Attachment C

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FAX TRANSMISSION COVER SHEET

Date:	***February 4, 2004***
To:	**William F Hall, Research and Innovation Division, Australian Pork Ltd.** **PO Box 148, Deakin West ACT 2600, Australia**
Fax:	**0061 2 6285 2288**
Subject:	**Epidemiology of Post-Weaning Multi-systemic Wasting Syndrome in Pigs**
Sender:	***Professor Roger S Morris***

1 Page(s) including cover sheet

Dear Bill,

The following is my current scientific assessment of the state of knowledge concerning the causal factors involved in post-weaning multi-systemic wasting disease of pigs (PMWS).

I am a registered veterinary specialist in both pig medicine and epidemiology, and am Scientific Adviser to the New Zealand Pork Industry Board. I have been closely following research reports and field experience in relation to PMWS for the last decade, both because it was a new disease of importance, and because its rapid international spread raised concerns for New Zealand producers.

I found increasing difficulty reconciling the field epidemiological findings with research reports attributing the disease to porcine circovirus type 2 (PCV2) as the primary causal agent. In 2002 I presented the opening plenary paper at the International Pig Veterinary Congress on "Evolution of Diseases in the World's Pork Industry", and in that presentation I challenged the audience to reassess the situation with respect to this disease because the laboratory research results did not adequately explain the behaviour of the disease. I suggested that we needed to apply epidemiological investigation methods to clarifying the true field situation so that causal factors involved in the disease would become clear.

At the time (exactly as I intended), my comments were seen as challenging to the conventional wisdom, and there was some lively discussion with many people among the 2,000 at the Congress. I did not know at the time that we were shortly to have the opportunity to apply my proposed approach to the disease in New Zealand! Subsequently the growing body of epidemiological experience with PMWS in Europe and now in New Zealand has made the view that PCV2 is the primary causal agent of the disease very much more difficult to sustain, and there is rapidly growing acceptance even among former strong proponents of the PCV2 hypothesis that there is another underlying primary causal agent which initiates the disease, and stimulates the massive amplification of PCV2 virus in cells, which then kills the pigs. We currently call this "Agent X", since it has not yet been identified. It is expected that it will prove to be a virus, and must be one which is technically difficult to isolate, since strenuous efforts in many countries have so far failed to find it. There are various previous examples of such problems, such as rabbit haemorrhagic disease virus, which despite many years of research remains impossible to isolate in tissue culture, and must still be grown in rabbits.

A fundamental difficulty with the view that PCV2 is the primary cause of the disease is that this agent can be found in almost all pig herds throughout the world (including Australia), and virtually all pigs become infected with this virus after weaning, yet PMWS as a disease has behaved as a propagating epidemic which has moved between countries (in recent months Norway, Sweden and New Zealand have all reported their first cases), and has spread progressively within newly infected countries. Biosecurity measures within herds have provided valuable (though not perfect) protection against its introduction to herds, and depopulation/repopulation of herds has eliminated the disease despite the fact that the re-established herds were infected with PCV2 virus.

In New Zealand my staff member Dr David Lawton identified PMWS in 2003 as a possible explanation for a herd with high weaner mortality, and this diagnosis has now been confirmed. Detailed investigation of the national situation has so far found evidence of the disease only in a small cluster of inter-linked farms in the area around Auckland which are an unusual sub-group of the New Zealand industry. These are small herds which do not follow the strict biosecurity practices used in the main commercial industry, and exchange feed, pigs and equipment among their group, who are known to each other. The main feed source for these producers is discarded food items from the human food supply. In 1999 the requirement for cooking such material before feeding it to pigs was removed, and in 2001 a requirement for cooking imported pig meat on arrival in New Zealand was introduced. Between those dates appears from the evidence to be when the disease first developed in New Zealand, and circumstantial support has been accumulated through our investigations for the hypothesis that some of the farmers concerned used feed materials during this period which are likely to have contained uncooked or inadequately cooked pig meat of imported origin.

Conclusion

Therefore our experience in New Zealand is consistent with the body of overseas field evidence, that while the presence of PCV2 virus in a herd appears to be necessary for expression of this disease, a second as yet unidentified disease agent is the best available

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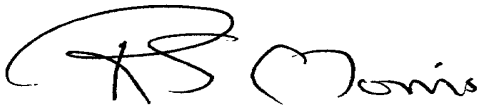
explanation for the development of PMWS and its spread through pig herds between and within countries.

Yours sincerely



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