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

A thesis presented in partial fulfilment of the requirements for the degree
of

Master of Veterinary Studies
in
Epidemiology

at Massey University, Palmerston North,
New Zealand.

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September, 2007

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.

Acknowledgements

In February 2005, when I commenced my Master of Veterinary Studies at Massey University, it was the beginning of an enjoyable time at the EpiCentre, loaded with numerous rewarding challenges, fulfilling experiences, and valuable companionship. There are a number of people to whom I express my gratitude for their considerable help and contribution to this comprehensive research project and thesis.

I express my gratitude to my supervisors Eric Neumann, Lachlan McIntyre and Prof. Roger Morris for their knowledge, inputs and guidance during the study and writing of my thesis, Dr. Naomi Cogger for her contribution to the statistical analysis of data, and Prof. Alastair Johnstone (Pathobiology, IVABS) for his passionate interest and histopathological investigation of all study animals.

I gratefully acknowledge Sandy Ferguson (Living Cell Technologies Ltd (LCT), Auckland), for the arrangement of the Auckland Island pigs used in the transmission study. I greatly appreciated the opportunity and trust of LCT to work with these, indeed unique, pigs. Special acknowledgements go to Olga Garkavenko and Divya Nathu (LTC) for their molecular analysis of numerous samples and to Ross Fraser in Invercargill for delivering the first group of his dear pigs and giving valuable advice on our outdoor research facility at Massey University.

This labour-intensive transmission study would have been impossible to conduct without the help of several EpiCentre colleagues and staff members. I am indebted to Solis Norton for spending time in building a fantastic ‘escape-safe’ outdoor shelter for the study pigs; Karma Rinzin, Sithar Dorjee, Kathy Goodwin-Ray, Birgit Schauer, Jackie Benschop, Ian Langstaff, Deb Prattley, Jutta Tebje-Kelly, Esther Richardson, Jo O’Leary, and Colleen Blair for their invaluable help on the study sites and/or in the not so enjoyable post mortem room for necropsies. Each spare hand was highly appreciated! I would also like to acknowledge our computer programmers Simon

Verschaffelt, Masood Sujau Maumoon, and Greg Bolton for simplifying the process of data entry for me otherwise I would still be typing numbers! Many thanks also to Julie Dunlop for issuing all the material orders and dealing with the administrative ‘extras’ of the project.

Diane Richardson deserves special appreciation. I could not have asked for a more enthusiastic, caring and calm veterinary technician. Her daily assistance at the study sites was so precious to me during the months of intensive work with the pigs and we also managed to have good fun together despite all the work.

I would especially like to thank Kathy Goodwin-Ray and Jaimie Glossop for proof reading my thesis chapters and correcting my ‘Swinglish’ (Swiss English). My sincere thanks also to people of other institutes at Massey for their technical assistance throughout my research project: Mike Hogan, Evelyn Lupton and Mary Gaddam from Pathobiology (IVABS); Errol Kwan and Jim Learmonth (Hopkirk Research Institute), for introducing me to molecular laboratory techniques; all staff of Jennersmead farm in Bunnythorpe; and last but not least, staff of the Ratanui pig farm in Feilding for their assistance with collecting samples.

I would like to acknowledge the support of New Zealand Pork Industry Board (NZPIB), Australian Pork Limited (APL) and the EpiCentre, Massey University, who generously funded the project.

Most of all I would like to express my special thanks and love to my parents in Switzerland and dear Bryce for continued moral support, encouragement and love throughout my studies in New Zealand.

Contents

Abstract.....	iii
Acknowledgements.....	v
List of Figures.....	xi
List of Tables	xiii
Chapter 1 Introduction.....	1
Chapter 2 Literature review	7
2.1 Introduction.....	7
2.2 History.....	7
2.2.1 Detection of PMWS	7
2.2.2 PCV virus as a causal agent	8
2.2.3 Differentiation between PCV types	9
2.2.4 The origin of PMWS	10
2.3 Epidemiology	10
2.3.1 Relationship between PCV2 and PMWS prevalence.....	10
2.3.2 Prevalence of PCV2 at farm, national and international level.....	11
2.3.3 Introduction of a novel agent	13
2.4 Case definition of PMWS	16
2.4.1 General definition of PMWS	16
2.4.2 International variations of PMWS definition.....	17
2.5 Clinical diagnosis of PMWS.....	21
2.5.1 Differential diagnoses for PMWS.....	21
2.5.2 Clinical indications of PMWS	22
2.5.3 Mortality rates	23
2.5.4 Infections coinciding with PMWS	23

2.5.5	Duration of PMWS outbreaks.....	24
2.6	Pathology of PMWS	24
2.6.1	Macroscopic findings.....	24
2.6.2	Microscopic findings	25
2.6.3	Virus detection.....	27
2.6.4	Antibody detection.....	28
2.6.5	Diagnostic issues.....	28
2.7	Investigation of the disease process.....	29
2.7.1	Hypotheses about aetiology of PMWS.....	29
2.7.2	Pathogenesis of PMWS.....	35
2.7.3	Prevention and control strategies for PMWS.....	38
2.8	Economic effects of PMWS.....	40
2.9	Conclusions.....	41

Chapter 3 An investigation into the natural transmission of postweaning multisystemic wasting syndrome and the clinical course of disease..... 43

3.1	Introduction.....	43
3.2	Materials and Methods.....	47
3.2.1	Study design.....	47
3.2.2	Source farms	47
3.2.3	Animals.....	48
3.2.4	Housing, feeding and biosecurity measures.....	49
3.2.5	Medical treatments.....	52
3.2.6	Clinical observations.....	53
3.2.7	Definition of clinical cases of PMWS.....	53
3.2.8	Sampling	53
3.2.9	Termination.....	55
3.2.10	Statistical analysis.....	55
3.3	Results.....	56
3.3.1	Clinical outcomes.....	56
3.3.2	Relationship between clinical cases, age and breed.....	65
3.4	Discussion	65
3.5	Conclusions.....	68

Chapter 4 Pathological and microbiological findings of a natural transmission study on postweaning multisystemic wasting syndrome in pigs	69
4.1 Introduction	69
4.2 Materials and methods	71
4.2.1 Case definition of PMWS	71
4.2.2 Experimental design	72
4.2.3 Necropsy and histopathology	76
4.2.4 <i>In situ</i> hybridisation	77
4.2.5 Bacteriology	77
4.2.6 Haematology	77
4.2.7 Serology	78
4.2.8 Polymerase chain reaction	80
4.2.9 Data management and analysis	82
4.3 Results	83
4.3.1 Macroscopic findings	83
4.3.2 Microscopic findings	87
4.3.3 Detection of PCV2 by the <i>in situ</i> hybridisation method	103
4.3.4 Bacteriology	107
4.3.5 Haematology	107
4.3.6 Serology	111
4.3.7 PCR analysis for viral pathogens	114
4.4 Discussion	118
4.5 Conclusions	128
Chapter 5 Genetic characterisation of porcine circovirus type 2 isolates from a natural transmission study on postweaning multisystemic wasting syndrome in pigs	129
5.1 Introduction	129
5.2 Materials and methods	130
5.2.1 Samples	130
5.2.2 Isolation of DNA from tissues	132
5.2.3 PCR amplification of complete PCV2 genome	132
5.2.4 Sequencing, sequence alignments and cluster analysis	133
5.3 Results	135

5.3.1	Electrophoretic band pattern	135
5.3.2	Alignments and phenetic analyses of PCV2 isolates	136
5.4	Discussion	141
5.5	Conclusions	143
Chapter 6	General discussion	145
Bibliography	151
Appendices	175

List of Figures

3.1: Study site 1 for the investigation of the natural transmission of PMWS.....	50
3.2: Study site 2 for the investigation of the natural transmission of PMWS.....	52
3.3: Average rectal temperatures of experimental groups of pigs	57
3.4: Average body condition scores in experimental groups of pigs.....	58
3.5: Photographs of pigs from Group 1 prior to exposure to PMWS-affected pigs and 18 days post exposure	58
3.6: Kaplan-Meier survival curves of experimental groups of pigs.....	59
3.7: Photographs of pigs from Group 2 prior to exposure to PMWS-affected pigs and 53 days post exposure	60
3.8: Photographs of pigs from Group 3 prior to exposure to PMWS-affected pigs and a PMWS infected animal at day 30 post exposure	61
3.9: Photographs of pigs from Group 4 prior to exposure to PMWS-affected pigs and 54 days post exposure	62
3.10: Photographs of pigs from Group 5 at the beginning of the transmission study and with visible signs of scruffy hair coats at day 36.....	63
3.11: Photographs of pigs from Group 7a prior to exposure to a PCV2-positive pig and faeces collected at Farm B and with no signs of wasting or PMWS disease 81 days post exposure to PCV2.....	65
4.1: Photographs of A: Enlarged mesenteric lymph nodes from a pig of Group 5 with wasting symptoms and B: Non-collapsed lungs from a study animal within Group 2 which presented clinical signs of severe dyspnoea.....	86
4.2: Microscopic slides of lymph node sections from pigs of Group 1 with typical clinical signs of PMWS. A: Recognisable follicular structure and B: Loss of follicular structure	90
4.3: Microscopic slide of a follicular centre of a lymph node section from a PMWS-affected pig of Group 1. Infiltration of large mononuclear and multinucleated histiocytes.....	91

4.4: Microscopic slide of a lymph node section from a PMWS-affected pig in Group 1. Presentation of large numbers of botryoid amphophilic intracytoplasmic inclusion bodies within histiocytes.....	91
4.5: Microscopic slides of a lung section from a pig of Group 1, affected by PMWS. Broncho-interstitial pneumonia with thickening of alveolar septa and histiocyte infiltration in peribronchial lymphoid tissue.....	92
4.6: Microscopic slides of a lung section from a pig in Group 3 with severe granulomatous interstitial pneumonia and infiltration of macrophages and other mononuclear leukocytes.....	94
4.7: Microscopic slides of a section of liver tissue from a pig of Group 3 with severe diffuse hydropic degeneration.....	95
4.8: Microscopic slide of a section of kidney tissue from a pig of Group 5 with focally extensive non-suppurative interstitial nephritis	97
4.9: Microscopic slides of representative lymphnode changes of Group 6 control pigs	98
4.10: Microscopic slides of a lymph node section of a Group 1 animal clinically affected by PMWS. Detection of PCV2 nucleic acid by <i>in situ</i> hybridisation.....	104
4.11: Ratios of mean neutrophils to lymphocytes percentage in blood samples of experimental groups of pigs in a natural transmission study on PMWS, stratified by sampling day (0, 8, 11, and 29 days post exposure)	108
4.12: Ratios of mean neutrophils to lymphocytes percentage in blood samples of Groups 7a and 7b during the transmission study on PMWS	111
4.13: Boxplots of percentage inhibition results from a commercial PCV2 ELISA test assessing PCV2 antibodies in pig sera from a natural transmission study on PMWS	112
5.1: Schematic outline of the complete circular genome of PCV2 with its capsid protein region and replication protein region.....	133
5.2: Gel electrophoresis of amplified fragments of PCV2 DNA detected in one sample	135
5.3: Comparative amino acid alignment of the viral capsid protein of 16 PCV2 isolates	139
5.4: Cluster analysis of 16 full-length PCV2 genomes selected from pigs from a transmission study on PMWS, acutely infected pigs from commercial farms in New Zealand and comparable isolates available from the GenBank	140

List of Tables

2.1: Publication details of ‘first case’ reports of PMWS for several countries.....	13
2.2: Major viral and bacterial diseases with comparable clinical signs to PMWS	22
2.3: Typical macroscopic lesions of PMWS	25
3.1: Disease status of source farms which supplied pigs used to investigate the natural transmission of PMWS	48
3.2: Study design of the natural transmission study on PMWS.....	49
3.3: Summary of sampling days per experimental group stratified by collected serum, peripheral blood mononuclear cells, and whole blood samples.....	54
4.1: Summary of experimental groups of the transmission study on PMWS	73
4.2: Summary of clinical PMWS cases and case fatalities, stratified by experimental group	75
4.3: Blood sampling days of experimental groups.....	78
4.4: Tissue samples of experimental groups and references of the virus-specific primers used in nested PCR, stratified by DNA and RNA viruses	81
4.5: Summary of macroscopic findings	84
4.6: Summary of microscopic findings in examined tissues.....	88
4.7: Associations between body condition score categories 0 and 1 and the presence of microscopic lesions in various lymphoid tissues of pigs	101
4.8: Frequency of study animals affected with clinical signs typical of PMWS and/or positive to <i>in situ</i> hybridisation technique, stratified by experimental group.....	105
4.9: Results of <i>in situ</i> hybridisation, stratified by tissue type and experimental group of pigs	106
4.10: Frequency of viral antibodies detected in porcine sera on day 0 and post mortem	114
4.11: Frequency of viral DNA detected in faeces, tonsil tissue, and peripheral blood mononuclear cells of study pigs on day 0 and at post mortem using polymerase chain reaction method	115
4.12: Frequency of viral RNA detected in faeces of study pigs on day 0 and at post mortem using reverse transcription-polymerase chain reaction method.....	116

4.13: Herd profile of viral pathogens tested within a commercial pig farm from which healthy, PCV2-positive, 4 and 12-week-old pigs were sourced for the natural transmission study on PMWS	118
5.1: Summary of selected tissues for complete sequencing of the PCV2 genome	131
5.2: Identification of PCV2 isolates retrieved from the GenBank database	134
5.3: Pairwise comparison of complete PCV2 genomes and amino acid sequences of the capsid protein (ORF2).....	137