# European experience with post-weaning multi-systemic wasting syndrome (PMWS) and other PCV2-associated diseases: an update

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## Introduction

Walters and Mackinnon [1], at the last Pig Genetics Workshop, described and discussed several aspects of Post-weaning multi-systemic wasting syndrome (PMWS) and Porcine dermatitis and nephropathy syndrome (PDNS). Since then the diseases have assumed major political importance in Australia as the pig industry has challenged the federal government intention to import pigmeat. Rather than comment on this situation, this brief report aims to update on 'technical' developments in Europe over the past eighteen months.

## Pathological Background to PMWS and PCV2

There is now a large amount of evidence to confirm that infection with porcine circovirus type 2 (PCV2) is absolutely necessary for the expression of PMWS [2,3,4]. The virus is also associated with PDNS, Porcine reproductive disease complex (PRDC) and reproductive failure [5]. However, as PCV2 is extremely widespread in pig populations [6,7,8,9,10] it is not known why PMWS, PDNS and PRDC have emerged as major problems in recent years.

Because experimental infection with PCV2 alone in pigs is not sufficient to cause PMWS [11,12] and because not all pigs in affected herds develop PMWS, it is known that factors in addition to PCV-2 are necessary for the expression of disease.

Experimentally, these have been shown to include immune modulation [13,14] as well as co-infection with porcine reproductive and respiratory syndrome virus (PRRSV) [15,16], porcine parvovirus (PPV) [16,17,18], swine influenza virus (SIV), *Mycoplasma hyopneumoniae* and a variety of other bacterial pathogens, notably *Streptococcus suis* and *Pasteurella multocida* [19,20]. However, an examination of recent European cases of PMWS showed that PRRSV co-infection, although common, is not an essential component of PMWS [18]. What is emerging is the fact that co-infection with PPV and/or PRRSV apparently results in increased PCV2 replication and enhances PMWS infection [21,22,23].

Aside from classical PMWS, the most common clinical syndrome with which PCV2 has been associated in the field is respiratory disease. Indeed, in the United States, data indicate that PCV2-associated respiratory disease is a more common clinical outcome of PCV2 infection than is PMWS [24].

## Other co-factors

A range of other co-factors have been reported, including management practices, genetics, sow effects and litter factors. Some recent research reports from Europe are highlighted below.

### 1. Management practices

In France, studies on the impact of PMWS in severely affected farms have identified husbandry practices which might have an important effect on the clinical expression of the disease [25]. The key factors are breeding management, vaccination of the breeding herd, general hygiene and the rearing conditions in the farrowing and weaning herds.

#### *Breeding management*

PCV2 has been reported to be present in semen [26]. In France, clinical PMWS was less likely to occur in farms purchasing semen than in farms with on-farm collection [25]. It was postulated that on-farm collection would result in circulation of PCV2 within the herd. Also, farm boars are often housed together with the pregnant sows, allowing a greater possibility of cross-contamination.

Similarly, it was observed that herds which were 'closed' to the importation of replacement gilts had a lower incidence of PMWS. This is hardly surprising as evidence [5] shows that PCV2 infection occurs horizontally (pig to pig).

Finally, in farms where pregnant sows were housed together in groups, the impact of PMWS was found to be lower [25]. The latter findings suggest that a better homogeneity of immune status in the sow herd may be protective against PMWS.

Recent studies in Denmark have looked at the possible role of Vitamin E (alpha-tocopherol) as it is important for the normal function of the immune system. In one study on three affected farms [27] there was a marginal significant difference (P=0.07) in the drop in Vitamin E between pigs dying and pigs surviving the PMWS critical period post-weaning. However, comparison sampling at weaning showed a trend (P=0.08) of increasing mortality with decreasing levels of the vitamin. As a result it was suggested [28] that an extra supply of Vitamin E to suckling pigs might be one way to reduce PMWS losses in weaners. This could be achieved by increasing the level in sow lactation diets or by injecting piglets with Vitamin E.

#### *Herd Vaccination*

Vaccination, via immune stimulation, has been suspected as a possible triggering factor for PMWS [29]. For example, the influence of *Mycoplasma* vaccines on the occurrence of clinical PMWS has been reported by several authors [30,31]. However, a recent UK study [32] showed that vaccination of commercial pigs against *Mycoplasma hyopneumoniae* between one and three weeks of age produced no significant change in the incidence or severity of

PMWS. Similarly, results obtained in France in an epidemiological cross-sectional survey [25] were inconclusive. At farm level, clinical PMWS can precede the introduction of vaccination, it can occur on farms that have not employed vaccination of piglets and it has not emerged on some farms that do use vaccination. However, in another French study carried out in PMWS affected farms [33] there was some evidence of a possible link between Parvovirus/Erysipelas vaccination schemes and PMWS. Specifically, piglets born to sows exhibiting very high PPV antibody titres when pregnant, were more likely to further develop PMWS clinical disease. The cause for these high antibody titres could be linked to PPV circulation, thus further supporting the need for good vaccination practice in the breeding herd [34]. Sows with neck injuries as a result of improperly performed injections were also more likely to give birth to piglets that eventually developed clinical PMWS. Those results indeed suggest a possible connection between some practices and infectious co-factors.

***Importance of the general hygiene and rearing conditions in farrowing and weaning herd***

Several husbandry measures have been identified that can help control and mitigate the impact of the PMWS [1,25,35]. These include good hygiene practices in the farrowing and weaning facilities (assessed by all-in/all-out procedures, good cleaning/disinfection routines and appropriate duration of pig-empty periods); raised weaning age (minimum 28 days), minimisation of cross-fostering and maximisation of maternal immunity; elimination of mixing of cohorts and constant stability of pig throughput and comprehensive control of parasites and rodents.

**2. Genetic, maternal and litter effects**

The immune system plays a major role in PMWS [36,37] as seen from the dramatic lesions observed within the lymph organs of affected pigs. There is some evidence [38] that selection for immune response parameters may be possible so this implies that genetic resistance to PMWS may be important. Indeed, there have been rumours in the global industry that pigs from some breeding companies and from some breeds have been more susceptible to PMWS. For example, in France, it was reported that pigs derived from the Pietrain breed had a lower susceptibility to develop PMWS than pigs derived from either Large Whites or Landrace [34]. To test this theory, a cohort study [33] was carried out in four PMWS affected farms using random semen from Pietrain and non-Pietrain boars. No effect of genotype was identified.

In the UK, data on piglet mortality were collected over two years on a 360 sow unit with PMWS [39]. Overall mortality was 11.9% with mortality due to PMWS at 8.8%. The key findings were:

1. 54% of litters showed at least one PMWS death and the average mortality within those litters was 18%.

2. Parity and farrowing batch significantly affected PMWS mortality with mortality lower in parities 1 and 2

3. Piglet sex and litter size did not affect mortality

4. Sows whose litters experienced PMWS mortality in one parity have both a higher probability of PMWS mortality in the next litter, as well as higher overall mortality.

The genetics and maternal effects were also studied with the following findings:

1. The heritability of piglet survival was non-significant, indicating that genetic effects were minimal.

2. However, there was a significant maternal effect influencing piglet survival

3. There was a small but significant maternal effect on age at death

Sow and litter effects have been reported in several countries. In France [40] it was found that the PPV status of sows can change between each farrowing cycle because of interactions between vaccination and natural infection. Thus, susceptibility of individual sows to PPV and (possibly to PCV2) could vary from one cycle to the next. Similarly, the PCV2 immune status of the sow at farrowing time and the related maternal antibodies can alter dramatically. For example, a small proportion of sows (5%) test seronegative for PCV2 at farrowing time. Thus, the piglets derived from these sows do not receive a high level of specific antibodies *via* colostrum intake and they would be more likely to get infected earlier than those receiving good passive immunity. As a result, all non-infectious factors which decrease colostrum intake could increase the risk of clinical PMWS occurrence. According to this assumption [34], large litter size and/or a high level of cross-fostering might be detrimental, while early removal of heavy piglets in large litters to make place for the weaker ones, artificial feeding with colostrum and oxcytocin injections to promote colostrum production might have a positive effect

## Conclusions

Despite the fact that significant knowledge is now available regarding PMWS- and PCV2-

associated diseases there are many questions that remain unanswered. Of vital importance is the search for an understanding of the interactions between all the possible significant co-factors, both infectious and non-infectious. Furthermore, there remain many aspects of PCV2 infection and PMWS that are very difficult to explain [5]:

- Why did PMWS occur in many countries at almost the same time?

- Why did PMWS outbreaks occur in the late 1990's when PCV2 had been present for several years previously?

- How have outbreaks occurred in remote 'closed' units with no stock introductions and excellent biosecurity?

- What protects a significant proportion of an infected herd?

- Why does PMWS have such devastating long-term effects on herd productivity when acquisition of immunity might be expected?

Clearly, much work is still needed in order to conquer PMWS and PCV2-associated diseases.

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