We are delighted that the International Pig Veterinary Society Congress 2004, decided to select South Africa as the host country for the 20th IPVS Congress. The Pig Veterinarians of South Africa will ensure that this congress lives up to the best traditions of previous congresses; incorporating an interesting and topical scientific programme, fascinating accompanying persons tours and an excellent social programme, allowing delegates the opportunity to network with their overseas colleagues.

This, the first IPVS congress on the African continent, will undoubtedly be of enormous benefit in generating solutions to the emerging pig veterinary challenges, especially those related to exotic and changing viral diseases, decreased use of antimicrobials and nutritional advances. The congress is important to further pig veterinary science in South Africa, to encourage younger veterinarians to join the pig industry, as a vehicle to generate funds for research and to improve the pig industry in Southern Africa.

South Africa is a magnificent and beautiful country, and offers tourists value for money. Thus, pre and post congress tours will be a major attraction for delegates to come to South Africa. Durban, in KwaZulu Natal, is a vibrant multi-cultured city with magnificent beaches, easily accessible game parks, theme villages and a moderate winter climate making it an ideal tourist destination. We urge our colleagues throughout the world to use this opportunity to get a glimpse of the continent’s rich and fascinating wonders and to enjoy the hospitality of their African friends.

Dr Peter Evans
Chairman: Local Organising Committee: IPVS 2008
PMWS/PCVD: DIAGNOSIS, DISEASE, AND CONTROL: WHAT DO WE KNOW?

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Introduction

Postweaning multisystemic wasting syndrome (PMWS) is now recognised as a global, epizootic disease that causes significant economic losses to pig producers. A "new" circovirus of pigs, porcine circovirus type 2 (PCV2) is now recognised as being the essential infectious agent of PMWS (1). However, in addition to PMWS, strong evidence from field and experimental studies has also linked PCV2 infections to reproductive problems in pigs and early (post weaning) and late porcine respiratory disease complex (PRDC), (2). PMWS is now seen as the most important clinical manifestation of a range of porcine circovirus diseases (PCVDs). The global explosion of PCVDs in the last 5 to 10 years has raised many questions regarding the source and nature of the disease epizootic. Retrospective testing of sera and tissue samples from pigs has shown that PCV2 infections occurred in pigs from at least 1969 (3) and sporadic cases of classical PCVD have now been identified in Spain and England from 1986 onwards (4).

PCV2 is not a "new" virus and PCVD is not a "new" disease. However, in contrast, field observations and epidemiological studies in the UK and Denmark strongly suggests that the spread of PCVD since 1999 has been consistent with the introduction of a "new" infectious agent into a naïve population. To date research into PCV2 and PCVD has provided some answers to specific questions. However, the results generated by different research groups have been equivocal. This short article will highlight some of the areas where results of research and field studies have generated more questions than answers.

PMWS: Diagnosis

The individual pig: We have to start somewhere! PMWS is now recognized as the major clinical manifestation of PCVD. The disease in individual animals of all age groups is characterized by clinical signs which can include growth retardation, dyspnoea, enlargement of inguinal lymph nodes, diarrhoea, and/or occasionally jaundice (2). However, not all of these clinical signs will be seen in all individual pigs affected with PMWS. At necropsy, the most frequent lesions seen are enlargement of lymph nodes and non-collapsed, tan-mottled lungs (5). The main histological lesions consist of a variable degree of lymphocyte depletion with loss of follicles together with histiocytic and multinucleate giant cell infiltration in the lymphoid tissues, and lymphohistiocytic inflammatory infiltrations in a wide range of tissues (5). The association of moderate to high amounts of PCV2 virus antigen and/or nucleic acid with these lesions is an essential criterion for the diagnosis (6). It has been suggested that the definition of a PMWS-affected pig should not include the inclusion of PCV2 association with lesions. It is difficult to understand why the presence of PCV2 associated with lesions should not be acceptable as an essential criterion for diagnosis of a PCVD, and, if this is not to be included, what diagnostic criteria should be used in determining if an individual pigs has PMWS/PCVD? Certainly thin or wasted pigs alone do not fulfil the criteria for PMWS/PCVD (7) and a constellation of histological lesions have been recorded in cases of PMWS/PCVD, all of which do not always appear in individual affected pigs.

The herd case: An attempt to clear muddy waters! The criteria for a herd diagnosis of PMWS/PCVD are still under debate. Essentially this debate revolves around the fact that some herds can have occasional individual deaths, which fulfil the criteria of PMWS outlined above. This situation is undoubtedly similar to what was seen prior to the global explosion of PMWS in the mid 1990s when individual cases of PMWS occurred on some farms and were misdiagnosed. In an attempt to clarify the definition of a herd case of PMWS, the EU multidisciplinary consortium currently working on PCVD (Control of Porcine Circovirus Diseases (PCVDs): Towards Improved Food Quality and Safety. [pcvd.org]) has proposed their definition of a PMWS herd case. An abridged version of this definition is presented below.

1) The occurrence of PMWS is characterised by an excessive increase in mortality and wasting post weaning compared with the historical level in the herd. There are two options (1a and 1b) for recognising this increase, of which 1a should be used whenever possible:

1a: If the mortality has been recorded in the herd, then the increase in mortality may be recognised in either of two ways:
Current mortality mean of historical levels in previous periods + 1.66 x SD2 or statistical testing of whether or not the mortality in the current period is higher than in the previous periods by the chi-square test. In this context, mortality is defined as the prevalence of dead pigs within a specific period of time. The current time period is typically one or two months. The historical reference period should be at least three months.

1b: If there are no records of the mortality in the herd, the following approach may be used: An increase in mortality exceeding the national or regional level by 50% is considered indicative of PMWS.
were successful in infecting foetuses and in producing reproductive problems by intranasal inoculation of pregnant sows. Reproductive disease associated with PCV2 infection has been described under field conditions (8, 23). Some authors failed to reproduce clinical disease (15). Attempts at experimental reproduction of PCVD using tissue homogenates from PCVD-affected animals consistently and/or PCVD can be transmitted through feeding untreated products from PCVD-affected animals to pigs. Indeed, early proposed that PCVD can be introduced into a farm through feeding untreated swill containing “contaminated” pig. Findings in that only one of ten in-contact animals developed disease (un-published observation). Although it has been the disease is not highly contagious. Similar studies on co-mingling of pigs at our institute have confirmed these completed with a low rate of disease transmission being recorded (P. Baekbo, personal communication), indicating that horizontal transmission of PCV2 between pigs is very efficient. Seroconversion to PCV2 between 2 and 4 months of age (19, 20), indicating that horizontal transmission of PCV2 between workers to propose that PDNS should be recognised as a PCVD. However, to date, PDNS has not been reproduced experimentally following infection with PCV2 virus nor is PCV2 antigen consistently found in typical PDNS—histological lesions. Although many epidemiological studies and field observations suggest a link between the occurrence of PMWS and PDNS, it is still too early to class PDNS as a PCVD.

**PCVDs: Diagnosis**

Although PMWS is currently considered the major disease presentation of PCV2 infection, a number of other disorders have been linked to infection with this virus and some of these should be considered under the umbrella of porcine circovirus diseases (PCVDs). PCV2 is now recognised as a causal agent of reproductive disorders in pigs (8). The case definition for PCV2-associated reproductive problems should include three main criteria:

1. Abortions and/or stillbirths and/or mummified foetuses
2. The presence of foetal heart lesions characterized by extensive fibrosing and/or narcotizing myocarditis
3. The presence of PCV2 in the myocardial lesions and other foetal tissues

PCV2 antigen has been demonstrated in abundance in lung lesions from pigs with proliferating and necrotising pneumonia (2), in tissues from sows with sow abortion and mortality syndrome (SAMS) (9) and PCV2 is also considered a contributor to porcine respiratory disease complex (10, 11). Recently PCV2 has been associated with acute respiratory disease in fattening pigs in the UK (Jake Waddilove: personal communication). It is currently not possible to definitively outline the role of PCV2 infection in some of these disease complexes as experimental reproduction of the diseases has not been carried out with an inoculum containing PCV2 virus. Porcine dermatitis and nephropathy syndrome (PDNS) is a disease that may affect nursery and growing pigs, and, sporadically, adult animals (12). An increased prevalence of PDNS has been reported in association with outbreaks of PCVD (13) which has led some workers to propose that PDNS should be recognised as a PCVD. However, to date, PDNS has not been reproduced experimentally following infection with PCV2 virus nor is PCV2 antigen consistently found in typical PDNS—histological lesions. Although many epidemiological studies and field observations suggest a link between the occurrence of PMWS and PDNS, it is still too early to class PDNS as a PCVD.

**PCVD: The Disease**

Although multiple attempts to experimentally reproduce PCVD have been published in the literature, to date, the disease progression of PCVD following experimental infection or natural infection in the field has not been fully elucidated. Indeed, very little is actually known about the pathogenic process of PCV2 infection in pigs leading to clinical PCVD.

Transmission of PCV2 infection and PCVD: Still a debate! The oro-nasal route is considered the most likely and frequent route of PCV2 transmission (14, 15, 16, 17, 18). Under commercial farm conditions, the majority of pigs seroconvert to PCV2 between 2 and 4 months of age (19, 20), indicating that horizontal transmission of PCV2 between pigs is very efficient. Transmission of PCV2 following co-mingling of affected pigs from a diseased farm with un-affected pigs from a non-diseased farm (21) has been demonstrated. A repeat of this experiment which included better controls has recently been completed with a low rate of disease transmission being recorded (P. Baekbo, personal communication), indicating that the disease is not highly contagious. Similar studies on co-mingling of pigs at our institute have confirmed these findings in that only one of ten in-contact animals developed disease (un-published observation). Although it has been proposed that PCV2 can be introduced into a farm through feeding untreated swill containing “contaminated” pig products (22), to date, there is no scientific evidence that supports this proposal. It is not known if PCV2 infection and/or PCVD can be transmitted through feeding untreated products from PCV2-affected animals to pigs. Indeed, early attempts at experimental reproduction of PCVD using tissue homogenates from PCV2-affected animals consistently failed to reproduce clinical disease (15). Reproductive disease associated with PCV2 infection has been described under field conditions (8, 23). Some authors were successful in infecting foetuses and in producing reproductive problems by intranasal inoculation of pregnant sows.
PCVD: PCV2 replication and lymphoid depletion. Still a puzzle! Still unresolved is the identification of the main target cells in the pig that support PCV2 replication. The large amount of PCV2 found in lesions in macrophages and dendritic cells of diseased pigs appears to be the result of accumulation of viral particles (28, 29) and not the result of active virus replication in these cells. However, it is still possible that PCV2 replicates in a small, as yet unidentified sub-population of these cell types. A recent in vitro study (30) has reported the significant up-regulation of PCV2 replication in lipopolysaccharide (LPS) stimulated porcine alveolar macrophages (PAMs). This is in contrast to the results reported by others (28, 29, 31), and although a clear differences in susceptibility of PAMs from different pigs to PCV2 infection has been reported (31) the authors failed to demonstrate evidence of production of new infectious PCV2 virus in these studies. PCV2 replication has been demonstrated in vitro in porcine aortic endothelial cells, porcine gut epithelial cells and porcine fibrocytes (32).

Recent in vitro studies have identified that PCV2 enters cells of the porcine monocyte line 3D4/31 via clathrin-mediated endocytosis and requires an acid environment. Additionally it was demonstrated that PCV2 can use a heparin sulfate and chondroitin sulfate B glycosaminoglycan as a receptor for cell attachment (33). In contrast, it has also been reported (32) that PCV2 does not bind to heparin sulfate receptors on porcine-derived dendritic cells. Further studies on receptor sites for PCV2 on a range of primary cell types, derived from pigs, are required. A number of experimental infection studies have been reported that have attempted to determine the primary sites of replication of the virus in the host and the disease progression of PCVD (14, 9, 34). In these studies PCV2 has been demonstrated in diseased pigs in a wide range of lymphoid tissues, liver, lung, myocytes, endothelial and epithelial cells. PCV2 antigen has been demonstrated in small numbers of porcine B and T lymphocytes in tissues from field cases of diseased pigs (35), however the presence of the virus in these cell types following experimental infection is not a common finding (14, 17, 34, 36). To date none of these studies have convincingly identified early replication sites for PCV2 in infected pigs nor have they elucidated the mechanisms for establishment of the primary lesions of lymphoid depletion and granulomatous inflammation infiltration of cells containing, but not replicating PCV2.

An ongoing study at Veterinary Sciences Division, Belfast is attempting to identify the early replication sites of PCV2 in experimentally infected pigs. In this study preliminary results indicate that PCV2 can be recovered on day 1 post infection (PI) from the small and large intestine, mesenteric lymph node, tonsil, bone marrow and nasal mucosa. At 3 days PI virus can be recovered from the large intestine, bronchial and mesenteric lymph nodes, nasal mucosa and trachea. From day 5 PI PCV2 was recovered from lungs, large intestine, nasal mucosa, bronchial mucosa, all 6 lymph nodes sampled and oesophagus of all sacrificed pigs. By day 14 PI, PCV2 was recovered from all tissue samples taken. Although others have reported PCV2 viremia (PCV2 DNA) at ≥ 3 days following experimental infection (37), in the current experiment infectious PCV2 was not recovered from serum or PBMCs until day 7 days PI. Further studies on these samples have yet to be completed.

The mechanism by which lymphoid depletion occurs in PMWS-affected animals remains to be elucidated. Potential mechanisms for a viral induced lymphoid depletion include a direct consequence of virus replication in immune cells or an indirect consequence of virus replication such as interference with antigen presentation, apoptosis induction, altered cytokine expression of immune cells or inhibition of the complement (38). However, PCV2 infection of lymphocytes has not yet been conclusively demonstrated, suggesting that lymphocyte depletion is more likely to be an indirect effect of PCV2 infection such as cytokine imbalance, apoptosis or alteration of migration pathways (39). Systemic lymphoid depletion following PCV2 infection has been attributed to apoptosis (39) who concluded that lymphocyte depletion with apoptotic death of B lymphocytes was caused by PCV2. However in a later experimental study in gnotobiotic pigs (40) and a study of field cases of PCVD (41) both groups concluded that apoptosis was not the primary mechanism of lymphoid depletion in PCVD. Recently (42) have reported that, following transfection of PK/15 cell cultures, the PCV2 protein coded for by ORF 3 is involved in PCV2-induced apoptosis by activating caspase-8 and caspase-3 pathways. Conversely (29) failed to demonstrate any apoptotic effects following infection in vitro of monocytes cells with PCV2 virus nor in lymphocytes co-cultured with PCV2-infected monocytes. The significance of apoptosis as a possible mechanism of lymphoid depletion in PCVD-affected pigs needs further study.

To date, studies on immune functions, specifically related to cytokine profiling, following PCV2 infection and progression towards PCVD have proven non-conclusive. Details of these studies will not be reviewed in this article but can be found elsewhere (29, 43, 44, 45, 46, 47, 48, 49, 50, 51). A recent study on cytokine profiles in blood samples from 50 pigs from 5 different litters on a PCVD-affected farm in Northern Ireland has shown no significant differences in levels of IL10, IFN gamma or IFN alpha in sequential blood samples taken over a seven week period from pigs that did or did not develop PCVD (52). Clearly the results generated to date by different workers on possible mechanisms of lymphoid depletion in PCVD-affected pigs and cytokine profiling differ considerably and further controlled studies are required.
PCVD: Control

Control without PCV2 vaccination. Sometimes it works, sometimes it does not! Until recently effective control measures for PCVD have focused on the understanding of the co-factors and triggers involved on individual farms and the control or eradication of these triggers.

Management measures and the implementation of what is today known as the Madec’s 20-point plan has significantly decreased the percentage of mortality in some severely affected farms (53). These measures were designed to reduce “infection pressure” in regard to PCV2 and any other infections, improve hygiene and to reduce stress at the different production stages (54, 55). Significant positive results have been obtained when these measures are applied and significant improvement in loss rates are achieved when the rate of compliance with the recommended measures is higher (56). However these measures have not produced satisfactory results in all situations and their application in the field is sometimes difficult or unpractical depending on the system and existing buildings that have to be worked with.

The control of concurrent viral infections in the postweaning period has also been used in an attempt to decrease the incidence of PCVD. However, in this respect, attempts to control the clinical severity of PCVD in an experimental model by the use of PPV vaccination to protect young piglets from infection with this virus were unsuccessful (57). Although it is quite clear that co-infections with PRRSV virus can make PCVD problems worse, to date, no published results are available on the control of PRRSV infection (by vaccination or other systems) to mitigate the effects of PCVD. However, it is known that experimental co-infection of pigs with PCV2 and a modified live PRRSV vaccine up-regulates PCV2 replication leading to more severe PCV2-associated histological lesions, when compared to PCV2 infection alone (58). Indeed because it has been demonstrated both experimentally and in the field that certain commercial vaccines can potentiate PCV2 replication, leading to disease, producers with PCVD-affected herds should consider determining the approximate timing of PCV2 infection, with the objective to re-schedule vaccination to minimize the disease (59).

Injection of PCV2 hyperimmune sera from slaughterhouse age pigs (serum therapy) in suckling or nursery pigs has been reported as successfully reducing mortality in several PCVD-affected farms (60, 61). The success of this procedure has been variable. The mode of action of serum-therapy has not been elucidated.

Field observations from farmers and veterinarians have suggested that certain genetic lines of pigs, specifically in relation to boar lines, are more or less susceptible to PCVD. This observation has been supported by recent experimental studies where Landrace pigs were experimentally shown to be more susceptible to develop PCVD lesions than Duroc and Large White pigs (62). Other studies have shown contradictory results with the use of Pietrain boar line; while the use of this genetic line did not seem to have any effect on the offspring in one study (63), another study showed lower general postweaning and PCVD-associated mortalities (64). Field trials on selected farms in Northern Ireland using different boar lines have also indicated a highly significant difference in mortalities due to PCVD between offspring. These findings with regard to the role of genetics in susceptibility/resistance to PCVD need to be expanded.

PCV2 vaccination. Hope for the future (at last)? PCVD is not usually observed in pigs younger than 4 weeks of age (5). This may be associated with protective maternal immunity as suggested by field and experimental studies (65, 66, 67). In contrast, other field studies have demonstrated that high levels of colostrum-derived serum antibodies to PCV2 had no significant protective effect against PCVD (68, 69) although a statistically significant relationship between the levels of colostrum-derived antibodies to PCV2 in young piglets and the time of appearance of clinical PCVD, with piglets with higher titres of maternally-derived antibodies developing disease later in life was demonstrated (69). The protective effect of maternal-derived passive immunity on PCVD development is supported by the fact that disease occurs once these titres have declined (19, 70), and as such, measures that increase maternal immunity may diminish PCVD impact on piglet mortality. An inactivated, adjuvanted PCV2 vaccine for use in sows and gilts that potentially offers protection from PCVD through passive transfer of PCV2 antibodies is now commercially available (Merial: Circovac). This vaccine has been shown experimentally and in field trials to reduce the incidence of PCVD on affected farms (71, 72) and the efficacy under extended field conditions is currently being elucidated. The vaccine is licensed for use in parts of France, Germany, Denmark and Canada and preliminary results and feedback from producers and field veterinarians are encouraging.

Other commercially available inactivated PCV2 vaccines (Fort Dodge and Intervet) are being produced for use in young pigs and are, or probably soon will be, available for use in North America and elsewhere. One of these vaccines (Fort Dodge; Suvaxyn PCV2-One Dose) is based on a chimeric infectious DNA clone containing the immunogenic ORF2 capsid gene of PCV2 cloned into the non-pathogenic PCV1 genetic backbone (73) and is designed for single dose use in 3-4 week old piglets. Several challenge studies done in Europe and the USA demonstrate that pigs vaccinated with Suvaxyn PCV2-One Dose prior to PCV2 infection show a significant decrease in viremia and histopathologic lesions vs. unvaccinated pigs. The vaccine was proven safe in USA safety trials including at least one site that was known to be PCV2 positive with gross clinical signs of PCVD (Johanne Elsener, personal communication). The PCV2 vaccine produced by Intervet is based on a baculovirus expressing PCV2 protein. To date, no information on efficacy or safety of these vaccines has been placed in the public domain, nor has it been made available for this article. However it should be noted that both these adjuvanted vaccines are to be used in young piglets and the proven potentiation of PCV2 replication following administration of some commercial vaccines to young pigs (74) should be evaluated with respect to these 2 products.
PCV2: Agent X, “genotypes”, “strains” and PCVD

PCV2 virus isolates, genotypes and strains: More confusion and misnomers! Recent retrospective epidemiology studies the UK, Denmark and New Zealand have concluded that the outbreaks of PCVD in those countries were the results of an incursion of a “new” infectious agent (Agent X) into a naive population. Following the study in the UK, it has been proposed that Agent X (probably a virus) spread slowly through Britain and also spreads slowly once on a farm (75). The agent spread by pig to pig contact and survived in the environment to be spread by humans and/or wildlife. These authors further suggested that PCV2 virus is associated with PCVD but is probably not the cause and the “risk factors” associated with a herd breaking down with PCVD included purchasing replacement gilts, closeness (3 to 5 miles) to an affected farm and permitting visitors who were not 3 days pig-free onto the farm. Similar conclusions with respect to Agent X have been drawn following a retrospective study in Denmark (76, 77). In these studies the authors concluded that recent outbreak and spread of PCVD in Denmark was consistent with what would be expected from an incursion of a “new” virus or a new highly virulent strain of PCV2 into a naive population. However, in contrast to the findings reported in the UK study (75) no risk association of disease breakdown was found in relation to the distance to other PCV2 positive herds and number of pig herds within a zone with a radius of 3 km². A retrospective epidemiology study in New Zealand into an outbreak of wasting disease in pigs in backyard farms on the North island concluded that an “exotic” agent was introduced into their country, probably in the late 1990’s in untreated pig swill, resulting in an outbreak of PCVD which was spread by contact of other farms with contaminated equipment and/or pigs. To date, it has not been determined if PCVD can be spread by feeding untreated meat products from PCV2-affected animals to pigs.

Although epidemiological studies in Brittany, France on the initial outbreak of PCVD in Europe concluded that PCV2 they did not observe any epidemic “wave” similar to what was seen in 1981 with swine influenza (H1N1) and ten years later when porcine reproductive and respiratory syndrome (PRRS) struck (Francois Madec, personal communication), certainly, in the case of the UK and Denmark the data generated from retrospective questionnaires would seem to give some support to the new Agent X hypothesis. However, the dilemma that still remains un-resolved between some epidemiological results and laboratory-based experimental studies is that clinical PCVD can be produced experimentally in colostrum-fed (78),colostrum-deprived (2) and gnotobiotic pigs (40) using PCV2 as the only infectious agent.

Recent studies in Sweden and Eastern Canada can perhaps help to square this circle. It would appear from genomic sequence data generated on PCV2 isolates from PCVD-affected and non-affected farms in Sweden that two distinct genotypes (Swedish genotypes 1 and 2) of PCV2 are now circulating in this country (79). It is of interest that, to date, Swedish genotype 1 has only been detected on farms without epizootic PCV2 and Swedish genotype 2 predominates on farms (16 of 16 farms tested) with epizootic PCV2 in Sweden. However Swedish genotype 2 virus has also been detected on 4 of 11 farms in Sweden without epizootic PMWS. Additionally, although recent experimental infection studies at our institute using Swedish genotype 2 virus did produce clinical PCVD in the inoculates the severity and extent of the disease produced was similar to that produced in the same model following experimental infection with Swedish genotype 1 virus (unpublished). Similarly, a dramatic increase in the number and severity of PCVD outbreaks in Quebec and Ontario has recently been reported. In Ontario this increase seems to have occurred at the same time as a “change” in the PCV2 isolates found in these cases. (80). Using RFLP typing, Canadian PCV2 isolates from recent cases of PCVD have been shown to be different (RFLP type 321) to PCV2 isolates found in previous years (RFLP type 422). It is not known however if these differences in RFLP patterns, which appear to fit chronologically with the onset of serious problems in that province, are truly significant in respect of diseases severity. To date, the genomic sequences of these new Canadian viruses have not been published, however genomic analysis of 4 isolates from diseased pigs from 4 different farms in Eastern Canada in our laboratory and in the laboratory of our Swedish colleagues have shown strong similarities to the Swedish genotype 2 viruses. The hypothesis that “virulent” isolates of PCV2 with distinct genotypes may exist and are associated with diseases outbreaks is not supported by other studies (81,82). In both these studies, one in The Netherlands and one in Canada, the authors failed to recognise any consistent genomic differences in PCV2 viruses recovered from pigs with and without PCVD. Indeed (82) concluded from their study on over 70 isolates of PCV2 that viruses associated with PMWS were scattered throughout the phylogenetic tree, often in groupings including PCV2 viruses identified from cases other that PCV2 such as, porcine reproductive and respiratory syndrome (PRRS), generalized tremors, porcine dermatitis and nephropathy syndrome (PDNS), arthritis, nervous signs, erysipelas and even healthy pigs. Nevertheless, recently it has been reported that when comparing the virulence of different isolates in a colostrums-fed (CF) experimental model, one of them was found to be more virulent than the other (83). Although differences in virulence between PCV2 isolates might play a role in the variability and severity of clinical presentations associated with this organism, it is important that further controlled laboratory studies are carried out in different experimental models before definitive answers can be given to this question. Subsequent to the clinical observations and genomic sequence data findings in Canada and the occurrence of new outbreaks of PCV2 on the South Island of New Zealand it has been proposed by some workers that these “new” PCV2 viruses are different “strains” of PCV2. The premature classification of the “new” PCV2 viruses into strains should be discouraged until biological differences between these viruses and “old” PCV2 viruses have been clearly demonstrated. Certainly the use of the terminology “strains of PMWS” should be avoided. It is entirely possible that biologically distinct strains of PCV2 do exist, however, to date we do not have data to support the use of this terminology “strain” in relation to different PCV2 isolates. Currently we do have PCV2 isolates, we may have distinct and conserved PCV2
PCV2 and PCVD: What we do not know

The circus comes to town! In conclusion, it is clear that a lot of questions regarding the epidemiology, pathogenic processes and control of PCVD have not all been answered and controversy still even surrounds the criteria for diagnosis of these diseases, the “causal agent” and even nomenclature to be used in describing disease outbreaks (the AASV have now changed the name porcine circovirus diseases (PCVDs) to porcine circovirus-associated diseases (PCVAD), which seems to be a not unexpected re-invention of the wheel). Hopefully answers will be provided in the coming years by international, multidisciplinary collaborative research incorporating field veterinarians and producers and it is also hoped that the PCV2 vaccines currently appearing on the international market will go some way to alleviating the losses being incurred by producers around the world due to PCVDs. Some researchers and laboratory-based diagnosticians have suggested that PCVD is no longer a major disease concern for the global pig industry. This is an assumption that needs to be challenged. Although the official figures for laboratory diagnosed outbreaks of PMWS have declined, this does not necessarily mean a decline in the incidence of the disease in the field as field veterinarians and producers are now “self-diagnosing” the disease. Additionally, it is notable that when PCVD-positive farms have “recovered” chronic losses and flare ups of disease still occurs, leading to loss of production. If you are a farmer then residual mortality, especially in the mid fattening due to underlying PCV2 circulation and PCVD combined with an increase of secondary infections, an increased use of antibiotics with poor results and heterogeneity of the pig batches, runts and low value pigs are all severely impairing your profit margin. The requirement for multidisciplinary, focused and trans-national (and hopefully trans-Atlantic) research on PCV2 infections and PCVD’s is as important as ever.

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References


