Clinical and virological outcome of experimental PCV2a and PCV2b infections in mid-gestational porcine foetuses

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Introduction

PCV2 may cause reproductive failure in the field (1), and therefore the effects of experimental PCV2 infections, mainly PCV2a, on reproduction have been studied extensively (2). Recent field observations on the occurrence of PMWS suggest that PCV2b may be more pathogenic than PCV2a (3). The aims of the present study were to investigate if there are differences in viral replication and clinical outcome between PCV2a and PCV2b infections in porcine foetuses.

Materials and Methods

Four conventional sows were submitted to laparotomy at 55 days of gestation. In each of four sows, three foetuses were inoculated with PCV2 and one foetus was mock inoculated. Inoculations were performed by transuterine injection with 10^4.3TCID50 of PCV2 into the peritoneal and amniotic cavities of the foetuses (4). In the first sow, two foetuses were inoculated with PCV2a strain Stoon-1010 and one with PCV2a strain 1121. In the second sow, two foetuses were inoculated with PCV2b strain 48285 and one with PCV2b strain 1147. In the third sow, one foetus was inoculated with PCV2a strain Stoon-1010, one with PCV2a strain 1121 and one with PCV2b strain 1147. In the fourth sow, one foetus was inoculated with PCV2a strain 1121, one with PCV2a strain 1121, one with PCV2b strain 48285 and one with PCV2b strain 1147. Twenty-one days post inoculation (dpi), the sows were euthanized and all foetuses were collected. All foetuses were examined for gross lesions and tissue samples from various organs were taken for titration on PK-15 cells and for localization of viral antigens. In order to verify that the foetuses had been inoculated with the specific PCV2 strain, capsid gene was amplified using heart and/or lung tissue from infected foetuses followed by sequencing. Abdominal fluids were tested for the presence of PCV2-specific antibodies by IPMA.

Results

All PCV2-inoculated foetuses, except one PCV2 (1147)-inoculated foetus, were oedematous and had distended abdomens (Fig.1.a). Haemorrhages and congestion in internal organs and liver enlargement were observed (Fig.1.b). One PCV2 (1147)-inoculated foetus had a normal external appearance, but liver enlargement and generalized lymph node enlargement were observed. Gross pathological lesions were not observed in mock-inoculated and non-inoculated foetuses. High PCV2 titres (> 10^4.3TCID50 / g tissue) were found in all PCV2-inoculated foetuses, especially in the heart, spleen and liver. High numbers of PCV2-infected cells (> 1,000 infected cells / 10 mm² tissue) were observed in the heart (Fig.1.c). PCR and DNA sequencing of the capsid gene recovered pure PCV2a and pure PCV2b sequences from PCV2a- and PCV2b-inoculated foetuses, respectively. No evidence for mixed samples (e.g. containing more than one PCV2 strain) was seen. All adjacent foetuses of PCV2-inoculated foetuses were negative in PCR assays with the exception of one adjacent foetus of Stoon-1010-inoculated foetus, which was positive in PCR assays and a pure Stoon-1010 sequence was obtained. All mock-inoculated foetuses and their adjacent foetuses were negative in PCR assays. Three PCV2-inoculated foetuses had a low anti-PCV2 Ab titre of 10 to 40.

Conclusions

From this model, it can be concluded that different PCV2 strains of both genotypes induced similar gross pathological lesions and replicated to similar high titres in different foetal organs.

References


Fig. 1. Different aspects of PCV2 replication after inoculation of a 55-day old foetus. a) Subcutaneous oedema and abdominal distension. b) Haemorrhages and congestion in internal organs and liver enlargement. c) Immunofluorescent staining for the PCV2 capsid protein in the heart. Bar = 50 μm.