all lung lobes. A high proportion (53%) of pigs showed a disturbance of the large bowel (oedema, dilatation). The lymph nodes were often enlarged but those affected differed from pig to pig. Lymphoid depletion in the lymph nodes and Peyer’s Patches was also a common finding but was not the general rule. Eleven pigs with dermatitic skin lesions type were sent to the laboratory and kidney lesions were mainly found in these pigs. Bacteriology was not systematically performed since the largest number of the pigs had received antibiotics over the days before
necropsy. A variety of infectious agents were isolated from lesions in the viscera and from the tonsils. *Haemophilus parasuis*, *Pasteurella multocida* and *Streptococcus suis* were commonly found (50, 41 and 10% of the pigs respectively).

3.3. The results of the detailed on-farm clinical observation (farms 5, 6 and 7)

On these three farms, there were no obvious signs of disease during lactation. Average weaning weight was 6.2 kg, 7.5 kg and 8.2 kg for herds 5, 6 and 7 respectively. The pigs were weaned at 21 days on farm 5; and at 27 and 28 days on average respectively in herds 6 and 7. Average litter size at weaning was 11, 10.2 and 9.5 for the three cohorts. During the first month post-weaning, things went on normally and only some mild diarrhoea was noticed during the second week post-weaning. Growth rate remained moderate. Average liveweight at 8 weeks of age was 18.8 kg (±3.7), 19 kg (±3.9) and 19.7 kg (±3.5) for the three cohorts respectively.

3.3.1. Farm 5 cohort

Two hundred and ten pigs (19 litters) were studied. Some pigs could be heard coughing after weaning (on week 6 and 7) without any apparent detrimental consequences. At the end of the 10th week of age and during the 11th, several pigs became progressively hyperthermic, and 46% of the pigs reached ≥40.5°C at least once, most of them without any sign of depression. A proportion of the pigs looked pale (12%) and diarrhoea was seen in 13% of the pigs. Respiratory signs (cough and sneezing) were also detected. Different treatments were administered especially antibiotics (tiamuline and/or colistine and neomycine depending on the individuals). Anti-inflammatory compounds (Dexamethasone) were also injected to affected pigs without real success. Twenty three piglets (11%) died between weeks 10 and 13 (2, 7, 10 and 4 respectively for the 4 weeks). Average weight at the end of week 12 was 35 kg (±7 kg). One week later, at the end of week 13 the group of pigs looked unequal with numerous pigs in poor condition. They had to
Table 2
External aspect and lesions observed in a sample of piglets taken on farms severely affected by PMWS. 
$n=108$ piglets from 12 farms, average age at necropsy: 81 days (SD±12)

<table>
<thead>
<tr>
<th>External aspect</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pale</td>
<td>72</td>
<td>66</td>
</tr>
<tr>
<td>Emaciated (more or less)</td>
<td>88</td>
<td>81</td>
</tr>
<tr>
<td>icteric</td>
<td>2</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Skin lesions:

- Of dermatitis type: papules or red-to-purple plaques, becoming crusty: 11 (10.1%)
- Cyanosis (ears, Perineal area): 3 (2.7%)

Gross lesions:

- Enlarged lymph nodes:
  - Inguinal nodes: 28 (25.9%)
  - Mesenteric nodes: 36 (33.3%)
  - Broncho-tracheal: 25 (23.1%)
  - Generalised: 13 (12%)
  - Necrotic, atrophic lymph nodes (mesenteric): 2 (1.8%)
- Pneumonia: 73 (67.5%)
- Lang interlobular oedema: 12 (11.1%)
- Pleuresy: 19 (17.5%)
- Pericarditis: 13 (12%)
- Stomach ulcer: 33 (30.5%)
- Oedema/colon: 20 (18.5%)
- Caecum: dilatation, inflamed mucosa, diarrhoeic content: 57 (53.2%)
- Peritonitis: 9 (8.3%)
- Spleen (enlarged, «meaty»): 15 (13.8%)
- Kidney (enlarged and/or focal discoloration): 15 (13.8%)

Histopathology:

- Lymphoedema depletion (lymph nodes, Peyer patches): 76 (70.3%)
- Intertitial pneumonia: 83 (76.9%)
- Multinucleated giant cells: 65 (60%)
- Hystiocytic infiltration: 60 (55.5%)
- Intracytoplasmatic viral inclusion bodies: 62 (57.4%)

be removed from the room and by adding the cull piglets to the wasting ones total losses reached 15.7%.

It became obvious that some of the litters were more affected than others. (For example seven pigs that died were from the same litter of 11 weaned piglets).

3.3.2. Farm 6 cohort

Three hundred and two pigs (30 litters) were studied. Until the 10th week of age there was no obvious sign of disease. By the end of the 10th week, one pig showed fever and unthriftiness and during week 11, pigs with fever became progressively more numerous (up to 41°C was recorded). The pigs exhibited diarrhoea (12%) at clinical examination in the middle of week 11 and treatment was rapidly prescribed (Colistine + Neomycine, oral route).

A recrudescence of coughing was also observed and patent signs of wasting became visible soon in the first sick pigs. During week 12, dermatitic skin lesions were present in three pigs and cyanosis in two other individuals. Dermatitis was first seen as papules, then as erythematous and crusty lesions (1 to 3 cm diameter) located first in the perineal area and later covering the whole rear part of the body including the legs. Cyanosis, when it occurred, lasted about 2 days and was located in the perineal area but
also on the ears. When cyanosis occurred the pigs had already started to decline, coughed and had been pyrexic (≥ 41°C) for two days. One pig became icteric in week 12 after starting to lose weight. Fig. 2 shows the clinical pattern of four selected pigs raised in the same room. At the laboratory, typical lesions of PMWS were observed in the pigs. Table 3 gives their liveweight at weaning, at 8 and at 12 weeks. Pig D was still apparently healthy at the end of week 12. At that time, average live weight of the cohort was 34.7 kg (±7 kg). Wasting stopped after week 13. Within the cohort 21 pigs had died by week 14 and 7 pigs were in a bad physical state (total: 9.3%).

3.3.3. Farm 7 cohort

Two hundred and fifty nine pigs (24 litters) were studied. The first clinical sign was pyrexia associated with depression in 5 pigs during week 10, and the proportion increased (15%) with time. The same week one pig showed typical lesions of skin dermatitis and a rectal temperature of 41°C. The pig died 3 days later. The pigs that showed the first high rectal temperatures were spread over different pens in the room but they were littermates of four sows (out of the 24 sows studied). Respiratory signs were also common. Three pigs died, 2 with signs of wasting. They became pale, dyspnoeic and died within 3 days. In the remaining pigs, external signs of wasting were obvious in 12 pigs by the end of week 12. Amongst these, 10 died or were euthanased later (total loss of 13 pigs, 5%). Average liveweight at 12 weeks was 37 kg (±6.5 kg).

<table>
<thead>
<tr>
<th>No. pigs</th>
<th>Week 4</th>
<th>Week 8</th>
<th>Week 12</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>4.6</td>
<td>13.2</td>
<td>15*</td>
</tr>
<tr>
<td>B</td>
<td>9.5</td>
<td>23.6</td>
<td>28.2</td>
</tr>
<tr>
<td>C</td>
<td>7.8</td>
<td>19.4</td>
<td>25.4</td>
</tr>
<tr>
<td>D</td>
<td>7.7</td>
<td>22</td>
<td>37</td>
</tr>
</tbody>
</table>

Group average (n = 302 pigs)  
7.5 19.7 34.7

* Week 3 at weaning for this pig (A) and the corresponding litter; weight later on at week 7 and 11.

† At death.

Fig. 2. On-farm clinical observations made on contemporary pigs (farm 6). Number of pigs: A, ○ --- ○; B, ○ — — ○; C, ♦ — — ♦; D, × — — ×.
4. Preliminary epidemiology

4.1. The role of the sow (or «litter-effect»)

As had already been mentioned, some litters seemed to react more severely. On the farms where cross-fostering was limited during the suckling phase and where it could be properly recorded, the litter effect was investigated (Table 4). Whilst 42% of the litters were not at all affected, 16% accounted for 54% of the losses. The figures obtained were significantly different (P < 0.001) from the theoretical distributions especially the Poisson distribution. On the other hand, no clear relationship could be found with the parity of the sow.

4.2. The role of PRRS

Three of the 12 farms (No. 2, 8 and 10) were seronegative for PRRS and had never experienced infection (no clinical signs were observed and regular serology remained negative over the years). On these farms the clinical picture of PMWS was not different and the level of mortality was also very similar to that of the seropositive farms (Table 1).

In the detailed on-farm investigations carried out in farms 5, 6 and 7 all the piglets except six in farm 7 were seronegative to PRRS at 12 weeks of age. Fever, cough and cyanosis were observed starting in week 10, the illness culminating during weeks 11 and 12 after transfer to the finishing pens. The time lapse between the PRRS-like troubles and blood sampling for serology was probably too short to demonstrate seroconversion. By week 16 all the pigs had seroconverted showing clear evidence of PRRS virus infection on the farms. However, only some of the sows which gave birth to the piglets were PRRS seropositive at weaning time (36 sows out of 75, 48%). The litters of PRRS seronegative sows tended to be more predisposed to exhibiting signs especially fever than those of PRRS seropositive sows. On the other hand no difference could be seen regarding wasting and mortality.

The PRRS virus was found in only one pig (Pig B, farm 6) but the results strongly suggested circulating PRRS virus in the growing pigs during the survey on the 3 farms.

In conclusion, from our observations on all 12 farms, the real impact of PRRS infection on PMWS could not be established but was estimated to be low.

4.3. The role of environment

In the cohorts which were followed, mortality was reduced (11%) compared to the previous situation of the farms (14.6%). Part of the explanation was found in the changes in the environment offered to the pigs especially on four farms (No. 3, 4, 7 and 9). The technical conditions of production were investigated and in agreement with the farmers, husbandry was modified. The changes were directed at reduction of infection pressure in the herds through better hygiene and management. Over-crowding and mixing were reduced and the cleaning strategy was improved. An all-in/all-out hygiene policy system (one batch of contemporary pigs per room) was strictly applied and other measures including emptying the pits below the slatted floors introduced on those farms. Whereas wasting was still observed, mortality from weaning to slaughter was considerably reduced (from 12% or more to 6% or less for farms 3, 4, 7 and 9).

In herd 9, three batches of pigs, each born one week apart, were observed (total = 350 pigs, 123 litters). In each batch, half of the randomly selected pigs were weaned at 12 days of age and housed in appropriate accommodation off the farm. The remaining litters were weaned at 21 days with strict hygiene and husbandry. From weaning to 100 days of age, mortality was limited to less than 1% for all pigs. After the trial the farmer tried to continue the strict husbandry. Mortality from wasting was dramatically reduced before 100 days of age. Un-

### Table 4

<table>
<thead>
<tr>
<th>No. litters</th>
<th>No. mortality per litter (dead/sacrificed)</th>
<th>Total losses</th>
</tr>
</thead>
<tbody>
<tr>
<td>93</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>45</td>
<td>1</td>
<td>45</td>
</tr>
<tr>
<td>26</td>
<td>2</td>
<td>52</td>
</tr>
<tr>
<td>19</td>
<td>3</td>
<td>57</td>
</tr>
<tr>
<td>16</td>
<td>4</td>
<td>64</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>50</td>
</tr>
<tr>
<td>8</td>
<td>6</td>
<td>48</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
<td>21</td>
</tr>
</tbody>
</table>
Fig. 3. Mortality of pigs (subsequent batches of contemporary animals, one week between each batch). Farm 9: farrow-to-finish unit; ◯, mortality weaning ± 100 days of age; ■, mortality after 100 days of age.

Fortunately after remission in 4 subsequent batches the troubles re-occurred during the finishing phase, at 15–17 weeks of age. Mortality (wasting mainly but also dermatitis-nephropathy) was 9% on average and persisted for 5 months (from 8 to 12% mortality, Fig. 3).

After 5 months, the problem recurred at an earlier age (9–10th week). A failure to adhere to the previous hygiene recommendations was clearly observed.

5. Discussion

Post-Weaning Multisystemic Wasting Syndrome (PMWS), a new condition affecting the pig was reported first in Canada (Clark, 1996; Harding, 1996). Since then it has been recognised in several other countries (Daft et al., 1996; Segales et al., 1997; Le Cann et al., 1997; Kennedy et al., 1998; Kiupel et al., 1998). The descriptions highlight the fact that the post-weaning/growing period is the most risky regarding PMWS expression. However published on-farm observations remain rather general.

The present work which focused on follow-up studies on severely affected farms (some for several months), describes detailed clinical observations. Our results show a real critical phase for disease occurrence. On our French farms, it starts at around eight weeks of age and ends at about 13 weeks. Obviously, signs of the disorders are still clear after this period especially the weight heterogeneity. Under Canadian conditions, Harding and Clark (1997) observed an earlier occurrence (5–6 weeks). The average mortality prevalence registered on the 12 farms from weaning to slaughter was 14.6% for the 3 months preceding our first visit. This figure, although it includes all the losses for any reason, is high. It is higher than the levels already published (Harding and Clark, 1997). These authors only described the post-weaning phase and there were limited losses associated with PMWS. On our farms, by comparing overall mortality levels after and before PMWS occurrence, the losses attributable to the disease were estimated to be approximately 10%, which is closer
to the data published. Another source of variation may have come from the farm status with respect to potential pathogens. The reports from Canada dealt with farms known to be of high health level.

The clinical signs we have seen are similar to those reported earlier in the different countries (Harding and Clark, 1997; Segales et al., 1997). However icterus was uncommon on our affected farms. Our laboratory findings are also in general accordance with the literature. Lung consolidation was common and histopathology showed interstitial pneumonia in a high percentage of the pigs. In the abdominal cavity, the caecum and colon were the most often affected. The caecum was often dilated and had a diarrhoeic content. The lymph nodes were enlarged and a severe lymphoid depletion was observed. In the lymph nodes and Peyer’s patches, viral inclusion bodies were present. On the other hand, as suggested by Clark (1997) and Segales et al. (1998) the question of whether skin lesions and especially those of dermatitis type (Hélie et al., 1995) are part of the PMWS or not, cannot be answered. Such lesions are not regularly observed. The PMWS-associated circovirus might enhance different infections. The PRRS virus was suggested to be involved (Thibault et al., 1998) but a priori, our findings can hardly support this hypothesis, since dermatitic skin lesions were also observed, though at a low prevalence, on PRRS-free farms. It was surprising to notice that the disease expression was centred around the period eight to thirteen weeks of age. Even at the beginning of the PMWS outbreak, no impact was detected on reproductive parameters. Sow productivity was and remained good-to-excellent (according to the French ITP recording scheme covering about 5000 farms, ITP, 1997). Up to weaning no health disturbances could be seen in the piglets. It is uncommon to have such a disease, strongly believed to be infectious (Ellis et al., 1998; Kiupel et al., 1998; Allan et al., 1998), and capable of killing 10% of growing pigs, without any detrimental consequences in other categories of pigs.

The literature on PMWS tends to describe an intermittent or epidemic picture of PMWS (Kiupel et al., 1998; Harding and Clark, 1998). On our group of 12 farms, three experienced problems at a severe level for about 2 years, showing the persistence of the disease under certain circumstances.

From follow-up studies of individual pigs, we learned that wasting could occur in heavy pigs at weaning as well as in light pigs for a given age. On the other hand, a strong “litter effect” was observed, some litters being decimated whilst others did not show adverse effects. No obvious parameter has been found to have a predictive value for the phenomenon. However it can be suggested that the sow plays a key role, which may be the infection load transferred to the piglets since the sow is supposed to be the reservoir for circovirus and the other pathogens found on conventional commercial farms. On the other hand the protection level present (natural constitution, acquired protection from suckling etc.) may be important. The interaction of these antagonistic forces might influence the vulnerability of the littermates in PMWS expression. However, this reasoning is limited by the strong individual (nearly binary or bimodal) expression of the disease. Certain pigs (the large majority) did not exhibit any wasting illness whereas others were severely affected. Since the exposure level to the infectious agents is “a priori” identical for littermates, only individual resistance capability may be involved. Several years ago it was reported that there was in the pig an important inter-individual variability of response to antigenic challenge (Buschman et al., 1974).

From our study, a causative role of PRRS virus in PMWS expression can be excluded. At the best, under particular circumstances, it can be a complicating factor. Confusion comes from the frequent concomitant PRRS virus activity in eight to thirteen week-old pigs. Severe PMWS sequelae were recorded on three of the 12 farms which were PRRS seronegative and that had never experienced the disease (PMWS persisted 2 years in Farm 2).

In conclusion, our observations favour an important influence of environment on the overall severity of PMWS on the farms. Our proposals were based on 2 main points. The first, from our experimental studies, suggests that a certain infection pressure is necessary for the disease to be fully expressed and therefore for wasting to occur. Secondly, the immune system was also involved. Proposals were made to reduce the microbial load through technical means by better hygiene, segregation of batches, better ventilation and reduced over-
crowding. In addition, an effort was directed at reduction in mixing. This is because the higher risk of pathogen transfer. On the other hand, mixing of unacquainted litters may lead to intensive fighting. This might interfere with lymphocyte blastogenesis (Deguchi and Akuzawa, 1998).

The preliminary response in four farms has looked promising despite wasting signs remaining visible (Madec et al., 1999). These investigations are to be continued. In this task, veterinary practitioners and other professionals will be greatly helped when new relevant diagnostic tools and, in particular serology become available.

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