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Induction of porcine post-weaning multisystemic wasting syndrome (PMWS) in pigs from PMWS unaffected herds following mingling with pigs from PMWS-affected herds

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ABSTRACT

In this paper we present the results from two experimental studies (I and II) investigating whether post-weaning multisystemic wasting syndrome (PMWS) can be induced in pigs from PMWS unaffected herds by mingling with pigs from PMWS-affected herds and to observe whether transportation and/or mingling of healthy pigs from unaffected herds could induce PMWS.

The studies comprised pigs from 12 different herds. Eight herds had PMWS while four were unaffected. All 12 herds were found to be infected with PCV2. Pigs from PMWS-affected herds were mingled with pigs from unaffected herds in four separate compartments in both study I and study II. In addition, in study II, four groups of pigs from unaffected herds were included. Two groups with pigs transported and mingled from unaffected herds and two groups with pigs which were only transported. The PMWS diagnoses on the individual pigs were based on lymphoid depletion, histiocytic proliferation and the presence of giant cells or inclusion bodies together with the demonstration of PCV2 in lymphoid tissue.

Healthy pigs, in both studies, developed PMWS 4–5 weeks after mingling with pigs clinically affected with PMWS. None of the pigs from unaffected herds which had no contact with pigs from PMWS-affected herds developed clinical signs of PMWS. Transportation and mingling of pigs from PMWS unaffected herds in combination or alone was insufficient to provoke PMWS.

1. Introduction

Post-weaning multisystemic wasting syndrome (PMWS) is an important disease in weaned pigs worldwide. PMWS was first described in Canada in 1991 as a chronic disease with progressive weight loss in pigs from 4 to 16 weeks of age (Harding and Clark, 1997). Since then, the disease has been diagnosed in many countries in North America, Europe and Asia (Allan and Ellis, 2000), and in Denmark since 2000 (Hassing et al., 2002). The clinical signs of PMWS comprise unthriftness/wasting, paleness of the skin, enlarged lymph nodes and occasionally jaundice, respiratory symptoms or diarrhoea (Harding and Clark, 1997; Sorden, 2000; Ladekjær-Mikkelsen et al., 2002). Affected animals have lesions in lymphoid organs characterized by lymphoid depletion and the presence of giant cells and inclusion bodies (Allan et al., 1998; Ellis et al., 1999; Ladekjær-Mikkelsen et al., 2002; Segalés et al.,

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Table 1
Distribution of pigs at the research facilities.

Study	Research facility	Compartment	Pigs from PMWS-affected herds		Pigs from non-affected herds	
			Herd	Number	Herd	Number
I	I	1	A	28	1	27
I	I	2	B	27	1	29
I	I	3	C	27	2	27
I	I	4	D	29	2	29
II	I	1	E	27	3	54
II	I	2	F	27	3	54
II	I	3	G	27	4	54
II	I	4	H	27	4	54
II	I	5			3 and 4	9 + 9
II	II	6			3	18
II	II	7			3 and 4	9 + 9
II	II	8			4	18

2004). PCV2 has proved to be necessary but not sufficient for development of PMWS, since the virus is present in both affected and unaffected pigs and herds (Allan et al., 1999; Ladekjær-Mikkelsen et al., 2002).

The PCV2 virus is probably transmitted between pigs by the oro-fecal and/or respiratory routes (Caprioli et al., 2006) and vertical transmission has also been documented (West et al., 1999; Ladekjær-Mikkelsen et al., 2001). The high prevalence of PCV2 in almost all herds of all pig producing countries indicates that the transmission of PCV2 is very effective (Rose et al., 2003). In contrast, only a few studies have been performed on the "transmission" of the PCV2 associated disease complexes (PCVDs), i.e., whether PMWS can be "transmitted" from affected to unaffected pigs. A study performed in New Zealand demonstrated disease development in healthy pigs in direct or indirect contact with PMWS-affected pigs when they were mingled at 4 weeks of age but not when they were mingled at 12 weeks of age (Jaros et al., 2006). The purpose of the present studies was to confirm if PCV2 positive pigs from PMWS unaffected herds can develop PMWS following mingling with pigs from PMWS-affected herds. Control groups were included to exclude the possibility that transportation or mingling by itself could induce PMWS.

2. Materials and methods

Two very similar studies were performed as detailed in Table 1. Study I was performed by mingling pigs from PMWS-affected and unaffected herds in four different compartments in research facility I. Study II was performed with the same basic experimental setup as in study I. However, in addition, it included four groups of pigs from unaffected farms that were either transported or both transported and mingled but remained free from contact with pigs from PMWS-affected herds.

2.1. Pigs

Pigs were obtained from 12 different herds (Table 2). Serological screening showed that all 12 herds had antibodies against PCV2. All the herds were also seropositive for *Mycoplasma hyopneumoniae* and porcine reproductive and respiratory syndrome virus (PRRSV) according to the owner's information. Herds G, H and 4 were additionally found to be infected with toxigenic *Pasteurella multocida*. Vaccine against porcine parvovirus was used in sows from all 12 herds and pigs in herds G, H, and 4 were vaccinated against toxigenic *Pasteurella multocida*. None of the herds vaccinated against PCV2. Eight of the herds were

Table 2
Characteristics of the PMWS-affected herds (A, B, C, D) and the non-affected herds (1, 2, 3, 4) delivering pigs to the studies.

Herd	Number of sows	Pigs sold at	Post-weaning mortality in the herds at onset of the study	Post-weaning mortality in the herds during the study and 3 months later
A	185	Slaughter	30%	10–15%
B	200	30 kg	10–15%	10–15%
C	330	30 kg	8–10%	8–10%
D	386	30 kg	18%	13%
E	720	30 kg	6–7%	2.3%
F	340	Slaughter	5–6%	5–6%
G	910	Slaughter	10–12%	10–12%
H	420	Slaughter	15%	15%
1	150	30 kg	3%	3%
2	225	30 kg	Below 1%	Below 1%
3	1050	30 kg	2.6%	2%
4	400	30 kg	1.7%	1.2%