factsheet
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Porcine Circovirus Associated Disease: Description, Cause and Transmission

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Introduction

Postweaning multisystemic wasting syndrome (PMWS) is now called porcine circovirus disease (PCVD) in Europe, and porcine circovirus associated disease (PCVAD) in North America. Starting at the end of 2004, and particularly since the beginning of 2005, cases of PCVAD in Quebec increased dramatically. Simultaneously in Ontario and a little later in North Carolina, the same phenomenon of dramatic increase in PCVAD cases was observed. The disease has now spread to other parts of both Canada and the US. This fact sheet will cover what the condition looks like, what is causing it, how it gets transmitted and what reasons could explain why we now have such significant problems compared to what we had in the past.

What does it look like?

The clinical picture appears to always include wasting, a lack of response to conventional treatments and an increased mortality. This mortality can vary significantly from one herd or system to another. In some situations the cases are sporadic and only of minor importance. In others the mortality can get to 10-15%, and sometimes much higher when other micro-organisms, like PRRS virus for example, are involved. Other clinical signs like diarrhea, paleness, thumping, jaundice and skin lesions like those seen in porcine dermatitis and nephropathy syndrome (purple to black spots on the skin) also vary in severity from one case to another. The clinical signs can usually be observed anytime between 5 and 20 weeks of age. In North America there are many cases that occur 3 to 5 weeks after placement in the finishing units.

The post-mortem gross lesions that can be seen more commonly include lungs that do not collapse normally, an enlargement of lymph nodes and kidneys that are either larger than normal and/or have white spots on them. The lymph nodes and kidney lesions are particularly suggestive. Many other lesions can be seen, but they seem to vary quite a bit in frequency. There are cases for example where we may see severe interlobular edema (jelly like material forming lines on the lungs), edema of the mesentery (jelly-like material between the intestinal loops) and on occasion the presence of lesions on the spleen (small infarcts or blackish buttons, or enlarged spleen with dark areas). No single lesion appears to be present in all cases, so it is often necessary to perform several necropsies of sick, representative pigs to have a better chance of identifying some of the more suggestive lesions. Final confirmation of the diagnosis however requires sending pigs or tissues to the laboratory.

In fact, a confirmed diagnosis of PCVAD requires three things:

- Animals that are wasting (exhibiting weight loss)
- Characteristic lesions observed with a microscope (lymphoid depletion, granulomatous inflammation; inclusion bodies may or may not be present)
- Identification of porcine circovirus type 2 (PCV2) in the lesions

The description above concerns what we were previously calling PMWS, but there are many other conditions for which a potential association with PCV2 has been suggested. These conditions include porcine dermatitis and nephropathy syndrome (PDNS), sow reproductive problems, boar infertility, porcine respiratory disease complex (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. There are also reports of a possible association between PCV2 and porcine epidemic diarrhea (not thought to be present in North America), exudative epidermitis (greasy pig disease) and other skin problems like ear necrosis. For some of these conditions or lesions however the data are often not consistent and more studies are needed to better understand if and to what extent the virus may be involved with them. Others (reproductive, enteric and respiratory problems, multifocal interstitial nephritis, hepathopathy, myocarditis and vascular lesions, gastric ulcers) were observed in pigs that had been experimentally infected with PCV2 alone, so for them the potential link appears stronger. There is one report of four sows that were experimentally infected with PCV2 and one of them developed skin lesions similar to those observed in PDNS [1]. While this appears to be the only time when PDNS may have been reproduced in an experimental infection with PCV2, many field observations made in different countries suggest that PCV2 could be associated in one way or another with PDNS.

What causes PCVAD?

Two main positions on the cause of PCVAD have been debated. There are those who believe that PCV2 is the cause of the condition, although other factors or agents may be involved and contribute significantly to the losses associated with it in the field. There are other researchers who consider that another as-yet-unidentified agent, sometimes referred to as agent X, might be the real culprit [2-5]. Three main reasons seem to favour the first hypothesis. The first one is that several different teams of researchers, from four different countries, have been able to experimentally reproduce clinical signs, characteristic PCVAD histological (seen with a microscope) lesions, and mortality, using PCV2 alone [6-13]. Admittedly there are also several other studies in which clinical signs were not observed following experimental infection with PCV2 alone, but this is also true for many other pathogens. For example, there are many experiments in which inoculation of PRRS virus in pigs did not produce clinical signs. The second reason is that many different studies have reported a direct correlation between the quantity of PCV2 found in the blood and tissues, and the severity of PCVAD [7-11,14-16]. In other words, the more PCV2 in tissues, the more severe is the condition. If PCV2 was not important in this condition, why would there be such a direct correlation? Finally, the results obtained so far with vaccines that contain only PCV2 antigens suggest that there are many situations where these vaccines appear to totally eliminate losses associated with PCVAD. Again if PCV2 was not involved, why would a PCV2 vaccine control the problem so efficiently in these situations?

This however does not mean that other agents that we could call X, Y or Z cannot or are not playing any role in the field expression of that condition. We already know that pigs experimentally inoculated with both PCV2 and PRRS virus, or PCV2 and Mycoplasma hyopneumoniae, or PCV2 and parvovirus can be more severely affected than if they are infected with PCV2 alone. There are published scientific reports substantiating that and realistically, there are no reasons to believe that the list of potential triggering or aggravating agents would be limited to these ones. So it may well be that other pathogens or factors, even some that have not yet been identified as such, could be involved and could exacerbate directly or indirectly PCVAD problems.

One of the objections from agent X believers is that almost all herds tested so far in Canada and the US are infected with PCV2, and only a fraction of them are suffering PCVAD losses. In fact of all the herds that have been tested so far in Canada and the US, it seems that only one has been found negative. So if PCV2 was the cause of PCVAD, why is it that so many infected herds are not showing anything? And while all tested herds in Quebec were positive to that organism before 2004, why did we suddenly begin to have

such frequent and severe problems late that year? There are in my view three main possibilities:

- We are now dealing with new, more virulent isolates of the virus
- Something else, other than PCV2 and that we did not have before, is triggering PCV2 problems
- Or we may have a bit of both.

Opriessnig et al have shown experimentally that there can be virulence differences between PCV2 isolates [17]. In that study, while none of the animals showed clinical signs, one isolate was shown to produce more severe gross and microscopic lesions than the other one tested. Work conducted by Lager et al [13] also suggests that virulence difference could exist between isolates. Eastern Canada has seen a sharp increase in PCVAD cases in the last 2 years and that was chronologically associated with a change in the type of isolates identified in these cases [18]. Recently, Timmusk et al [19] reported that in Sweden, a comparison between isolates found in pigs from healthy herds and pigs with clinical signs of PCVAD revealed differences both at the nucleotide and amino acid levels, differences which could possibly be responsible for variations in pathogenicity. However other studies failed to find significant differences between isolates that could explain why the disease was found in some herds, and not in others [20-22]. To what extent the problems that we are now facing in North America could be explained by an increased virulence of the PCV2 isolates involved remains one of the main questions to be answered.

How deoes it get transmitted?

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

- Virtually all farms are infected with one or a few strains of PCV2, so when a farm is populated, it is introducing breeding animals, weaned pigs or feeder pigs that are already infected. So basically all farms are infected at the moment they introduce pigs.

- The virus has been isolated or detected by PCR from nasal, rectal, urinary, salivary, ocular and tonsillar swab specimens, as well as from colostrum [23,24].

- It is also present in semen and some boars have been found to shed it for at least 27 weeks [25]; French researchers have shown that if the virus is placed in the uterus of sows just after insemination, they can get infected and have reproductive problems [26]. However, epidemiological investigations conducted in different countries are suggesting that semen did not play an important role in the diffusion of the disease in these countries [2,27,28]. So the exact role of artificial insemination in the epidemiology of the infection is not clear at this time.

- PCV2 is very resistant in the environment.

- The oro-nasal route is considered the most likely and frequent route of PCV2 transmission [23]

- After experimental infection, some animals were found to be carriers for at least 125 days [7]

- Pigs from herds with no clinical signs can contract the disease if placed in contact with sick pigs, or if placed in close proximity [29]

- Pigs can become infected while in the uterus of the sow, and the virus can cause acute reproductive problems, particularly in start up herds [30,31]. This however is rare. There are some data suggesting that PCV2 could be a cause of chronic reproductive failure, but other data suggest that this is not likely to be very significant.

- In recent studies conducted to determine if species other than swine could become infected with PCV2, negative results were obtained for all the species tested (humans, horses, cattle, sheep, goats, dogs, cats, rabbits, Guinea pig, mice and poultry) [32-34]. However, certain laboratory mice can be infected with PCV2 experimentally, become viremic and have lesions [35].

- 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 polymerase chain reaction (PCR; a laboratory technique which identifies PCV2 DNA), 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 culture 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. In several experiments conducted at Iowa State University where spray dried plasma was used to feed negative animals, these animals have remained non-infected. For the time being we can probably say that pigs fed this feedstuff will not always become infected with PCV2, but it is too early to say if it is impossible for pigs to get infected that way.

Finally, epidemiological studies conducted in different countries have yielded variable results concerning potential risk factors. For example in the UK, data from Cook et al [36], and Green et al [16] suggested that proximity to affected herds was a risk factor to develop PCVAD problems. In contrast, distance to affected herds was not found to be a significant risk factor in a recent Danish study conducted by Enøe et al [27].

There is still much to learn about this condition, but what is known today is way better than what it was yesterday.

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