Introduction

Porcine circovirus type 2 (PCV2) is thought to be the cause of PMWS (postweaning multisystemic wasting syndrome), a condition affecting mainly pigs between 6 and 18 weeks of age. It is usually characterized by pigs that are wasting, do not respond well to conventional treatments and the mortality in those that get sick is high. Other clinical signs like thumping and diarrhea vary in severity from one case to another.

My interest for PCV2 has been significantly increased over the last two years because the situation in my home province has changed quite dramatically. Up to 2004 PMWS was considered as a relatively sporadic disease in Quebec. This would be by itself, since it was regularly found to be associated with PRRS in nursery and finishing unit problems. However starting at the end of 2004, and particularly since the beginning of 2005, the number of cases where this condition was diagnosed and considered to be of primary importance has greatly increased. The same can be said about Ontario and North Carolina, and it seems that the condition is now observed in an increasing number of farms and states in the US. In this paper, I will try to shed some light on the possible reasons why this may have occurred, on what can be done to control the losses and on conditions, other than PMWS, that could be associated with the organism.

What causes PMWS/PCVD/PCVAD?

A few words should probably be said first about nomenclature, since it has become a bit more complicated lately. The acronym PMWS is now gradually changed to either PCVD, for porcine circovirus disease, in Europe, or to PCVAD, for porcine circovirus associated disease, in North America. The switch from PMWS to these names is probably due mainly to three reasons. First wasting can be caused by many organisms other than PCV2. Secondly PCV2 has been associated to problems in pigs other than PMWS and thirdly, possibly, the word wasting might have a negative impact on public perceptions of the swine industry, and of the safety of the meat it produces. As for the slight difference between the two new names chosen in Europe and North America, it could be due to variations in the relative importance that scientists on each side of the ocean believe PCV2 plays in the condition.

In fact looking at the cause of PMWS is a good place to start. Two main positions are presently debated. There are those who believe that PCV2 is the cause of PMWS, although other factors or agents may contribute significantly to the losses associated with it in the field, and those who believe that another as yet unidentified agent, often called agent X, might be the real culprit. I belong to the first group for three main reasons. The first one
is that at least seven different teams of researchers, from four different countries, have been able to experimentally reproduce clinical signs, characteristic PMWS histological lesions, and mortality, using PCV2 alone.\textsuperscript{1-7} The second reason is that many different studies, from at least nine different countries have reported a direct correlation between the quantity of PCV2 found in the blood and tissues, and the severity of PMWS.\textsuperscript{2-5, 8-21} So the more PCV2, the more problems. If PCV2 was not significantly involved with PMWS, why would there be such a correlation? Finally, the results obtained so far with commercial or experimental vaccines that contain only PCV2 antigens suggest that there are many situations where these vaccines provide an excellent protection. If PCV2 was not a component of that condition, why would vaccines based solely on PCV2 antigens help prevent problems, and sometimes completely solve them? This does not mean that agents X, Y or Z cannot or are not playing any role in field situations. We already know that PRRS virus, parvovirus and \textit{Mycoplasma hyopneumoniae} can either trigger PMWS problems experimentally, or make them worse. So it could well be that other organisms, including some that may not have been identified yet, may play a role. But for the reasons mentioned above to totally exclude PCV2 from the equation and consider agent X as the sole cause of this condition would not, at this time, seem logical to me.

One of the objections from agent X believers is that all herds tested so far in North America are infected with PCV2, and only a fraction of them are suffering PMWS losses. So if PCV2 was the cause of PMWS, why is it that so many infected herds are not showing anything? And while all herds in Quebec were positive to that organism, why did we suddenly begin to have such frequent and severe problems late in 2004? There are in my view two main possibilities: either we are now dealing with new, more virulent isolates of the virus, or something else, that we did not have before, is triggering PCV2 problems.

Using specific experimental models, it has been possible to reproduce PMWS experimentally with different isolates of PCV2. This made researchers believe initially that differences in the virulence of isolates was probably not what explained the wave of problems associated with that disease worldwide. In reality there have been only two studies so far that compared side by side the virulence of PCV2 strains. One was conducted in Sweden and compared a Swedish isolate and an old one from Canada.\textsuperscript{22} The conclusion from that study was that both strains were capable of producing problems, although the Swedish isolate produced slightly more severe clinical signs. The second one was recently conducted by Dr. Opriessnig et al\textsuperscript{23}, at Iowa State University, between two isolates identified in the US. One of the isolates was found to produce more severe clinical signs and lesions than the other one, and the researchers concluded that there were differences in the virulence of PCV2 isolates, and that these differences might be in part responsible for the variable severity of problems found in the field.

Similar to Quebec, Ontario has seen a sharp increase in the number of PMWS cases lately. In fact the number of cases identified in the Animal Health Laboratory went from about 25 in 2003, to approximately 60 in 2004 and to about 350 in 2005.\textsuperscript{24} Interestingly, this increase in the number of cases seems to have occurred at the same time as a change in the PCV2 isolates found in these cases. While the predominant RFLP pattern (based on the use of 2 restriction enzymes on a 902bp portion of the genome) was a type 422 in 2004 and
years before, it became a pattern 321 in 2005. When this 902bp portion of the genome was sequenced for 4 of these new type 321 viruses, they were found to have more than 99% sequence homology, and be only 91.6% similar to an older 422 isolate (Carman S, personal communication, 2005). Only one isolate tested had a 321 pattern in 2004, and 135 in 2005. An obvious question is thus to ask if there is a connection between the recent increase in the number and severity of cases in Ontario, and the fact that a different type of PCV2 isolates is now recovered from these cases.

Some results from Quebec are also of interest. The whole genome of 13 Quebec PCV2 isolates, obtained from cases with a history and histological lesions compatible with PMWS, was sequenced by researchers of the Faculté de Médecine Vétérinaire of the Université de Montréal and all isolates were found to be 99.9 to 100% homologous (Gagnon CA, personal communication, 2005). When compared to isolates that circulated in Quebec in the past, before significant problems were observed in the province, the homology found was only about 95-96%. When the RFLP pattern of recent Quebec isolates was deduced from the sequence, using the Ontario RFLP typing procedure, it was found to be the same (pattern 321) as the main one presently found in Ontario.

So if I summarize the recent Quebec\Ontario situation, we had a great increase in PMWS cases in 2005. In each province the isolates that are now isolated from sick pigs seem to be different than what they were before we had significant problems. Some of these recent isolates from each province were compared and found to be of the same 321 RFLP pattern. With all this, one can evidently be tempted to conclude that there must be a connection and that these new PCV2 isolates may simply be more virulent than the ones we had before. This is clearly a possibility, but until someone compares these ‘old’ and ‘new’ strains in an experimental model, this will remain an hypothesis.

**How does PCV2 get transmitted?**

Here are some points that may help to understand the various ways by which the organism could get transmitted:

- The virus has been reported to be excreted through nasal and ocular secretions, urine, feces and colostrum.
- It is also present in semen and some boars have been found to shed it for at least 24 weeks; the exact role of artificial insemination in the epidemiology of the infection however is not clear at this time.
- It is very resistant in the environment.
- Experimentally it has been possible to infect pigs by intra-nasal, oral, intra-muscular and intra-uterine inoculation.
- After experimental infection, some animals were found to be carriers for at least 125 days.
- Pigs from herds with no clinical signs can contract the disease if placed in contact with sick pigs, or if placed in close proximity.
- Pigs can become infected *in utero*, and the virus can cause acute reproductive problems, particularly in start up herds; this however is not frequent; there are some
data suggesting that PCV2 could be a cause of enzootic of chronic reproductive failure, but this is also an area that would need more investigation

- Isolates with different genotypes can be found between herds, and within the same herd
- Early work conducted on other species like cattle, horses and even humans suggested that they could become infected with this organism. More recent studies on the subject were unable to detect the presence of infection in people, horses, cattle, sheep, dogs, cats, mice, and poultry. However, certain laboratory mice can be infected with PCV2 experimentally, become viremic and have lesions.
- There has been speculation about the fact that feed ingredients, like spray dried plasma, could be a potential source of infection for pigs. In a recent experiment where six samples of spray dried plasma were tested by PCR, five were found positive (Gauthier R, personal communication, 2005). However the organism could not be cultured from any of the samples. This could suggest that either the organism was present in the samples but dead, the technique used was not sensitive enough to detect it or the type of sample itself (plasma) may act as some kind of inhibitor for isolation of the organism.

How can we control PCVAD?

Since PMWS was first described, in 1997, there have been lots and lots of suggestions on what could be done to reduce the severe losses that can be associated with it. As always, some appear better than others. In my book, at this time, so this could change, the following points are among those that seem to offer the best chances of success or improvement when PMWS is a problem: genetic changes, vaccination, management changes, serotherapy, the control of other diseases, like PRRS, that can trigger the condition or increase its severity and finally, depopulation/repopulation.

Genetic changes

It has become clear that genetics can have a significant impact on the occurrence of this disease, and on the losses associated with it. What remains to be clarified are the details and subtleties of this role. Here are some examples of situations where genetics appeared to have an impact:

- For many months the same herd had problems with PMWS in Landrace animals, but not in the Large White
- In an experimental infection, Opriessnig et al7 infected pigs of the Large White, Duroc and Landrace breeds. One Landrace pig developed PMWS and two others had characteristic gross and microscopic lesions of the condition. No pigs of the other two breeds showed clinical signs or gross lesions of PMWS. The authors concluded their results suggested that the Landrace pigs were predisposed to PCV2-associated lymphoid depletion and PMWS.
- Lopez-Soria et al28 reported the results of a comparison between the progeny of three different boar genetics (Pietrain, Large White (50%)/Pietrain (50%), and Large White (25%)/Duroc (75%) from 2 large 5000 sow Spanish herds. The sows were of the same genetic background (37.5% Large White x 37.5% Duroc x 25% Landrace) for all three genetics of boars used. In pigs from the two sow farms, the total
mortality rate and the mortality rate specifically associated with PMWS were, for the three different boar genetics, respectively 1.8 and 1.1%, 5.4 and 2.7%, and 16.3 and 12.4%.

- A similar experiment was conducted by Boivent et al.\textsuperscript{29} in Brittany, France. A sow herd was supplying pigs to two different wean to finish farms. Interestingly, one of these farms never had PMWS problems while in the other one these problems were persistent and responsible for a high weaning to slaughter mortality (about 11%). It was decided to evaluate the impact of boar genetics on mortality in the affected farm. Sows were either inseminated with semen from purebred Pietrain boars or from Large White (50%)/Pietrain (50%) boars. Two batches of pigs going in the farm with problems were compared. Globally, the mortality rate in pigs from the Pietrain boars was 3.9% while it was 12.9% for those from the Large White/Pietrain boars.

- In Brittany again, Larours et al.\textsuperscript{30} looked at 38 producers who went from various boar lines to the Pietrain breed. Of these producers 24 made the switch mainly in an attempt to reduce losses caused by PMWS. Only one was unsatisfied with the results obtained. Ten producers were totally satisfied and judged that the change in genetics solved their PMWS problems. For another six the losses were reduced, the problem was better controlled but still present. Finally the last seven concluded that the problems were either reduced or eliminated, but since they had also made other changes it was difficult to know if the improvement was only associated to the switch in genetics or not. Incidentally, for various reasons that likely include a better control of PMWS problems, the percentage of semen doses sold in France from Pietrain boars has dramatically increased over the last few years, and is probably at 50% or more today.

- Belgium is surrounded by countries (France, Germany and The Netherlands) that have been hit hard with PMWS. Yet this small country did not have a significant PMWS problem in the past, and still doesn’t. Not that it’s not present, but the cases are not frequent and usually not associated with high losses. The Flanders area is very hog dense and the health status is not thought to be better there than in the neighbouring countries. Why is it then that of all the countries in South-Western Europe, only this one seems to have avoided the major pain and suffering associated with PMWS? Well, nobody knows for sure, but one of the possible explanations could be that the vast majority of producers in that country use Pietrain boars.

- A minimal disease 500 sow herd in Northern Ireland began experiencing significant losses because of PMWS in 2005. The decision was made to breed a percentage of the sows with a different commercial boar line, that we will call boar A. This genetic line does contain Pietrain blood. While the losses attributed to PMWS were at 7.9% in the progeny of other boars used on the farm for a period of 5 months, it was only 0.9% in the pigs born from boar line A. In a second herd from Northern Ireland the mortality associated to PMWS had been between 4 and 10% for about a year. When a switch was made to the same boar line as used in the previous herd, the mortality dropped to less than 2% (Allan G, personal communication 2006).

- A Canadian company had significant problems with PMWS. Pigs from the same sow genetics but from two different boar genetics were raised together in two finishing units. When compiling together the results in these two units, pigs born
from Duroc boars had about twice the mortality (9.6 vs 5.1%) observed in the pigs born from the other boar genetics, which does contain Pietrain blood (Moore C, personal communication, 2006). The same comparison with the same genetic sow and boar lines was done in four finishing units of another Canadian company. Again pigs from both genetics were raised in the same finishing units. Although the batches were not completed at the time of this writing, the data obtained so far show the same trend. While the pigs born from Duroc boars were clinically affected, the clinical signs suggestive of PMWS were much reduced in pigs born from the other boar genetics. The mortality rates for the two different genetics in these four batches were: 13.4 vs 5.2, 17.8 vs 6.1, 14.6 vs 8.5 and 4.3 vs 3.3%. It should be mentioned that the PRRS virus is also part of the problem in these batches, and that the last batch (4.3 vs 3.3%) has shown few clinical signs of PMWS up to now and may end up unaffected (Bellavance M, personal communication, 2006). In yet a third company, this time in the US, the same boars that appeared to have a relative protective effect in some Canadian herds were used in matings with females of a different sow line. The pigs produced from this different genetic combination suffered severe losses associated with PMWS (Jones R, personal communication, 2006). If all this was standing the test of time, it would suggest that the same boar line used with different sow lines could produce pigs with different susceptibilities to the disease. So both the female and the males used may have an impact, which is not necessarily a big surprise.

• In the Netherlands, an epidemiological study involving herds with and without PMWS and PDNS problems was undertaken to try determining what factors could play a role in the occurrence and severity of the problems. An association was observed between PMWS/PDNS and the historical use of breeding stock from an Anglo-Saxon origin. When switching from commercial genetic lines of Anglo-Saxon origin to another one, the losses in many farms dropped sharply (de Jong M, personal communication, 2006).

• Back to Canada, a company used different genetic lines and over time it appeared that there were variations in the susceptibility of pigs to PMWS depending on their genetic composition. One particular genetic line seemed to be much less susceptible to PMWS problems than others (Surprenant C, personal communication, 2006).

• In the province where I live (Quebec) gilts are frequently bought at about 3-4 weeks of age and mixed with commercial pigs from the farm. The idea is to have them acclimated to the microorganisms of the herd before they are ready to be bred. A frequent observation, by many different practitioners, is that these gilts often have fewer PMWS problems when compared to commercial pigs in the same barns. The gilts are in most cases Large White/Landrace females, while the commercial pigs are usually 25% Landrace, 25% Large White and 50% Duroc. So the boar line generally used is of the Duroc breed. Recently data were compiled in 18 different finishing units that contained both F1 gilts and commercial pigs (Bellavance M, personal communication, 2006). The average mortality in the F1 gilts was 4.8%, while it was 12.6% in the commercial pigs raised in the same barns and receiving the same feed. It is known that the total mortality rate in females is normally lower than it is for barrows, so since the F1 gilts are all females and they are compared to commercial pigs that are 50% barrows and 50% females, a small difference in mortality could be
explained that way. But in the present case we are talking about a mortality rate that is 2.6 times higher in commercial pigs than it is for the F1 animals, and this can evidently not be explained solely by the difference in the percentage of females in each group.

- Even within the same breed there might be differences in the relative susceptibility or resistance to that condition. Table 1 shows the mortality of pigs born from two different Duroc boar lines, within the same system, before and after PMWS became a problem (Turner M, personal communication, 2006).

<table>
<thead>
<tr>
<th>Period</th>
<th>Duroc A</th>
<th>Duroc B</th>
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<tbody>
<tr>
<td>Jan-June 2005 (before</td>
<td>2.5 % (245,945)*</td>
<td>3.5 % (316,297)</td>
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<tr>
<td>PMWS)</td>
<td></td>
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<tr>
<td>Jan-Dec 2005</td>
<td>2.6 % (278,704)</td>
<td>4.3 % (898,280)</td>
</tr>
<tr>
<td>Jan-Apr 2006 (with PMWS)</td>
<td>3.0 % (29,504)</td>
<td>7.5 % (490,319)</td>
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* Number of pigs included

As can be seen, one genetic line within the Duroc breed appeared to be much less affected with PMWS than the other. While the mortality of progeny from line A was increased by only 0.5% after PMWS became a problem, it was increased by 4% in the progeny of line B. Interestingly, there was a 1% mortality difference between the two lines even before the PMWS problems, suggesting a genetic difference on the mortality associated to causes other than that condition.

As mentioned above, we don’t yet understand all the details and subtleties of the role played by genetics, but there is clearly one. The use of the Pietrain breed, or of lines containing Pietrain blood has appeared to have a beneficial impact in several different situations, but not in all. In a study conducted by Rose et al\(^32\) in four French farms, no beneficial effect of the Pietrain boars against PMWS could be detected in their progeny. One must thus be careful not to generalize or jump to conclusions too quickly. This is particularly true for comparisons that are made before and after a change, so without contemporary controls, since there are situations where the results improve without changing anything. It is also important to realize that the relative susceptibility or resistance of pigs to that condition is only one of the aspects to evaluate before deciding to switch from one particular genetic line to another. For example, the Pietrain breed has a reputation of producing a meat that has less marbling than desired by at least some markets. So factors other than the possible resistance to PMWS evidently have to be considered before making a change.

**Vaccines**

A PCV2 vaccine for use in sows and gilts has been available in France and Germany for about two years. In one study from the first country, the weaning to slaughter mortality rate dropped from 11.0% to 7.7% in 15 herds where the vaccine was used.\(^33\) In the second country the birth to slaughter mortality rates in 38 herds where the vaccine was used dropped from 28.7% to 17.9%.\(^34\) In each of these studies the results were compared before
and after the use of the vaccine, so it could be that in some herds the performance might have improved in the absence of vaccination. Nevertheless, the information I got from several French practitioners suggests that the vaccine is frequently useful to prevent losses associated with PCVAD.

Similarly, results obtained in Canada and the US with pig vaccines, both commercial and experimental, suggest that these vaccines are efficacious. The field information gathered to date indicates that losses have been reduced by about 50% in some cases, while in others a total elimination of PCVAD losses has been observed. In fact in Quebec there are even situations where the performance after vaccination appears to be better than it was before the PCVAD outbreak. This would tend to suggest that in the past some losses may have been associated to PCVAD without being recognized as such.

Management
As for most other diseases the quality of management can help to prevent or reduce the negative impact of PMWS. A French scientist, Dr. François Madec, has proposed a list of 20 rules which, when followed, have reduced the severity of losses in a number of herds: 35

Farrowing room
- Emptying of pit, cleaning, disinfection
- Wash sows and treat for parasites
- Adoptions: limit cross fostering to what is strictly necessary and only within 24 hours of farrowing; observe parity rank
- Conformity of vaccination plans

Nursery
- Small nursery pens, solid partitions
- Empty pit, clean, wash and disinfect
- Lower stocking density (3 pigs/m²)
- Increased feeder space (7 cm/pig)
- Perfect ventilation
- Perfect temperature
- No mixing of batches (1 batch per room)

Finishing
- Small pens with solid partitions in finishing
- Empty pit, wash and disinfect
- 0.75 m²/pig
- Temperature, ventilation: OK
- No mixing of pens
- No mixing of batches

Other measures
- Respect flow of air and animals within buildings
- Strict hygiene (tail and teeth clipping, castration, injections…)
- Early removal of sick pigs to hospital pens
As can be seen, many of the measures proposed are basically applying good husbandry practices, and as for some of the others they would be difficult to implement in many of our North American systems (e.g. multi-site systems). Nevertheless, it is believed in France that the more of these rules are applied, the more chances there are to reduce losses associated to PMWS.

Lots of other strategies have been suggested by different authors to help control PMWS. These include: reduce the number of weaned or feeder pig sources; reevaluate the vaccines and vaccination programs used; use disinfectants (e.g. Virkon S) that have good activity against PCV2; batch farrowing every 2, 3, 4 or even 5 weeks; partial depopulation of the nursery; bioflavonoids, vitamin E and Se, antioxidants, mash feed, feeds with larger particle size, restricted feeding, no feed changes after moving pigs, richer diets; no hospital pens, so either euthanize sick pigs or move them elsewhere; increase weaning age; acetaminophen, acetylsalicylic acid, florfenicol, tilmicosin; closing the herd; use measures to improve colostrum intake; all piglets to suckle their natural mothers for the first 24 hours; etc.

The list seems almost endless and one must admit that the results obtained have been very variable, and quite frequently disappointing. There are however situations showing that management strategies and infection pressure may have a significant impact on the outcome. For example Boivent et al\textsuperscript{29} reported that the same sow herd was sending piglets to two different weaning to finish operations, and for about one year one had major problems with PMWS while the other had none.

**Serotherapy**

Ferreira et al\textsuperscript{36} were the first to propose serotherapy as an alternative to prevent losses associated with PMWS. Piglets that were about 33 days of age were injected subcutaneously with 20 mL of serum from pigs ready for market that had gone through the problem and had recovered. The results obtained in three trials were excellent (15.2% vs 4.9%; 18.5% vs 2.7%; 17.9% vs 2.8% mortality). Different variations of this strategy, in most cases using lower volumes of serum, have been used successfully in other countries like Spain, UK, the Czech Republic and Canada. But the procedure is not easily applied, there are risks associated with it and except for a few particular situations, the results obtained in Quebec have overall been relatively disappointing.

**Control of other diseases**

Other diseases, PRRS in particular, can either trigger PMWS problems or make them worse. In Quebec, losses in PMWS affected farms are on average much higher in PRRS positive than in PRRS negative farms. The mortality rate in PRRS negative farms does not often exceed 10 or 15%, while we have seen cases where 50% or more of the pigs were dying in PRRS positive farms. Furthermore, in some experimental infections the losses were much higher when both PCV2 and PRRS virus were inoculated, compared to either virus given alone.\textsuperscript{2,6} It is thus important to try controlling all infections that could potentially make things worse, and this includes among others enzootic pneumonia and swine influenza. The same can be said about parvovirus if it is found to circulate in pigs close to the time when they get infected with PCV2.
Depopulation/Repopulation

Hassing et al.\textsuperscript{37} reported that of six Danish herds that were depopulated, cleaned, disinfected and left emptied for 3–4 weeks, then repopulated with animals from herds without PMWS, five got rid of the problem. In the last herd it reappeared about three months after the program, but in that case the supplier of pigs was the same as before the depopulation/repopulation. Gresham et al.\textsuperscript{38} also reported that PMWS had not recurred in three farms after complete depopulation and re-stocking with pigs from unaffected farms. Thus successes in the control of PMWS have been obtained in the past with depopulation/repopulation, and it could constitute an alternative. It should be reminded though that since we still don’t fully understand the epidemiology of that condition, and the ways by which it can become a problem, caution should be exercised when deciding to take a costly decision such as depopulation and repopulation. We had very few herds in Quebec with significant PMWS problems before late 2004, we have many since early 2005 and we’re not sure yet why.

Are there other conditions that could be associated in one way or another to PCV2?

Desrosiers\textsuperscript{39} has reviewed some conditions for which a potential association with PCV2 has been made. These conditions include porcine dermatitis and nephropathy syndrome or PDNS, sow reproductive problems, boar infertility, porcine respiratory disease complex or PRDC, enteritis, necrotizing lymphadenitis, multifocal interstitial nephritis, necrotizing tracheitis, proliferative and necrotizing pneumonia, hepatopathy, myocarditis and vascular lesions, necrosis of skeletal muscles, gastric ulcers and congenital tremors.

As can be seen, the number of conditions to which PCV2 has been associated over the last few years has increased significantly. In fact there may be other conditions, different from those described in this list that could also be associated with PCV2 in one way or another. For example, some French veterinarians believe that there could be an association between PCV2 and cases of ear necrosis. Similarly some reports are suggesting that PCV2 could potentially be associated with porcine epidemic diarrhea and exudative epidermitis.

Of course the role that the organism may play in many of these conditions requires further clarification, but what is clear is that PCV2 is not only the PMWS organism.

Concluding remarks

PMWS/PCVD/PCVAD has produced severe losses in different areas of the world. While North America was to a certain extent spared by these severe losses up to recently, we now have areas where these losses are unacceptably high and solutions had to be found. Different control alternatives have been briefly discussed in this paper. In my opinion the two main ones that will make our lives much easier with this condition are genetics and vaccines. Some genetic combinations are clearly more resistant to it than others, and the preliminary results obtained with vaccines are such that an effective and practical control of the problems associated with PCV2 is now a reality.
References