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Experimental reproduction of postweaning multisystemic wasting syndrome (PMWS) in pigs in Sweden and Denmark with a Swedish isolate of porcine circovirus type 2

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Abstract

An experimental model using 3-day-old snatch-farrowed colostrum-deprived piglets co-infected with porcine circovirus type 2 (PCV2) and porcine parvovirus (PPV) is at present one of the best methods to study factors affecting development of postweaning multisystemic wasting syndrome (PMWS). A Swedish isolate of PCV2 (S-PCV2) retrieved in 1993 from a healthy pig has been used in this model to reproduce PMWS in pigs from Northern Ireland. This virus has been present in the Swedish pig population for at least a decade without causing any known PMWS disease problems, despite its potential pathogenicity. The reasons for this are unknown, but could be related to genetics, absence of triggers for PCV2 upregulation (infectious agent and/or management forms) within Swedish pig husbandry. In order to confirm the pathogenicity of S-PCV2, Swedish and Danish pigs were experimentally infected with this isolate according to the established model. Swedish pigs were also infected with a reference isolate of PCV2 (PCV2-1010) to compare the severity of disease caused by the two isolates in Swedish pigs. Both Danish and Swedish pigs developed PMWS after the experimental infection with S-PCV2. Antibodies to PCV2 developed later and reached lower levels in serum from pigs infected with S-PCV2 than in pigs inoculated with PCV2-1010. In general, pigs infected with S-PCV2 showed more severe clinical signs of disease than pigs infected with PCV2-1010, but pigs from all PCV2-inoculated groups displayed gross and histological lesions consistent with PMWS. All pigs inoculated with PPV, alone or in combination with PCV2, displayed interleukin-10 responses in serum while only pigs infected with PPV in combination with

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PCV2 showed interferon-α in serum on repeated occasions. Thus, the pathogenicity of S-PCV2 was confirmed and a role for cytokines in the etiology of PMWS was indicated.

Keywords: PCV2; PMWS; Experimental infection; Sweden; Denmark

1. Introduction

Porcine circovirus type 2 (PCV2) is now accepted as the causal agent of postweaning multisystemic wasting syndrome (PMWS). However, it is also recognised that other additional infectious (Allan et al., 1999; Allan et al., 2000a; Krakowka et al., 2000) or non-infectious factors (Rose et al., 2003) are necessary for the full clinical expression of the disease. PMWS was first observed in high health herds in Canada in 1991 (Allan et al., 1998; Ellis et al., 1998), and has since then rapidly become a major problem in many pig-producing countries throughout the world. Retrospective studies have demonstrated that a PCV2 virus has been present in pigs for many years prior to the recognition of PMWS without being associated with any specific disease syndrome (Rodriguez-Arrioja et al., 2003; Walker et al., 2000). The global epizootic spread of PMWS since 1996 suggests that the PCV2 virus in pigs may have mutated to a more pathogenic form or that another agent in combination with PCV2 is necessary for the development of PMWS. Alternatively, it has also been suggested that the susceptibility of the host to PCV2-associated clinical disease has, in some way, changed due to alterations in the pig industry. Efforts to identify new pathogenic genotypes of PCV2 (de Boisseson et al., 2004) or new common co-infecting micro-organism (Ellis et al., 2004) have, to date, failed and epidemiological studies including numerous aspects of husbandry forms have not yet revealed any particular factor(s) that predispose for PMWS (Larochelle et al., 2003; Pogranichniy et al., 2002; Rose et al., 2003).

Dual infection with PCV2 and porcine parvovirus (PPV) or porcine reproductive and respiratory syndrome virus (PRRSV), as well as immuno-modulators, have been used to successfully reproduce PMWS experimentally in pigs (for review see Allan et al., 2004) and one of the most reproducible infection models involves co-infection with PCV2 and PPV in 3-day-old snatch-farrowed colostrum-deprived (SFCD) piglets (Allan et al., 1999). This model was recently used in Northern Ireland to demonstrate the potential pathogenicity of a Swedish isolate of PCV2 from 1993 (Allan et al., 2003). The Swedish PCV2 (S-PCV2) was isolated from a clinically healthy pig, which was raised in a SPF-herd that seroconverted to PCV2 at that time (Wattrang et al., 2002).

Sweden remained free from PMWS until December 2003, and as of October 2004, only 12 farms have been diagnosed as affected by the disease. It has been suggested that differences in pig husbandry practices, animal genetics and/or viral pathogenesis could have contributed to relative freedom of Swedish pigs from disease. The present experimental infection with S-PCV2 and PPV was conducted using Swedish and Danish pigs. To allow comparison between various PCV2 isolates, one group of Swedish pigs was also infected with a reference isolate of PCV2 (Imp. 1010). Clinical manifestations of disease, histological lesions, levels of virus antigen in affected tissues and development of antibodies to PCV2 were recorded and the IL-10 and interferon-α responses were determined in serum obtained from Swedish pigs.

2. Materials and methods

2.1. Experimental model and virus

An experimental model, using a dual infection with porcine circovirus type 2 (PCV2) and porcine parvovirus (PPV) for induction of PMWS was used as previously described (Allan et al., 1999). Two isolates of PCV2 were used: PCV2-1010 (PCV2 Stoon) isolated from an outbreak of PMWS in a high health herd in Canada (Ellis et al., 1998) and S-PCV2 isolated from a lymph node collected in 1993 from a clinically healthy pig reared in a Swedish SPF-herd (Allan et al., 2003; Wattrang et al., 2002). For co-infection, PPV (isolate 1005 pool 7) recovered from