An investigation of the aetiology and natural transmission of postweaning multisystemic wasting syndrome in pigs

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Abstract

Postweaning multisystemic wasting syndrome (PMWS) is a wasting disease primarily affecting weaned pigs. The disease causes significant production and financial losses through increased mortality rates and reduced daily weight gain. The aetiology is controversial although reports commonly suggest that PMWS is associated with the presence of porcine circovirus type 2 (PCV2) with disease expression modified by a range of infectious and non-infectious factors. While PCV2 infection in New Zealand is ubiquitous, PMWS has behaved as a propagating epidemic since its first incursion beginning in about 1999. The initial outbreak of PMWS in New Zealand was limited to a small cluster of farms near Auckland, which were epidemiologically linked to a possible entry mechanism.

A transmission study was conducted in 2005 to critically evaluate alternative hypotheses which have been proposed for the causation and epidemiology of PMWS. The study set out to investigate the natural transmission of PMWS by direct contact between PMWS-affected and susceptible pigs, while managing the influence of proposed co-factors. Six different groups, comprised of pigs from PCV2-negative and positive herds were directly exposed to possible PMWS agents at 4 and 12-weeks-of-age and compared with two groups of unexposed pigs. All experimental groups were observed daily for 8 weeks or longer and evaluated clinically and pathologically.

After exposure to PMWS-affected pigs, disease characterised by wasting, dyspnoea and high case fatality rates occurred in both PCV2-positive and PCV2-negative pigs of four-weeks-of-age, but not in pigs older than 12 weeks. Histopathological lesions found in experimental groups with clinical cases were consistent with those previously reported for PMWS. A range of infectious pathogens proposed to have a modifying influence on PCV2 and to contribute to disease causation were absent as determined by molecular and serological test methods. In addition, there was not sufficient molecular evidence to explain the genomic difference between PCV2 isolates from healthy and PMWS-

affected pigs. Taking this, and supporting evidence from the other experimental groups into account, the findings of this study strongly support the conclusion that a transmissible agent other than PCV2 is involved in the causality of PMWS.

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