Manipulating Pig Production X

Proceedings of the Tenth Biennial Conference of the
Australasian Pig Science Association (Inc.) (APSA)
held in Christchurch, New Zealand
on November 27th to 30th, 2005

Editor: J.E. Paterson

AUSTRALASIAN PIG SCIENCE ASSOCIATION (INC.)
Werribee, Victoria, Australia

© Copyright 2005 by the Australasian Pig Science Association (Inc.) Victorian Institute of Animal Science, Werribee, Victoria 3030, Australia.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, electrostatic, magnetic tape, mechanical, photocopying, recording or otherwise, without the prior written permission of the copyright owner. Authorisation to photocopy limited amounts of this book for personal use only, is granted by the Australasian Pig Science Association.

Australasian Pig Science Association (Inc.) Conference
10th; 2005: Christchurch, New Zealand.
Manipulating Pig Production X

Includes bibiolgraphies and index
ISBN 978-0-9806880-4-7
ISSN: 1324-9177

Printed by OptimaPress, Perth, Western Australia

CONTENTS

A.C. DUNKIN MEMORIAL LECTURE

CARCASS QUALITY, MEAT QUALITY, GROWTH AND DEVELOPMENT

ONE PAGE PAPERS

HEALTH AND ANTIMICROBIAL RESISTANCE

SYMPOSIUM I - HEALTH STRATEGIES FOR THE MODERN PORK INDUSTRY

ONE PAGE PAPERS

NUTRITION, METABOLISM AND GENETICS

REVIEW I

ONE PAGE PAPERS

GENE MARKERS AND FEED EVALUATION

REVIEW II

ONE PAGE PAPERS

AIR QUALITY, DIETARY FIBRE AND FEED QUALTY

REVIEW III

REVIEW IV

ONE PAGE PAPERS

NUTRITION, FEED ADDITIVES, PRODUCTIVITY AND REPRODUCTION

ONE PAGE PAPERS

REPRODUCTION

SYMPOSIUM II - GILT MANAGEMENT FOR LIFETIME PERFORMANCE

ONE PAGE PAPERS

HOUSING SYSTEMS, NUTRITION AND FEED ADDITIVES

REVIEW IV

ONE PAGE PAPERS

MYCOTOXINS, ENVIRONMENT AND FEED ADDITIVES

REVIEW V

ONE PAGE PAPERS

FUNCTIONAL FOODS, MEAT QUALITY AND FEED EVALUATION

REVIEW VI

ONE PAGE PAPERS

CONTRIBUTORS

Alali, W.Q.	Department of Veterinary Integrated Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843 USA.
Aldenhoven, J.	Reprogen, Faculty of Veterinary Science, University of Sydney, NSW, 2006, Australia.
Anderson, R.C.	Food and Feed Safety Research Unit, Agricultural Research Service, USDA, 2881 F&B Road, College Station, TX 77845 USA.
Awati, A.	Animal Nutrition Group, Wageningen Institute of Animal Sciences (WIAS), Wageningen University, PO Box 338, 6700 AH Wageningen, The Netherlands.
Banhazi, T.	University of Adelaide, SA, 5371, Australia.
Barker, L.J.	Department of Primary Industries, Camden, NSW 2570
Barnett, J.L.	Animal Welfare Science Centre, Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Barton, M.D.	NSW Department of Primary Industries, Wollangbar, NSW 2477 Australia.
Baud, S.R.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Bean, A.G.D.	CSIRO Australian Animal Health Laboratory, Livestock Industries, Geelong, Vic. 3219, Australia.
Beltranena, E.	Prairie Swine Centre Inc., Saskatoon, Saskatchewan, S7H 5N9 Canada.
Binder, E.M.	Erber AG, Herzogenburg, Austria.
Boisen, S.	Danish Institute of Agricultural Sciences, Department of Animal Health, Welfare and Nutrition, P.O. Box 50, DK-8830 Tjele, Denmark.
Borg, S.S.	Animal Welfare Science Centre, Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Braun, J.A.	Myora Farm, Mount Gambier, SA. 5290, Australia.
Briscoe, S.J.	CSIRO Livestock Industries, Queensland Biosciences Precinct, St Lucia, Qld., 4067, Australia.
Broadway, M.M.	Australian Animal Health Laboratory, CSIRO Livestock Industries, Geelong, Vic. 3219, Australia.
Brumm, M.C.	University of Nebraska Haskell Agricultural Laboratory, Concord, NE 68728, USA.
Cadogan, D.J.	Feedworks, Eagle Farm, Qld. 4009, Australia.
Callesen, J.	Danish Bacon and Meat Council, Axeltorv 3, DK-1609, Copenhagen, Denmark.
Cauduro, J.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Cavaney, F.S.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA, 6150, Australia.
Channon, H.A.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Chapman, T.	Immunology Molecular Diagnostic Research Unit, Elizabeth Macarthur Agricultural Institute, Camden, NSW, 2570, Australia.
Chen, S.	Alltech Asia-Pacific Bioscience Center, Beijing 101407.
Chin, J.C.	Immunology Molecular Diagnostic Research Unit, Elizabeth Macarthur Agricultural Institute, PMB 8, Camden, NSW, 2570, Australia.
Choct, M.	Australian Poultry CRC, University of New England, Armidale, NSW, 2351, Australia.
Christensen, T.B.	Danish Institute of Agricultural Sciences, Department of Animal Health, Welfare and Nutrition, Denmark.
Clark, P.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA,6150, Australia.
Close, W.H.	Close Consultancy Ltd, Wokingham RG41 2RS, UK.
Cogger, N.	EpiCentre, Massey University, Private Bag 11222, Palmerston North, New Zealand.
Colgan, S.	University of Nebraska Haskell Agricultural Laboratory, Concord, NE 68728, USA.

Collins, C.L.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Corrigan, V.K.	New Zealand Institute for Crop & Food Research, Food Industry Science Centre, Palmerston North, New Zealand.
Cox, M.L.	Department of Primary Industries, 600 Sneydes Road, Werribee, 3030, Australia.
Cronin, G.M.	Animal Welfare Science Centre, Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Crump, R.E.	Animal Genetics and Breeding Unit, The University of New England, Armidale NSW, 2350, Australia.
Cui, Q.	Institute of Animal Nutrition, Sichuan Agricultural University, Yaan, China 625014.
Cumarasamy, S.	New Zealand Institute for Crop & Food Research, Food Industry Science Centre, Palmerston North, New Zealand.
Curtis, M.A.	Elanco Animal Health, Macquarie Park NSW, 2113, Australia.
D'Souza, D.N.	Alltech Biotechnology Pty Ltd, Dandenong South, Vic. 3175, Australia.
Davidson, A.A.	New Zealand Pork Industry Board, Wellington, New Zealand.
Diaz, G.	Department of Animal and Poultry Science, University of Guelph, Guelph, Ontario, Canada.
Distl, O.	Institute for Animal Breeding and Genetics, University of Veterinary Medicine, Hannover, Germany.
Driesen, S.J.	Department of Primary Industries, Epsom, Vic. 3551, Australia.
Dunshea, F.R.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Eagling, D.R.	Department of Primary Industries, Knoxfield, Vic. 3176, Australia.
Eamens, G.J.	NSW Department of Primary Industries, EMAI, PMB 8, Camden, NSW, 2570, Australia.
Eason, P.J.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Edmunds, B.L.	University of Western Australia, Nedlands, WA, 6009, Australia.
Edwards, A.C.	ACE Consulting, Cockatoo Valley, SA. 5440, Australia.
Evans, M.	Animal Science, Department of Primary Industries & Fisheries, P O Box 327, Cleveland, Qld. 4163, Australia.
Fahy, V.A.	Department of Primary Industries, Epsom, Vic. 3551, Australia.
Ferrari, J.M.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Gabler, N.K.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Gannon, N.J.	Ridley AgriProducts, Toowoomba, Qld. 4352, Australia.
Gauthier, R.	Jefo Nutrition Inc., C.P. 325, St-Hyacinthe, Qc, Canada J2S7B6.
Geraert, P.A.	Adisseo France S.A.S, Ets. de Commentry, rue Marcel Lingot, 03600 Commentry, France.
Gerrits, W.J.J.	Animal Nutrition Group, Wageningen Institute of Animal Sciences (WIAS), Wageningen University, PO Box 338, 6700 AH Wageningen, The Netherlands.
Giles, L.R.	Department of Primary Industries, Elizabeth Macarthur Agricultural Institute, PMB 8, Camden, NSW, 2570, Australia.
Gonyou, H.W.	Prairie Swine Centre, Saskatoon, Saskatchewan, Canada.
Gresham, M.J.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Gu, X.H.	Institute of Animal Science, Chinese Academy of Agricultural Sciences, Beijing 100094, China.
Halekoh, U.	Department of Genetics and Biotechnology, Tjele, Denmark.
Hampson, D.J.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch WA 6150, Australia.
Hansson, A.C.	Animal Genetics and Breeding Unit, University of New England, Armidale, NSW, 2351, Australia.
Harvey, R.B.	Food and Feed Safety Research Unit, Agricultural Research Service, USDA, 2881 F&B Road, College Station, TX 77845 USA.

Hedderley, D.I.	New Zealand Institute for Crop & Food Research, Food Industry Science Centre, Palmerston North, New Zealand.
Hemsworth, P.H.	Animal Welfare Science Centre, Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Hen, S.	Alltech Asia-Pacific Bioscience Center, Beijing 101407.
Hendriks, W.H.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Henman, D.J.	QAF Meat Industries, Corowa, NSW, 2646, Australia.
Hennessy, D.P.	Pfizer, Poplar Road, Parkville, Vic. 3052, Australia.
Hermesch, S.	Animal Genetics and Breeding Unit, University of New England, Armidale, NSW, 2351, Australia.
Hernández, A.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA, 6150, Australia.
Highfield, L.D.	Department of Veterinary Integrated Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843 USA.
Hofmeyr, C.D.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Hofstetter, U.	Biomin GmbH, Herzogenburg, Austria.
Holyoake, P.K.	University of Sydney, Camden, NSW, 2570, Australia.
Huang, R.	Nanjing Agricultural University, Nanjing 210095, China.
Huang, S.	Nanjing Agricultural University, Nanjing 210095, China.
Hughes, P.E.	Pig and Poultry Production Institute, South Australia Research and Development Institute, Roseworthy Campus, Roseworthy, SA. 5371, Australia.
Jakob, S.	Adisseo France S.A.S, Ets. de Commentry, rue Marcel Lingot, 03600 Commentry, France.
James, K.J.	Department of Primary Industries, Camden, NSW 2570, Australia.
Janz, J.A.M.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Jensen, S.K.	Danish Institute of Agricultural Sciences, Department of Animal Health, Welfare and Nutrition.
Jin, D.I.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Johnston, L.J.	West Central Research and Outreach Centre, University of Minnesota, Morris, MN 56267, U.S.A.
Jordan, D.	School of Veterinary Science, The University of Queensland, St Lucia, Qld. 4072, Australia.
Jung, K.C.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Jung, W.Y.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Karlen, G.A.	Animal Welfare Science Centre, Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Ketses, C	Department of Primary Industries, Werribee, Vic. 3030
Kerr, C.A.	CSIRO Livestock Industries, Queensland Biosciences Precinct, St Lucia, Qld. 4067, Australia.
Kerr, M.G.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Kerton, D.J.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Kim, J.C.	Murdoch University, Murdoch, WA, 6155, Australia.
Kim, J.E.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
King, R.H.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Kluess, J.	Schothorst Feed Research, 8200 AM Lelystad, The Netherlands.

Kwon, Y.S.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Laubitz, D.	The Kielanowski Institute of Animal Physiology and Nutrition, Poland.
Lauridsen, C	Danish Institute of Agricultural Sciences, Department of Animal Health, Welfare and Nutrition, Denmark.
Lawhorn, D.B.	Department of Large Animal Medicine & Surgery, College of Veterinary Medicine, Texas A&M University, College Station, TX 77845 USA.
Lawlor, P.G.	Moorepark Research Centre, Fermoy, Co. Cork, Ireland.
Lawton, D.E.	EpiCentre, Massey University, Private Bag 11222, Palmerston North, New Zealand.
Layton, D.S.	Australian Animal Health Laboratory, CSIRO Livestock Industries, Geelong, Vic. 3219, Australia.
Lee, J.H.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Lei, Q.	College of Animal Science and Technology, Henan Agricultural University, China, 100094.
Leury, B.J.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Levis, D.G.	University of Nebraska, Agricultural Research and Development Center, 1071 County Road G, Ithaca, Nebraska 68033.
Lin, D.	College of Animal Science and Technology, Henan Agricultural University, China, 100094.
Liu, Y.G.	Adisseo Asian-Pacific P/L, 1 Loyang Way 4, Singapore 507028.
Loibner, A.P.	IFA-Tulln, Department of Environmental Biotechnology, Tulln, Austria.
Lynch, P.B.	Moorepark Research Centre, Fermoy, Co. Cork, Ireland.
Mather, A.	Department of Primary Industries, Attwood Centre, Attwood, Vic. 3049, Australia.
Matthews, B.	Department of Primary Industries, Attwood Centre, Attwood, Vic. 3049, Australia.
McCauley, I.	Department of Primary Industries, Attwood Centre, Attwood, Vic. 3049, Australia.
McCullough, S.M.	Department of Agriculture Western Australia, South Perth, WA, 6151, Australia.
McEwan, J.	Access Pharmaceuticals Australia Pty Ltd, Unit 5, 15-17 Gibbes St, Chatswood, NSW, 2067, Australia.
McLeish, J.	ADM, Bondi Junction, NSW, 2022, Australia.
Meng, X.S.	Institute of Animal Science, Chinese Academy of Agricultural Sciences, Beijing 100094, China.
Mersmann, H.J.	USDA/ARS Children's Nutrition Research Center, Department of Pediatrics, Baylor College of Medicine, Houston, TX 77030.
Mézes, M.	Szent Istvan University, Department of Nutrition, Gödöllö, Hungary.
Miao, C.	Alltech Asia-Pacific Bioscience Center, Beijing 101407.
Mitchell, M.	Research Centre for Reproductive Health, University of Adelaide, SA. 5011, Australia.
Mitchell, P.M.	Department of Primary Industries, Epsom, Vic. 3551, Australia.
Montagne, L.	UMRVP, INRA, Rennes, 35042 France.
Moore, D.	Biomin Australia Pty Ltd., Berwick, Vic. 3806, Australia.
Moore, K.L.	Department of Agriculture Western Australia, Locked Bag 4, Bentley Delivery Centre, WA, 6983. Australia.
Moran, C.	Centre for Advanced Technologies in Animal Genetics and Reproduction (Reprogen), Faculty of Veterinary Science, University of Sydney, NSW, 2006, Australia.
Morel, P.C.H.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Morley, W.C.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Morris, R.S.	Massey University EpiCentre, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Palmerston North, New Zealand.

Morrison, R.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Mullan, B.P.	Animal Research and Development, Department of Agriculture, Locked Bag No. 4, Bentley Delivery Centre, WA, 6983, Australia.
Muralitharam, M.	School of Biological and Chemical Sciences, Deakin University, Pigdons Road, Geelong, Vic. 3217, Australia.
Murphy, A.	QAF Meat Industries, Corowa, NSW, 2646, Australia.
Nakavisut, S.	Animal Genetics and Breeding Unit, Armidale, NSW, 2351, Australia.
Newman, R.E.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Nicholls, R.R.	Animal Research and Development, Department of Agriculture, Locked Bag No. 4, Bentley Delivery Centre, WA, 6983, Australia.
Nisbet, D.J.	Food and Feed Safety Research Unit, Agricultural Research Service, USDA, 2881 F&B Road, College Station, TX 77845 USA.
Nitsch, S.	Biomin GmbH, Herzogenburg, Austria.
Norris, A.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Nugent, L.	Department of Primary Industries, PIRVic. Attwood Centre, Attwood, Vic. 3049, Australia.
O'Neil, T.E.	Australian Animal Health Laboratory, CSIRO Livestock Industries, Geelong, Vic. 3219, Australia.
O'Connell, M.K.	Moorepark Research Centre, Fermoy, Co. Cork, Ireland.
O'Doherty, J.V.	University College, Belfield, Dublin, Ireland.
Ostrowska, E.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Park, C.S.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Park, E.W.	Centre for Advanced Technologies in Animal Genetics and Reproduction, Faculty of Veterinary Science, University of Sydney, Sydney, NSW, 2006, Australia.
Payne, H.G.	Animal Research and Development, Department of Agriculture, Locked Bag No. 4, Bentley Delivery Centre, WA, 6983, Australia.
Pellikaan, W.F.	Animal Nutrition Group, Wageningen Institute of Animal Sciences (WIAS), Wageningen University, PO Box 338, 6700 AH Wageningen, The Netherlands.
Pethick, D.W.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA, 6150, Australia.
Pettitt, M.	Prairie Swine Centre Inc., Saskatoon, Saskatchewan, S7H 5N9 Canada.
Philpotts, A.C.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Pitchford, W.	University of Adelaide, SA, 5371, Australia.
Piva, A.	DIMORFIPA, Faculty of Veterinary Medicine, University of Bologna, Via Tolara di Sopra 50, 40064 Ozzano Emilia (Bologna), Italy.
Pluske, J.R.	School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA, 6150, Australia.
Purchas, R.W.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Revell, D.K.	CSIRO Livestock Industries, Private Bag 5, Wembley, WA, 6913, Australia.
Ridley, D.	Department of Primary Industries, Rutherglen, Vic. 3685, Australia.
Rijnen, M.M.	Hendrix UTD B.V., Nutreco, The Netherlands.
Rikard-Bell, C.	Elanco Animal Health, Macquarie Park NSW, 2113, Australia.
Roese, G.	Department of Primary Industries, Tamworth Agricultural Institute, Marsden Park Road, Calala, NSW, 2340, Australia.
Rothkötter, H.J.	Otto von Guericke University Magdeburg, Medical Faculty, 39120 Magdeburg, Germany.
Ru, Y.J.	Danisco Animal Nutrition, Singapore.

Russell-Jones, G.	Access Pharmaceuticals Australia Pty Ltd, Unit 5, 15-17 Gibbes St, Chatswood, NSW, 2067, Australia.
Rutley, D.	University of Adelaide, SA, 5371, Australia.
Sands, J.S.	Danisco Animal Nutrition, P.O. Box 777, Marlborough, Wiltshire, SN8 1XN UK.
Schatzmayr, D.	Biomin GmbH, Herzogenburg, Austria.
Schatzmayr, G.	Biomin GmbH, Herzogenburg, Austria.
Schauer, B.	EpiCentre, Massey University, Private Bag 11222, Palmerston North, New Zealand.
Schulz, S.	Research Centre for Reproductive Health, University of Adelaide, SA, 5011, Australia.
Scott, H.M.	Department of Veterinary Integrated Biosciences, College of Veterinary Medicine, Texas A&M University, College Station, TX 77843 USA.
Scrimgeour, K.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Seah, E.	New Zealand Institute for Crop & Food Research, Food Industry Science Centre, Palmerston North, New Zealand.
Shaw, K.	QAF Meat Industries, Corowa NSW, 2646, Australia.
Sheehy, P.A.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Shilton, A.N.	Centre for Environmental Technology and Engineering. Massey University, Palmerston North, New Zealand.
Simons, J.A.	Department of Primary Industries, Attwood Centre, Attwood, Vic. 3049, Australia.
Singh, D.N.	Department of Primary Industries & Fisheries, Animal Science, P O Box 327, Cleveland, Qld. 4163, Australia.
Sirisatien, D.	Department of Statistics, Macquarie University, NSW, 2109, Australia.
Skilton, G.	Aorere Farms, 161 Tayforth Road, R4, Wanganui, New Zealand.
Smith, C.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Smith, M.	School of Veterinary Science, The University of Queensland, St Lucia, Qld. 4072, Australia.
Smith, T.K.	Department of Animal and Poultry Science, University of Guelph, Guelph, Ontario, Canada.
Smits, R.J.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Soede, C.J.	Nutrifeed, PO Box 441, 5460 AK, Veghel, The Netherlands.
Souffrant, W.B.	Research Institute for the Biology of Farm Animals, RU Nutritional Physiology 'Oskar Kellner', 18196 Dummerstorf, Germany.
Sowa, E.	Otto von Guericke University Magdeburg, Medical Faculty, 39120 Magdeburg, Germany.
Spötter, A.	Institute for Animal Breeding and Genetics, University of Veterinary Medicine, Hannover, Germany.
Stephenson, G.S.	Australian Animal Health Laboratory, CSIRO Livestock Industries, Geelong, Vic. 3219, Australia.
Strom, A.D.G.	Australian Animal Health Laboratory, CSIRO Livestock Industries, Geelong, Vic. 3219, Australia.
Suárez, M.	Animal Genetics and Breeding Unit, Armidale, NSW, 2351, Australia.
Suster, D.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Swamy, H.V.	Department of Animal and Poultry Science, University of Guelph, Guelph, Ontario, Canada.
Swanson, M.	University of New England, Animal Science, Armidale NSW, 2351, Australia.
Tatham, B.G.	Department of Primary Industries, 600 Sneydes Rd, Werribee, Vic. 3030, Australia.
Taylor, G.	Department of Primary Industries, Tamworth Agricultural Institute, Marsden Park Road, Calala, NSW, 2340, Australia.
Thomson, P.C.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Trezona, M.	Western Australian Department of Agriculture, Locked Bag No 4, Bentley Delivery Centre, WA, 6983, Australia.

Trott, D.J.	School of Veterinary Science, The University of Queensland, St Lucia, Qld. 4072, Australia.
Tume, R	CSIRO, Food Science Australia, Qld. 4170, Australia.
Türk, M.	Leibniz-Institut für Agrartechnik Potsdam-Bornim e.V., 14469 Potsdam, Germany.
Turnley, K.	Alltech Biotechnology Pty Ltd, Dandenong South, Vic. 3175, Australia.
Tybirk, P.	Danish Agricultural Advisory Centre, Udkærsvej 15, DK-8200 Aarhus, Denmark.
van Barneveld, R.J.	Barneveld Nutrition Pty Ltd, 19-27 Coonan Rd, South Maclean, Qld. 4280, Australia.
van Wettere, W.H.E.J.	QAF Meat Industries, PO Box 78, Corowa, NSW, 2646, Australia.
Vaschina, B.K.	CSIRO Livestock Industries, Locked Bag 1, Armidale, NSW, 2350, Australia.
Verstegen, M.W.A.	Animal Nutrition Group, Wageningen Institute of Animal Sciences (WIAS), Wageningen University, PO Box 338, 6700 AH Wageningen, The Netherlands.
Verstegen, W.A.	Animal Nutrition Group Animal Sciences Department, Wageningen University, The Netherlands.
Walker, P.J.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Wang, R.	China Agricultural University, China, 100094.
Wang, Z.S.	Institute of Animal Nutrition, Sichuan Agricultural University, Yaan, China 625014.
Warner, R.D.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Welch, E.B.M.	PIC New Zealand, PO Box 348, Christchurch, New Zealand.
Wester, T.J.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Weston, P.A.	Department of Primary Industries, 600 Sneydes Road, Werribee, Vic. 3030, Australia.
Wilkinson, B.H.P.	Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.
Williams, I.H.	University of Western Australia, Nedlands, WA, 6009, Australia.
Wilson, R.H.	Wandalup Farms, PO Box 642, Mandurah, WA, 6210, Australia.
Wolinski, J.	The Kielanowski Institute of Animal Physiology and Nutrition, Poland.
Wood, G.R.	Department of Statistics, Macquarie University, NSW, 2109, Australia.
Wu, K.	Immunology Molecular Diagnostic Research Unit, Elizabeth Macarthur Agricultural Institute, Camden, NSW, 2570, Australia.
Wynn, P.C.	Faculty of Veterinary Science, University of Sydney, PMB 3, Camden, NSW, 2570, Australia.
Yu, S.L.	Division of Animal Science & Resources, Research Center for Transgenic Cloned Pigs, Chungnam National University, Daejeon 305-764, Republic of Korea.
Zabielski, R.	The Kielanowski Institute of Animal Physiology and Nutrition, Poland.
Zenke, T.	Leibniz-Institut für Agrartechnik Potsdam-Bornim e.V., 14469 Potsdam, Germany.
Zhu, W.	Nanjing Agricultural University, Nanjing 210095, China.

Acknowledgements

The quality of *Manipulating Pig Production X* represents the continued support from many members of the Australasian pig science community. The Australasian Pig Science Association (APSA) (Inc.) would like to thank everyone involved in the organisation, preparation and management of this year's conference. APSA (Inc.) would also like to thank all those who attended the conference especially those who presented papers and contributed to discussions. Thanks also to our many international delegates for taking the time and effort to attend the conference and also contribute papers. The scientific program for the Biennial Conference has always been based on the symposia and reviews provided by invited speakers. Appreciation is expressed to Professor Frank Dunshea who presented the Dunkin Memorial Lecture, to reviewers Harry Mersmann, Chris Moran, Trish Holyoake, Rebecca Morrison, Trevor Smith, Martin Verstegan and Darryl D'Souza, and to symposia leaders Pat Mitchell and Rob Smits. APSA also thanks the various chairpersons, who have the difficult job of ensuring that the program runs to schedule and the judges of the APSA Medal, APSA Poster Award and the Batterham Memorial Award.

APSA (Inc.) would also like to thank the team behind Par Excellence especially Leith Finnie, Janet Paterson, Dickson Poon, Jennie Pearce and Amanda Pringle for the excellent work that they have done in editing, producing and marketing *Manipulating Pig Production X*.

This conference is only possible because of the hard work of the organising committee. Accordingly thanks go to Dr. Bruce Mullan (Vice-President), Dr. John Pluske (Past-president), Mr. Ian Barugh (Secretary), Mr Graham Pearson (Treasurer), Prof. Frank Dunshea, Mr. Geogy Philips, Dr. Patrick Morel and Mr. Rob Smits. Thanks are also due to the New Zealand Pork Industry Board (NZPIB), especially Lisa Julian and Angus Davidson, who were the NZ Secretariat. APSA also acknowledges the invaluable help of all the referees (named elsewhere in the Proceedings) for their contributions.

APSA (Inc.) would also like to acknowledge the new collaborative agreement with the recently formed Pork Co-operative Research Centre. Not only will the relationship between the Pork CRC and APSA (Inc.) strengthen future pork research in this region but the additional opportunities the CRC will generate for collaboration and networking between pig scientists will strengthen our industry. Research funding in most industries can be difficult to source and the impact that this has on our industry and research groups cannot be ignored. The provision of an additional agency such as the Pork CRC bodes well for Australasian pig science.

Finally, the Xth Biennial Conference would not have been possible without the generous support of a number of sponsors. The conference sponsors, listed below, are gratefully acknowledged for supporting APSA (Inc.).

Principal Sponsors

- Australian Pork Limited (APL)
- NZPIB

Gold Sponsors

- ABB Grain
- Adisseo P/L
- AgITO
- Alltech Biotechnology
- Biomin
- DSM Nutritional Products (Australia) P/L
- Elanco
- Feedworks
- IFNHH, Massey University
- IVABS, Massey University
- Kemin
- Ridley Agriproducts P/L
- NRM

Silver Sponsors

- Pfizer
- Vitec Nutrition

Bronze Sponsors

- Chemeq
- Fort Dodge
- Cundy Technical Services

Sponsors

- Barneveld, Edwards, Choct Animal Nutrition (BECAN) Consulting Group
- Pork Corp
- Clarke & Associates
- QAF Meat Industries
- Jefo Australia P/L

Batterham Memorial Award

- BASF Animal Nutrition
- Purina Mills Inc
- Ridley Agriproducts
- Heartland Lysine Inc
- Adisseo Australia Pty Ltd

APSA Medal

- Barneveld, Edwards, Choct Animal Nutrition (BECAN) Consulting Group

APSA Poster Award

- Ridley Agriproducts P/L

Acknowledgement to Referees

The Proceedings 'Manipulating Pig Production X' contains 99 one-page papers, seven reviews and two symposia. As is the policy of the Association, all one-page papers, reviews and symposia were reviewed by external referees. The APSA committee and Editor gratefully acknowledge the assistance generously given during 2005 by the following referees and those who may have been inadvertently omitted from the list.

Banhazi, T.
Barnett, J.
Barton, M.
Black, J.
Blackshaw, J.
Brewster, C.
Cadogan, D.
Channon, H.
Chin, J.
Close, W.
Cronin, G.
Crump, R.
Cutler, R.
Driesen, S.
D'Souza, D.
Dunshea, F.
Edwards, A.
Fahy, A
Gannon, N.
Giles, R.
Hampson, D.
Hemsworth, P.
Henman, D.
Hermesch, S.
Holyoake, T.
Hughes, P.
Johnsson, I.
Kim, J.
King, R.
Luxford, B.
Martin, C.
McCauley, I.
Mitchell, P.M.
Moore, K.
Moran, C.
Morel, P.
Morrison R.
Mullan, B.
Nairn, K.
Nottle, M.
Payne, H.
Philip, G.
Pluske, J.
Pope, G
Sillence, M.
Singh, D.
Smits, R.
Taverner, M.
Trezona, M.
Trott, D.
van Barneveld, R.
Wester, T
Williams, I.
Willis, S.
Wilson, R.
Wynn, P.

Australian Pork Limited

Principal Sponsor

Australian Pork Limited (APL), the national body representing the Australian pig industry, is proud to be the principal sponsor of the 10th Biennial Conference of the Australasian Pig Science Association (Inc.) in 2005.

APL supports a variety of research projects aimed at improving pig production methods and processes in Australia, with the aim of ensuring that Australian pork maintains and improves its position in both the domestic and export markets. The work of APSA plays a pivotal role in the development, improvement and implementation of research in the Australian pig industry. APL looks forward to continuing this very productive relationship with the Association.

APL is delighted to be here in Christchurch, and looks forward to being part of a very successful conference.

'World class science for Australia's pork industry'

Postal address

Australian Pork Limited
PO Box 148
DEAKIN WEST ACT 2600

Website: www.australianpork.com.au

Telephone and facsimile numbers

Telephone: (02) 6285 2200
Freecall: 1800 789 099
Facsimile: (02) 6285 2288

Email: apl@australianpork.com.au

nzpork
INDUSTRY BOARD

The New Zealand Pork Industry Board is proud to be a sponsor of the Australasian Pig Science Association's conference in 2005.

NZPIB operates five key strategic areas of focus, one of which is

Research and Innovation.

The objective of this strategic area is to:

Secure the long term future of the New Zealand Pork Industry by facilitating the means of achieving competitive production and innovation throughout the value chain.

We are delighted to have the opportunity to meet you at this year's conference and look forward to hosting you at this year's premier science event.

100% NEW ZEALAND TRIM PORK ®
NATIONAL HEART FOUNDATION APPROVED CERT TM

100% NEW ZEALAND PORK ®

PREMULAC

Avilac E
For optimal fat digestibility

ABB Grain are suppliers of the newest technology and best quality ingredients to the pig feed industry. These products include grains, proteins, minerals, and Premulac - Highly digestible protein lactose concentrate for total nutrient availability as well as Avilac E - a new technology emulsifier for optimal fat digestibility. For all your feed requirements - ABB Grain is proud to be associated with APSA (Australasian Pig Science Association).

Keith Moore	027 480 6437	Sales - New Zealand
Christine Swadel	09 357 0669	Minerals, Premulac, Avilac E - New Zealand
James Wynn Williams	09 357 0674	Proteins - New Zealand
Richard Grigor	09 357 0670	Grains - New Zealand
Dean Plummer	0417 648 777	Minerals, Avilac E - Australia
Tim Henry	0419 117 847	Proteins - Australia

Growing for your future

ABB

Massey University

ivabs Institute of Veterinary Animal & Biomedical Sciences

POSTGRADUATE STUDY AT IVABS? YEAH RIGHT!!

Do you feel like a change?
Do you want to improve your veterinary knowledge?

IVABS offers a wide range of postgraduate possibilities including PhD and MVSc degrees, postgraduate diplomas and certificates to suit almost any requirement. A suite of post-graduate papers cover a wide range of topics, including small animal medicine and epidemiology, and special papers can be developed for almost any veterinary or animal science subject. Postgraduate degrees can be taken at Massey or extramurally.

Massey has had some great PhD graduates over the years. Two recent graduates believe completing a PhD was a life changing experience for the better.

Ron Jackson graduated with a PhD in 1996 when he was 61 years of age. Since then he has worked in many countries around the world on diseases like BSE, TB, Avian Flu and Brucellosis and been involved in several high profile research projects in New Zealand. At present he is in Tajikistan fulfilling a desire to improve life at the village level in developing countries. For Ron the PhD opened doors and allowed him to "work in lots of interesting places". He found the process of undertaking a PhD challenging but this was offset by the luxury of a great library and helpful academics. Not bad for an old bush vet!

In contrast Janet Patterson-Kane went into a PhD programme in equine orthopaedics a few years after her BVSc. She is now senior lecturer in the Royal Veterinary College in London. For Janet the PhD was the starting point of an academic career. She believes that the Massey PhD fosters creativity in research and develops an ability to work in "an innovative and collaborative manner not encouraged to the same degree ... in many overseas universities".

To find out more contact:
Kevin Stafford, Phone: +64 6 350 5548 or Email: K.J.Stafford@massey.ac.nz

Te Kunenga ki Purehuroa

www.massey.ac.nz
0800 MASSEY (627 739)

Food for thought

...better foods, better nutrition, better health, better lifestyle

The Institute of Food, Nutrition and Human Health is proud to be a sponsor of the Australasian Pig Science Association's 10th biennial conference. Through the MRC the Institute has a long standing association with the New Zealand and Australian pork industries as a key provider of research, extension and training.

The Institute of Food, Nutrition and Human Health is a leading provider of knowledge for food health innovation in New Zealand, offering integrated research and education across the entire food and health value chain:

- Agribusiness, Logistics and Supply Chain Management
- Animal Nutrition and Physiology
- Food and Feed Evaluation
- Food - Science, Technology, Engineering
- Human Nutrition and Health
- Sport and Life Sciences

Key Contact

Dr Gordon Reynolds
G.W.Reynolds@massey.ac.nz

APSA Committee Members

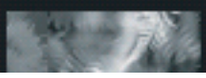
Dr Patrick Morel
P.C.Morel@massey.ac.nz

Ian Barugh
I.W.Barugh@massey.ac.nz

Graham Pearson
G.Pearson@massey.ac.nz

www.massey.ac.nz
0800 MASSEY (627 739)

Massey University



Nutrition Limited

The Animal Nutrition Professionals

Suppliers of

- *On-Farm Advisory and Analytical Service*
- *Hi-Spec Premixes for the pig industry*
- *Full range of Nutritional and Animal Health products to maximise pig production*
- *SCA Provimi Piglet Starter diets*
- *Alltech Nutritional Supplements*

VITEC COMPANY CONTACTS

North Island	*Auckland / Nutritionist:*	*Chris Whalley*	*027 473 3525*
	Auckland / Nutritionist	*Ashley Gibbs*	*027 431 8910*
	Waikato:	*Tom Van Der Oord*	*027 278 9705*
	Waikato:	*JD Van Der Oord*	*027 294 1809*
	Wanganui:	*Brian O'Dea*	*027 278 5846*
South Island	*Christchurch:*	*Rob McGregor*	*021 334 469*
	South Canterbury	*Gordon Biggs*	*0274 315 002*
	Clinton, Southland:	*Darin Dick*	*027 243 0030*

2/20 Kerwyn Avenue, East Tamaki
◈ P O Box 58 037, Greenmount, Auckland, New Zealand

Free Phone: 0800 4 VITEC (0800 48 48 32)
◈ Phone: 09 273 6318
◈ Fax: 09 273 6320

Hits your target, every time

Paylean® from Elanco Animal Health is the safe and easy way to increase the performance, consistency and profitability of your finishing programme. Simply added to rations 28 days before turn-off, this revolutionary feed additive directs nutrients to produce lean muscle instead of fat, significantly improving growth rates, feed efficiency and lean carcass yield.

No matter what your production goals, Paylean helps you to hit the target, every time. For further information, contact your local feedmill or Elanco Animal Health on **1800 226 324**.

ELANCO®
Paylean®

customer driven solutions for the global feed industry

For over 15 years Danisco Animal Nutrition has been at the heart of some of the major changes in animal nutrition. Danisco Animal Nutrition is focused on the development and application of feed enzymes and betaine offering value-added benefits to the pig producer.

porzyme®

Porzyme is a range of unique enzymes which improve the nutritive value of ingredients in pig diets. Its economic benefits to the pig industry include:

- *Improved daily gain, feed efficiency and feed intake*
- *Opportunities to reduce diet costs, whilst maintaining pig performance*
- *Reducing raw material variability giving more uniform pig performance*
- *Reduced effluent losses*

betafin®

Betafin is a pure form of natural betaine, extracted from sugar beet. Betaine has an osmolyte function, helping the pig maintain its water balance more efficiently. It is also a highly effective methyl group donor. Its economic benefits to the pig industry include:

- *Improved lean deposition, when energy intake limits lean gain*
- *Opportunities to reduce dietary energy content and formulation cost*
- *Sparing of methionine and choline for methylation in pig diets*
- *Improved productive performance in piglets, grower/finishers and sows*

phyzyme™ XP

Phyzyme XP is a new phytase feed enzyme that is highly efficient at releasing phosphorus from feed ingredients. Include Phyzyme XP in your pig feed to:

- *Reduce diet cost, by using less inorganic phosphorus*
- *Potential to reduce diet cost by using less amino acids and trace minerals*
- *Substitute for phosphorus sources in the diet, e.g. meat and bone meal*
- *Create a better environment, by reducing phosphorus excretion in the manure - typically 30% lower*

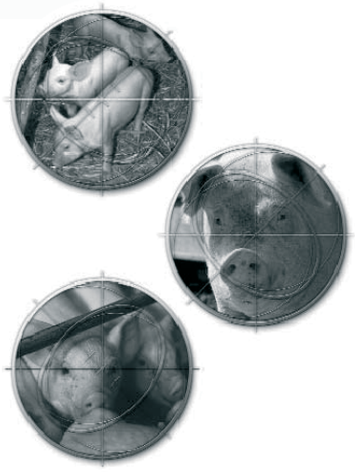
DANISCO
First you add knowledge...
Danisco Animal Nutrition

FEEDWORKS
"Performance through Science"

To obtain more information contact:

Feedworks, PO Box 369, Romsey, Vic 3434
Ph: 03 5429 6458 Fx: 03 5429 5327
Email: mal.mottram@feedworks.com.au
david.cadogan@feedworks.com.au
nickkempe@bigpond.com.au

Alltech® - delivering sustainable solutions to the pig industry ...naturally



Alltech, the international leader in natural, researched products, are committed to the future of the Australasian Pig Industry. Through sponsoring programs such as the "Premier Pig Program" and supporting local research, Alltech Australia strives to bring the industry to new heights.

ALLTECH BIOTECHNOLOGY P/L

68-70 Nissan Drive, Dandenong South • VIC, 3175 Australia
Tel: +61 3 9791 2800 • Fax: +61 3 9791 2122
E-mail: australia@alltech.com • *www.alltech.com*

Alltech products are exclusively distributed in New Zealand by Cundy Technical Services.
For more information, please contact: mike@cundytec.co.nz

Alltech®
...naturally

AA.2663.08.03

Improving feed performance, quality & safety

Anti-bacterial acidifiers:

Assist the young pig through the weaning transition via enhanced feed hygiene, assisting protein digestion and diarrhoea control

ACID LAC® brand Dry
ACID LAC® brand Liquid

Enzymes and Bio-surfactants:

Assist digestion in the young pig and also act on cereal and fibre components to increase nutritional value for growing pigs

A specific nutrient absorption enhancer designed for weaner pigs

KEMZYME® brand HF
KEMZYME® brand W
KEMZYME® brand B
KEMZYME® brand S
LYSOFORTE ™ brand Piglet Dry

Organic chromium:

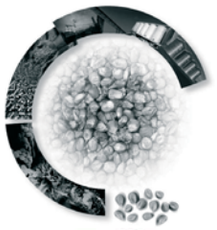
To aid in glucose metabolism and consequently increase litter size and lean:fat ratio plus enhance meat quality

KemTRACE™ brand Chromium

Odour inhibition:

Ammonia binding and urease inhibition to reduce odour emission from the piggery and enhance the pig's intestinal health

Bio CURB® brand Dry



Mould inhibition & mycotoxin binding:

For treating grain at the point of storage or in-feed application

Myco CURB® brand M Dry
Myco CURB® brand Liquid
TOXI BIND™ brand Dry

Anti-oxidants:

To protect fats/oils and feed against the negative effects of progressive oxidation and rancidity

RENDOX™ brand Liquid
ENDOX® brand Dry

For more information, please contact

1.1.1.1

Kemin (Aust.) Pty Ltd

1.02/10 Edgeworth David Avenue Hornsby, Sydney NSW 2077 Australia **Tel: +61-2-9482 2357 Fax: +61-2-9482 1837**
www.kemin.com

™Trademark of Kemin Industries Inc., U.S.A. ®Registered Trademark of Kemin Industries Inc., U.S.A.
©2001Kemin Industries (Asia) Pte Limited

Bring home more bacon.

Agriculture ITO arranges training for people of all ages and levels of experience, wherever you are in the country. Ask us how our training can improve your business or career in the pork industry.

Agriculture ITO is proud to be a Gold Sponsor of the 2005 Australasian Pig Science Association Xth Biennial Conference.

Talk to your local Agriculture ITO Industry Adviser today, call 0800 242 343 or visit www.agricultureito.ac.nz to find out more.

AgricultureITO
Industry Training Organisation

Call 0800 242 343 and talk to one of our Industry Advisers.

Preface

Manipulating Pig Production X is the record of the proceedings of the Tenth Biennial Conference of the Australasian Pig Science Association (APSA) (Inc.). Although starting from humble beginnings, the biennial conference and the proceedings have provided significant forums within Australasia for the presentation and discussion of pig science. Both the proceedings and the conference are held in high stead in the international pig science community because of the high standards maintained by APSA (Inc.).

In the last two years there have been great changes to the face of Australian pig science research with the development and formation of the Pork Co-operative Reserach Centre (CRC). The research and development programs for the Pork CRC auger well not only for pig science research but also for the sustainability and viability of the pork industry itself. The Pork CRC will provide a forum to be held on alternate years to the APSA (Inc.) Biennial Conference, providing pig scientists with an additional opportunity to promote, discuss and collaborate with their peers. The addition of the Pork CRC will complement the support that our principal sponsors, Australian Pork Limited (APL) and the New Zealand Pork Industry Board (NZPIB) , provide to pig science research and development.

The organisation of the Xth Biennial Conference has seen several changes. For the first time (but probably not the last), the conference has been held outside Australia, and to allow the organisation to proceed smoothly several members of the New Zealand (NZ) pig science community were voted (some would say press-ganged) onto the APSA (Inc.) Committee. A job well done Ian, Graham and Patrick.

The Xth Proceedings are also the first to be edited, produced and marketed by Par Excellence who have added their own special quality to the whole process. Leith Finnie, Janet Paterson, Dickson Poon, Jennie Pearce and Amanda Pringle have done an excellent job, going beyond the call of duty, under sometimes difficult circumstances. This conference has also seen great changes in the physical appearance of the proceedings. The first nine volumes of Manipulating Pig Production remained very similar in format and presentation. The Proceedings of the Xth Biennial Conference has received a total renovation, thanks once again to the efforts of Par Excellence. Janet Paterson, who was the editor for Manipulating Pig Production IX remained the editor for the Xth proceedings. Janet did an excellent job, continuing her high standard of editing. Par Excellence was also responsible for the design and maintenance of the new APSA (Inc.) website. For the first time, authors were able to register their interest, submit their papers and have them edited through links on the website.

The Batterham Memorial Award is a prestigious award offered by APSA(Inc.) in memory of the late Dr Ted Batterham. The award has been strongly contested in the past and it remains a feature of the APSA (Inc.) Conference. The award would not be possible without the continued support of its sponsor Ridley Agriproducts Pty Ltd.

Conferences like the Biennial APSA (Inc.) Conference do not just happen without the hard work and support of a number of people. The committee members have all devoted a great amount of time to ensure the success of the conference. I would like to thank the Organising Committee for their efforts over the last two years. The Committee consisted of: Dr. Bruce Mullan (Vice-President), Dr. John Pluske (Past-president), Mr. Ian Barugh (Secretary), Mr Graham Pearson (Treasurer), Prof. Frank Dunshea, Mr. Geogy Philips, Dr. Patrick Morel and Mr. Rob Smits. I would also like to thank the New Zealand Pork Industry Board (NZPIB), especially Lisa Julian and Angus Davidson, who were the NZ Secretariat for the conference. Finally, thanks are also due to all the sponsors, particularly Australian Pork Limited (APL) and the NZPIB, who were the Principal Sponsors for the conference.

Enjoy the conference!

Dr. Patricia Mitchell

President

Australasian Pig Science Association (Inc.)

A.C. DUNKIN
MEMORIAL LECTURE

Coping strategies for the modern scientist

F.R. Dunshea

Department of Primary Industries, Werribee, Vic. 3030.

Introduction

Scientific research is a wonderful career as it provides the exhilaration of discovery and every now and then the feeling that you have made a difference. It can also be a frustrating job as one deals with changing priorities, the vagaries of biology and the continual search for funding. While many scientists can make their fortune from a discovery that is commercialised, it is certainly not the career path to be contemplated if money is the end goal. Scientists have the opportunity to collaborate with and meet people from many walks of life, and this to me is one of the most exciting things about being a scientist. Provided they are able to satisfy their stakeholders or funding bodies scientists also have considerable flexibility to choose their area of study. Scientists also have reasonable freedom in how they organise their professional and personal lives and this has certainly been an added bonus to me. What I will attempt to do in this paper is to intertwine some of my experiences with some thoughts of others more insightful than myself and perhaps lay out some guiding principals for the young scientist. I will apologise in advance for any indulgent reminiscences.

Academic lineage

I think that it is very important for the young scientist to have a good understanding of the history of science, both in a general sense as well as within their discipline. While for many graduate students this simply means a competition to see who can cite the oldest reference in their thesis, a thorough understanding of what went before really aids in putting themselves and their work in perspective. I think that this has become a critical issue at the moment. For example, an observation I have made as I read the recent work of younger scientists is that very little appears to have happened before 1997, which is about the time that the first on-line journals became available. One of the more invidious reasons why many of the older papers are ignored is because modern scientists are under pressure to demonstrate impact and are trying to accept kudos for original work or discoveries. Of course this also applies to uncredited recent work. As more and more journals put their back issues on the electronic systems we may start to see some of the earlier work given the credit it deserves.

As well as providing background information and conflicting and supporting findings for the reader, citations also form part of the incentive and reward system that has operated in science since the publication of the Philosophical Transactions of the Royal Society of London in 1665 (Purver, 1967). For example, citations are often connected to funding appraisals, career assessments and promotion packages. Failure to properly credit the work of others undermines this system. Indeed this has recently appeared to become even more important as there is an increased reliance on citation indexes such as the Web of Science and Essential Indicators (http://www.isinet.com/) in assessing individual and organisation impacts. I will present more on this later.

One of the many interesting sections I found in the recent book by the Australian Medical Nobel laureate Peter Doherty was where he traced the history of his discipline through the Nobel laureate winners (Doherty, 2005). When I think about energy metabolism and body composition I think of the works of Kleiber (Kleiber, 1932;1961), Brody (Brody, 1945), Hammond (Hammond, 1932), Blaxter (Blaxter, 1962) and McMeekan (McMeekan, 1940) and modern scientists working in these areas should go back and read some of these works. The topic of academic lineage is particularly pertinent to this paper given that it is named in honour of Tony Dunkin, a man who had a profound effect upon many in the Australian pig industry. I only met Tony a couple of times but he has certainly indirectly influenced me through his colleagues and collaborators.

It is very important for a young scientist to maintain their academic lineage. The student-supervisor (or young scientist-mentor) relationship is a unique and evolving relationship. Initially, the student is often in awe of the mentor as they appear to know so much and the fledgling scientist does not believe they can ever approach this level of knowledge and worldliness. However, eventually the younger one gathers knowledge and experience, particularly in their narrow field of study. I often tell students that I expect that by the end of their candidature that they are the world's expert in their subject. Along the way they also realise that their mentor has human foibles and frailties.

I am particularly proud of my academic linage and I have been fortunate to have been mentored by a number of people who have had a profound effect upon me. I had a rather chequered undergraduate path (I did what could be called the gentlemen's degree) but one of my lecturers (Alan Bell) at La Trobe University saw at least some small spark in me and was willing to invest time in supervising my PhD. Perhaps one of the turning points in my relationship with Alan occurred about halfway through my candidature. I conducted my PhD on lipid metabolism in lactating goats. Given the fact that I could only conduct intensive metabolic studies on only a few goats at a time I needed to have the goats kidding every few weeks, which in turn necessitated having animals milking continuously. My kind-hearted supervisor, who grew up on a dairy farm, offered one Saturday to give me a break and he would do the milking. Well Friday was always my big day for enjoying a few refreshments (often at the campus bar) and this Friday was no exception. I was in no condition to ride my pushbike the 14 km home so I slept in my fold-away bed in an annexe off the animal room. I was awakened next morning to the sounds of goats bleating, milk cans being kicked over and much swearing. I glanced out into the animal room to view utter chaos but never let on that I was there. So the great ruminant nutritionist and dairy scientist was fallible after all. I didn't have the heart (or courage) to tell Alan until some months later just before he left to take up a position at Cornell University. Alan and I continued to work together when I moved to Cornell University a couple of years later and we have remained great mates ever since and make sure that we share a bottle of single malt scotch at least once a year (generally more often and more than one bottle). While at Cornell University I had the great fortune to work with Dale Bauman. The term gentleman and scholar never fitted anyone so aptly. So much did Dale influence me and my thinking that I decided to name my son after him. When our third daughter (and final child) was born, I had no choice but to call her Dayle. The next stage of my career was the move to Werribee where once again I was fortunate to have a great mentor and mate in Ray King who helped me when I was starting out in pig science.

I am fortunate to have been able to surround myself with and conduct research with many great scientists and people. I take great pride in the distinguished list of co-authors that appear in my CV. However, these collaborations did not just happen and it is important for the modern scientist to actively go out and seek and forge these relationships. This is not always easy for a young graduate student and I still remember the trepidation I felt when as a graduate I first approached David Pethick to talk to him about isotopic techniques to measure fatty acid kinetics at a Nutrition Society meeting in Adelaide in 1985. Dave and I are now great mates and collaborators and we were both filled with pride when he presented me with the inaugural Nutrition Society of Australia Research award in 1994. Incidentally, some of my proudest moments have occurred when my former graduate students received awards or accolades for their research activities, such as when Darryl D'Souza won the APSA Ted Batterham award. It is also a pleasure to continue to collaborate with former students well after they have left the nest and I would encourage scientists to continue to maintain these links, both personally and professionally.

One of the lessons here for the modern scientist is to realise that they cannot do everything themselves and so they should try to seek out the expertise that they need in the best people that they can. While I did both my undergraduate and post-graduate degrees at the same institution (La Trobe University) I did travel to the USA to do my post-doctoral training. I would advise any young scientist to do their degrees at different universities and to travel as much as their personal circumstances allow. This is certainly the norm in the USA where the young scientist may have conducted all their bachelors, Masters and PhD degrees and their post-doctoral training in four different institutions. If circumstances do not allow one to travel for extended periods away from home then look for shorter opportunities such as internships. I have certainly done this with a number of students over the years and all seem to have benefited from the experience. Even after obtaining an established job it is critical that the modern scientist travel and then not just to conferences. Continue to look for sabbatical opportunities, no matter how short they may be.

Two weeks in a laboratory that specialises in a particular work may save 12 months of frustrating bench or animal work as well as providing very valuable contact or potential collaborative links.

In a previous Dunkin lecture, David Lindsay presented an excellent treatise on how we might do better in the management of rural research (Lindsay 2001). He makes the point that researchers themselves have been largely neglected in the R&D administration process but that there are many compelling reasons for making them the focus when deciding the best way to use research funds. Regardless of this plea, research groups are shrinking while research organisations are rationalising the areas and activities in which they operate. For example, the old Departments of Agricultures no longer cover every species or discipline and there is a national focus now of rationalisation of research capabilities across Australia called the National Collaborative Research Infrastructure Strategy (http://www.dest.gov.au/sectors/research_sector/policies_issues_reviews/key_issues/ncris/default.htm). One of the aims of this strategy is to foster collaboration and reduce duplication and there is now doubt this will occur as a result of this initiative. However, the best collaborations are ones that occur through the coming together of people with a common vision or goal rather than those that occur through enforced necessity, although of course the latter can be successful. My advice to the modern scientist is not to rely solely upon the strategic alliances established by their organisations, but to actively seek out collaborations that add value to their research and development. The Co-operative Research Centre (CRC) system is a means by which successive Australian Federal Governments have been encouraging linkages between research organisations and industry. These are an excellent means by which to garner research capabilities and I am excited when I think what the CRC for an Internationally Competitive Pork Industry will do for the pork industry and the related scientific community. I have participated in three other CRCs as well as the National Centre of Excellence for Functional Foods and have seen the good science and benefits that can come out of these types of virtual research centres. However, the essence to their success is, as the title suggests, co-operation and this has to be worked upon.

Coping with change

A quick perusal of the psychology literature shows numerous works devoted to coping strategies, particularly how to cope with change. As scientists we are continually facing change; new technology, new information, industry reforms, changing government policy, new funding arrangements and just sometimes change for changes sake. In addition, the modern scientist has to cope with the pressures of funding organisations and an ever-increasing number of interested stakeholders many of who have different agendas and requirements for the work being undertaken. In addition, the reporting structures for each of these stakeholders are different and sometimes incompatible. On top of this there are a number of legislative requirements that must be addressed; for example OH&S, animal ethics and care committees, intellectual property protection (although this can be overemphasised in my opinion), gene regulations, training requirements, and any number of other interferences that sometimes appear to be designed to keep someone busy. All of this occurs in the face of rapidly escalating costs as we have to pay for all of these additional on-costs from our research funds. The scientific society is a microcosm of the wider community and so it is it is little wonder that a small, but perhaps significant, number of scientists succumb to the temptation of cheating and falsifying data (Martinson *et al*. 2005; see below). It is also not a surprise that the other problems that seem to afflict the wider community such as relationship breakdown, drug dependence and diseases of a sedentary lifestyle also afflict scientists and one can think of many examples in our pork science community. Travelling to conferences and meetings and having a few beers with colleagues is one of the most enjoyable aspects to my job. It has also allowed me to make many long-term friendships and establish excellent collaborations. While I recommend that the young scientist seeks out the conversation of their colleagues and alcohol can make the words flow more freely, these activities need to be balanced.

Perhaps it is an indication of where science communication is going that a number of the references and resources mentioned in this paper are available on-line. One such resource is the Advancing Science, Advising Community (AAAS the publishers of the prestigious Science journal) Science Next Wave website (http://nextwave.sciencemag.org/). This is a valuable resource developed specifically for graduate students and postdoctoral fellows, containing a number of papers that may be of assistance to the modern scientist. For example, an article by Peter Fiske dealt with the topic of coping with change (Fiske, 2000) where he discusses the 4 predictable steps of change ie. denial, resistance, exploration and commitment. Typically, the modern scientist may have three to five major career changes over their lifetime (often including leaving science) and many other major organisational changes. How well one copes with these changes as well as those of partners and colleagues will, to a large extent, determine the success of ones career and life. Discuss issues with a trusted colleague or other confidant rather than internalising problems. The reader is referred to this article by Fiske (2000) and other article on this website such as others dealing with change (Jensen, 2003), depression (Arney, 2004), women and science (Kuther, 2002; Benderly, 2005), chronic illness (Levine, 2005) and leaving science (Lopez, 1996; Sellwood, 2002).

My advice to the young scientist when faced with change is to try to get through the first stage early. Then, quickly determine if resistance is a viable option (such as when we were told that the Werribee site was to be closed and then with the pork industry were able to mount an immutable case to remain open) and then make a decision to act on this or move on to the latter two stages of exploration and commitment. One can waste much energy resisting organisational change and often the best remedy is to go with the flow and continue to concentrate on your research. The modern scientist should also remember that no matter how strong their funding position may be, it is always cyclical in nature and so there will be downturns in your chosen area. Be prepared to change species or disciplines either permanently or for a brief period. These may be incremental changes or a complete change. The easiest transitions are the incremental ones and these allow you to remain knowledgeable in your field without complete retraining. I trained in lactational physiology and metabolism in dairy ruminants but very quickly ended up being labelled a pig growth physiologist. However, I still continue to work in ruminant physiology and nutrition and lactation as well as pig lactation. I have also worked in the biomedical sciences using pigs and sheep as models of human nutrition and physiology. These relatively small incremental changes have allowed me to follow funding trends while still allowing me to maintain some sort of continuity that I can still revisit. For example, lipid metabolism has been a consistent theme throughout my career and I have revisited somatotropin many times over the last 20 years.

Even in the relatively short period of time that I have been a scientist there have been enormous changes in technology. My students are sick of me saying 'when I was a student' but a couple of examples will give the young scientist some sort of idea of the rapid changes in technology. When I was writing my PhD in 1986 my supervisor had moved to the USA and so I had to send copies of my chapter by mail for him to correct with red pen and return by mail. This was less than 20 years ago but it was before the FAX and definitely before email was available to the wider community. Also, preparing a simple graph was done with Letraset and would take three hours to do what might take 10 minutes now. Nevertheless, I would generally have my corrected chapters back inside two weeks whereas one of my colleagues who had his supervisor sitting in the next room might wait 3 months for his corrections. The internet and other technologies certainly make for much more rapid exchange of ideas and communication but it also brings with it some inefficiencies in unsolicited emails and also raises expectations of rapid responses. The modern scientist must be at least conversant with the new technologies and all of my graduate students must include some aspects of molecular biology in their training now, and they would be foolish not to continue to use these approaches where applicable. While on the subject of training it is also important for the modern scientist to be competent in experimental design and biometrics, subjects that are relatively poorly taught within most Australian graduate programs.

Responsible conduct in research

As mentioned earlier, the modern scientist is burdened by enormous pressures and competition for funds (Freeman *et al.,* 2001). Freeman *et al.* (2001) use the tournament model to describe a career in biosciences where the tournament offers the chance of winning a big prize (eg. an independent research career, tenure, scientific renown, awards) through competition. Often success may result from being only marginally better than the competition, but to the victor goes the spoils and so the differences in rewards far exceed the differences in work done. This places pressure on the modern scientist to work longer hours and push themselves harder. It can also create pressures to take short-cuts or display irresponsible behaviours and to counter these issues the United States National Academy of Sciences have published an excellent treatise on responsible conduct in research (NAS, 1995). I encourage all scientists, no matter what their stage of career, to read this document. Also, the USA federal government have an Office of Research Integrity and their website is a valuable resource for scientists (http://ori.hhs.gov/).

In a recent survey of several thousand US-based early and mid career scientists that are funded by the National Institute of Health there was a sizeable percentage of scientists who admitted to some form of cheating or other inappropriate behaviours (Martinson *et al.,* 2005) (Table 1). For behaviours that many consider as serious scientific misconduct, such as falsifying data or plagiarism, the incidence was less than 2%, although of course under-reporting cannot be discounted. I do have concerns that the ease with which web-based information can be 'cut and pasted' may increase the incidence of blatant plagiarism in the future, as children who use the internet for their assignments as students enter university and the work place. Fortunately many universities require student reports to be submitted electronically have introduced software that can detect plagiarism of anything published on the web. Other dubious behaviours occurred at much higher frequencies than plagiarism and raise serious concerns about the way many scientists are responding to the pressures of modern life. For example, there were a disturbing number of respondents who had inappropriately ascribed authorship (10%), which as discussed later is the fundamental credit reward system in science. Another behaviour that an alarming high number of scientists had exhibited in the previous three years was that of altering the design, methodology or results after pressure from a funder or stakeholder (15.5%). One assumes that this pressure may be both covert and overt and sometimes only just a perception but it is worrying that it exists and that some scientists feel compelled to respond to it. These findings led Martinson *et al.* (2005) to feel that certain

features of the research working environment may exacerbate the occurrence of misbehaviours, and in particular point to the system for distribution of funds (ie. the competitive grant system) that operates for the biosciences in the USA, and the ARC and NH&MRC systems in Australia. The Australian Rural Industry Research Councils have also used similar systems in the past although they are now moving more towards a tendering system. Others believe that these competitive grant systems allow the brightest scientists to shine and become independent early in their career although the model can appear to goal-driven (Doherty, 2005) which may contribute to the occurrence of these misbehaviours as individuals look for a competitive edge. Regardless, whatever the temptation the modern scientist must remain true to their profession and behave in a responsible and ethical manner. The effects of misconduct in both personal and professional senses can be profound with serious ramifications for the individual involved, their colleagues and the organisation (NSA, 1995).

Table 1. Percentage of scientists (early- and mid-career) who say they engaged in the following behaviours within the previous three years (n=3247) (Martinson *et al.,* 2005). Response rate was 47%.

Behaviours	%
Falsifying research data	0.3
Ignoring major aspects of human-subject requirements	0.3
Non-disclosure of possible commercial conflicts of interest	0.3
Inappropriate relationships with students, human research subjects or clients	1.4
Plagiarism	1.4
Unauthorised use of confidential information	1.7
Publishing the same data or results in more than one publication	4.7
Failure to present data that contradict one's own previous research	6.0
Circumventing minor aspects of human subject requirements	7.6
Inappropriate assignation of authorship	10.0
Withholding details of methodology or results in papers or proposals	10.8
Overlooking other's use of flawed data or questionable data interpretation	12.5
Using inappropriate research designs	13.5
Dropping outliers based on gut feeling that they were inaccurate	15.3
Changing the design, methodology or results after pressure from funder	15.5
Inadequate record keeping	27.5

Publish or perish

There is a well-worn adage in the scientific arena to 'publish or perish'. This has traditionally meant published in peer reviewed scientific journals. In recent times, and in the agricultural industries, this may mean technical bulletins or conference proceedings or, if valuable IP is involved, writing patents. While internal reports or reports to stakeholders (commercial or otherwise) are important to keep the work flowing and to satisfy contractual requirements, unless the work gets published externally there will be little value to the researcher, the scientific community or industry. My advice to the modern scientist is to publish their findings rapidly, after appropriate internal review and clearance, in the most influential or highest impact journal that they feel will accept their manuscript. The positive side to this is that they may have a high impact peer-reviewed article to add to their CV. However, the publishers and editors of these prestigious journals are generally overwhelmed with submissions and so the down side is that the manuscript is more likely to be rejected because it is not groundbreaking. Having a manuscript submitted and rejected may cause serious delays in publication and may even devalue the work in the eyes of the author(s), and so choosing the appropriate journal is an important decision. For example, if the data are largely of an applied nature then publication in a journal that is accessible to industry is probably the best choice. While these journals do not always have a high impact factor, they may be widely read and the information obtained in them broadly disseminated to industry. If, however, the work is more fundamental or basic in nature it is more appropriate to send to a more fundamental journal. A successful scientist working in the pork industry will most likely have publications in a full range of journals from applied to basic with the profile of these journals reflecting their research profile. I like to keep a balance of fundamental:applied research of 30:70 finding that this best matches my interests with my ability to obtain research funding. Others will decide on different ratios for themselves but I suggest that everyone should have at least some basic and applied research in their portfolio. I also make it a practice that if there is little IP in the research then the data should be presented quickly in abstract form at a conference, particularly if the conference proceedings have a wide audience. The danger here is that someone may repeat your study and have it published in full before you but at least the data are not gathering dust in a drawer (or electronic equivalent). The astute scientist will time it so that their peer-reviewed paper is in press when they make their conference presentation.

Deciding on the order of authorship and ensuring proper credit is also important. Bad blood can quickly develop if there is not agreement about who appears in the list of authors and the final order of authorship. This has become even more critical for the modern scientist as science has become a much more collaborative effort and more and more scientists are operating in CRCs and other virtual research centres. For example, the average number of authors for articles in the New England Journal of Medicine has risen from just over one to more than six between 1925 and 1994 (NAS, 1995). Closer to home the average number of authors of articles published in the Australian Journal of Agricultural Research has risen from 1.4 in the first year of publication to almost four this year (Figure 1). This places even greater importance on ensuring that the list of authors, and the order of listing, reflects their contributions made by the individual. Most journals only cite the first author in the text for multi-authored (more than two) articles and so first authored papers are essential to getting a young scientist noticed. The general convention in the life sciences is that the first author is the active scientist who did most of the animal or laboratory work while the last author is the principal investigator who probably conceived the idea for the study and obtained the funds. The rest of the authors are then generally listed in order of contribution. While this convention may vary between disciplines or organisations this is certainly how the ARC and NH&MRC attribute the kudos for research. Therefore, the young scientist needs to have the discussion with their supervisor and collaborators very early on in the design and initiation stages of the study to attribute authorship. Of course this can be reassessed at a later stage but the danger here is that expectations can be upset. Regardless, the earlier these discussions occur, the less chance of recriminations later on.

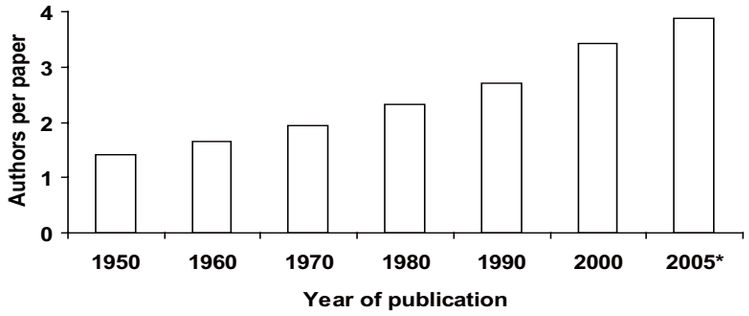


Figure 1. *Average number of authors per paper published in the Australian Journal of Agricultural Research (* data for 2005 are until the September issue)*

It is important that the modern scientist communicates their science well. It is compulsory for all graduate students in my laboratory to read David Lindsay's excellent guide to scientific writing (Lindsay, 1995) and I would encourage all scientists to keep a copy nearby when writing manuscripts. It is equally important for the modern scientist to be able to communicate orally to a wide variety of people, from the lay to the scientific and touching the political and administrative on the way through this spectrum. Spend time developing these techniques and seek the advice of experienced communicators. It will show. Over the years the University of Western Australia has included courses on scientific writing and public speaking in its curriculum and not surprisingly UWA graduates have featured prominently in the student and poster awards at various scientific meetings of professional agricultural societies.

Despite all the technology that is available it appears that the modern scientist is finding it even more difficult to prepare manuscripts and presentations. Indeed, I feel that a reliance on this technology can occasionally be an impediment. Too often I see young scientists "wordsmithing" their manuscripts a word or sentence at a time. It is far better to write or type all of ones's thoughts out in sizeable chunks and come back later to edit. This allows the thoughts to flow better whereas the former approach causes the author to lose their chain of thought. Many scientists also get caught up in formatting and want to have everything perfect before they will allow any one else to view their work. I have seen a number of scientists paralysed by their inability to get through this phase pass their work onto others for critique or review. Perhaps this is due to some fear of criticism but the scientist must be prepared to accept constructive criticism and counter destructive criticism. Of course, an editor or reviewer does not want to see an incomplete manuscript riddles with errors so the author must make sure that the manuscript is free of these types of mistakes, particularly in the bibliography. Some economies can also be achieved if the author does not have to rewrite every time they need to produce some documentation on their research project.

For example, much of the information contained in the original grant submission, animal ethics applications, protocols, reports to stakeholders, thesis chapters and manuscripts their work should be common and not need to be rewritten. However, I still see this happening.

Conclusions

There are no hard and fast rules on how to cope with the changing environment and continue to be a successful scientist. However, I hope that I have been able to provide some insight into what I feel are the important qualities of a scientist and a few clues on how to put these into practice. I feel that it is important for the scientist to understand the basic principles of the scientific process and also to have some knowledge of the history of science and where they sit in this line of history. They should be appreciative of their mentors and give freely to their proteges. The modern scientist should seek out excellence and attempt to establish collaborations with other scientists who will complement their skills and expertise. They should also be prepared to change disciplines and to travel to extend their training. The modern scientist should embrace new technologies that enhance their ability to conduct and publish excellent science but they must not be a slave to these same technologies. The modern scientist must communicate and publish their work and not let it languish in their filing system. All of these things must be done in a responsible manner. Science is a rewarding career for the right people and so above everything else the modern scientist must continue to be challenged and rise to these challenges as many have done before them.

References

ARNEY, K. (2004). Even Scientists Get the Blues. http://nextwave.sciencemag.org/cgi/content/full/2004/03/31/8.

BENDERLY BL (2005). Gender and Scientific Achievement: Views from the Bench. http://nextwave.sciencemag.org/cgi/content/full/2005/03/03/7?.

BLAXTER KL (1962). 'The Energy Metabolism of Ruminants.' (C.C. Thomas: Springfiled, IL).

BRODY S (1945). 'Bioenergetics of Growth, With Special Reference to the Efficiency Complex in Domestic Animals.' (Reinhold: New York).

Committee on Science EaPP (1995). 'On being a scientist: Responsible conduct in research.' (National Academy Press: Washington).

DOHERTY PC (2005). 'A beginners guide to winning the Nobel prize.' (Melbourne University Press: Melbourne).

FISKE P (2000). Coping with change. http://nextwave.sciencemag.org/cgi/content/full/2000/01/06/2

FREEMAN R, WEINSTEIN E, MARINCOLA E, ROSENBAUM J, SOLOMON F (2001). Competition and careers in biosciences. *Science 294*, 2293-2294.

HAMMOND, J. (1932). 'Growth and Development of Mutton Qualities in Sheep.' (Oliver and Boyd: London).

JENSEN, D. (2003). The Times They Are A'Changin'. http://nextwave.sciencemag.org/cgi/content/full/2003/12/17/5?

KLEIBER, M. (1932). Body size and metabolism. *Hilgardia* **6:** 315-353.

KLEIBER, M. (1961). 'The Fire of Life: An Introduction to Animal Energetics.' (Wiley: New York).

KUTHER, T. (2002). Women, Work, Stress, and Health. http://nextwave.sciencemag.org/cgi/content/full/2002/10/07/9.

LEVINE, I.S. (2005). To Tell or Not to Tell: Coping With Chronic Illness as a Science Trainee. http://nextwave.sciencemag.org/cgi/content/full/2005/06/09/6?.

LINDSAY, D.R. (1995). 'A guide to scientific writing.' (Addison Wesley Longman: Melbourne).

LINDSAY, D.R. (2001). Productive research environments in the rural industries - Can we do better? In 'Manipulating Pig Production VIII'. pp. Need page numbers ed. PD Cranwell. (Australasian Pig Science Association: Werribee).

LOPEZ, L. (1996). Success Means Coping With Sudden Demands. http://nextwave.sciencemag.org/cgi/content/full/1998/04/17/15?.

MARTINSON, B.C., ANDERSON, M.S. and DE VRIES, R. (2005). Scientists behaving badly. *Nature* **435:** 737-738.

MCMEEKAN, C.P. (1940). Growth and development in the pig with special reference to carcass quality characters. I. Age changes in growth and development. *Journal of Agricultural Science* **30**: 276-343.

PURVER, M. (1967). 'The Royal Society : concept and creation.' (Routledge & Kegan Paul: London).

SELLWOOD, C. (2002). A Small Step to a Big Career Change. http://nextwave.sciencemag.org/cgi/content/full/2002/04/02/6?.

1 Carcass quality, meat quality, growth and development

Dietary lecithin improves the healthiness of pork

D.N.D'Souza*†, B.L. Edmunds**, I.H. Williams**, J. McLeish***, B.P. Mullan*, D.W. Pethick**** and F.R. Dunshea*****

*Department of Agriculture, South Perth, WA 6151. **University of Western Australia, Nedlands, WA 6009. ***ADM, Bondi Junction, NSW 2022. ****Murdoch University, Murdoch, WA 6150. *****Department of Primary Industries, Werribee, Vic. 3030. †Current address - Alltech Biotechnology PL, Dandenong, Vic. 3175.

Dietary lecithin may provide health benefits to pork as well as improving its eating quality by reducing chewiness and hardness (D'Souza *et al.,* 2005). Human studies have shown lecithin supplementation can reduce cholesterol significantly (Spilburg *et al*., 2003) and we hypothesised that lecithin supplementation would have a similar effect in pigs. The use of lecithin supplementation to improve the 'healthiness' of pork or pork products, while also improving the tenderness of pork, could provide the pork industry with significant marketing opportunities. The aim of this experiment was to investigate the effect of lecithin supplementation on the fatty acid profile of pork and also on the plasma cholesterol of pigs.

Forty crossbred (Large White x Landrace x Duroc) female pigs in the grower and finisher growth phases were fed either 1) Control - a commercial diet, 2) 3 g lecithin per kg of feed (soybean lecithin, ADM Australia Pty Ltd), 3) 15 g lecithin per kg feed or, 4) 75 g lecithin per kg of feed. The pigs were housed individually and had *ad libitum* access to feed and water. Pigs were slaughtered at about 23 weeks of age (100 kg ± 1 kg) and their blood sampled to determine plasma cholesterol concentrations. The *Longissimus thoracis* muscle was removed at 24 h after slaughter and frozen at –80˚C for fatty acid analysis. All data were analysed by ANOVA.

Dietary lecithin supplementation at 75 g/kg significantly increased the levels of linoleic acid and reduced the levels of myristic acid in pork. Although not significant, pigs fed the diet supplemented with 75 g/kg lecithin tended to have lower plasma cholesterol at slaughter than pigs fed the control diet.

Table 1. Fatty acid composition (%) and total fat of the *Longissimus thoracis* muscle and plasma cholesterol concentrations of pigs fed diets supplemented with lecithin.

	Control	Lecithin 3 g/kg	Lecithin 15 g/kg	Lecithin 75 g/kg	lsd	P- value
Myristic acid 14:0	1.8	1.7	1.9	1.5	0.262	0.041
Palmitic acid 16:0	23.3	22.3	22.0	23.1	1.21	0.121
Heptadecenoic acid 17:0	0.47	0.57	0.36	0.15	0.554	0.470
Stearic acid 18:0	11.1	11.2	11.1	11.4	0.72	0.776
Oleic acid 18:1	33.8	34.7	34.0	31.2	4.65	0.462
Linoleic acid 18:2	16.6	16.8	15.4	20.8	3.91	0.043
Eicosadienoic acid 20:2	4.1	4.0	3.3	3.4	1.24	0.453
PUFA:SFA*	0.58	0.62	0.53	0.70	0.151	0.150
Total fat (%)	1.3	1.2	1.1	1.2	0.326	0.905
Plasma cholesterol (mmol/L)	2.7	2.2	2.5	2.0	0.507	0.064

*PUFA polyunsaturated fatty acid; SFA saturated fatty acids.

These results suggest that dietary lecithin works in pigs similarly to humans due to its effects on lowering cholesterol and increasing the levels of polyunsaturated fatty acid in muscle. We conclude that supplementing diets with lecithin has the potential to improve the 'healthiness' of pork.

References

D'SOUZA, D.N., MULLAN, B.P., MCLEISH, J. PETHICK, D.W. and DUNSHEA, F.R. (2005). In 'Manipulating Pig Production X, p. 270, ed J.E. Paterson. (Australasian Pig Science Association, Werribee).

EDMUNDS, B.L, WILLIAMS, I.H. MULLAN, B.P., MCLEISH, J., DUNSHEA, F.R. AND D'SOUZA, D.N. (2005). In 'Manipulating Pig Production X', p. 271, ed J.E. Paterson. (Australasian Pig Science Association, Werribee)

SPILBURG, C.A., GOLDBERG, A.C., MCGILL, J.B., STENSON, W.F, RACETTE, S.B., BATEMAN, J., MCPHERSON T.B., and OSTLUND, J.R. (2003). *Journal of the American Dietetic Association* **103:**577-581

Sensory profile of pork is not affected by animal byproducts

S. Cumarasamy*, V. K. Corrigan*, D. I. Hedderley*, W.H. Hendriks**, J.A.M Janz**, P.C.H. Morel**, R.W. Purchas**, E. Seah* and B.H.P. Wilkinson**

*Food Industry Science Centre, Palmerston North, New Zealand. **Massey University, Palmerston North, New Zealand.

Certain feed ingredients, most notably fishmeal (Maw *et al.,* 2001) and acorn (Cava *et al.,* 1999), can impact directly on pork flavour due to either diet related changes in fatty acid composition of lean and fat tissues or as a result of direct transfer of aromatic compounds from the feed to the meat (Hansen *et al.,* 2002). Use of animal byproducts in pig feeds is common in New Zealand while plant-based feeds are more typical in countries where soybean or canola meals are plentiful and locally available. Diversity in feeding practices may lead to distinctive "local" sensory characteristics of meat. In this study we examined the impact of diets containing animal byproducts on pork flavour and odour profiles.

Longissimus muscle samples from 16 female Duroc x (Large White x Landrace) pigs (slaughter weight 101.6 kg ± 3.2 SD) fed diets containing animal byproducts (barley + blood meal, fish meal, meat and bone meal, skim milk powder, soybean meal, tallow – typical for New Zealand) or plant feedstuffs (barley + soybean meal, soy protein isolate, peas, soybean and linseed oils) were prepared, minced and cooked for sensory evaluation by 13 trained sensory panelists who scored intensity of flavour and odour attributes on a 0 (none) to 10 (strong) scale. Mixed effect models that treated panellists, sessions and pigs as random effects were fitted to the data.

No significant differences were observed in any of the flavour or odour attributes of pork from pigs fed diets containing animal byproducts versus solely plant material. Few comparative studies are available, although Lettner *et al.* (2001) reported no difference in taste panel scores for tenderness, juiciness or flavour of pork from pigs fed diets containing 10-12% meat meal *vs.* 25% soy. In conclusion, replacing 16-18% of animal byproducts with 14-16% soybean meal in pig diets did not affect subsequent pork flavour or odour profiles.

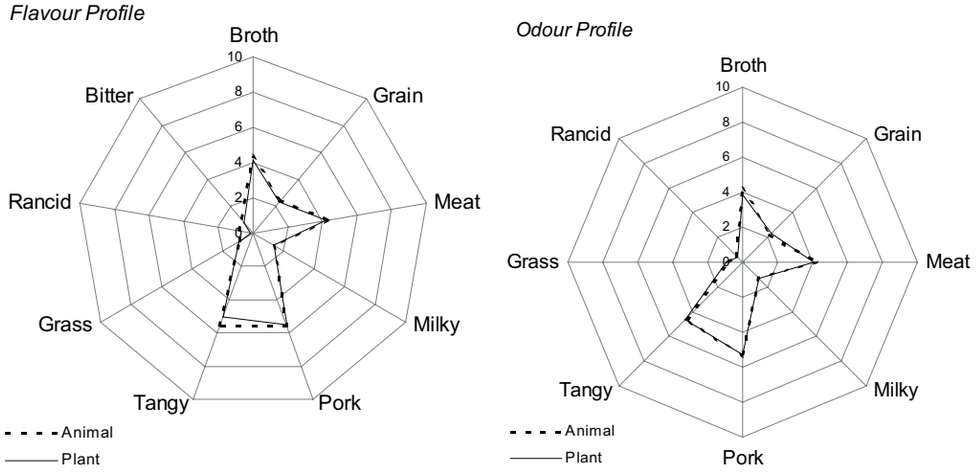


Figure 1. Flavour and odour profiles of pork from pigs fed diets containing animal byproducts or plant material.

Supported by the Foundation for Research, Science and Technology and the New Zealand Pork Industry Board.

References

CAVA, R., RUIZ, J., VENTANAS, J. AND ANTEQUERA, T. (1999). *Meat Science* **52**:165-172.

HANSEN, L.L., AGERHEM, H., ROSENVOLD, K. AND JENSEN, M.T. (2002). *Meat Science* **61**:441-448.

LETTNER, F., WETSCHEREK, W. AND BICKEL, S. (2001). *Die Bodenkultur* **52**:247-254.

MAW, S.J., FOWLER, V.R., HAMILTON, M. AND PETCHEY, A.M. (2001). *Livestock Production Science* **68**:119-130.

Brine enhancement or tender-stretching improves eating quality of leg, loin and shoulder pork roasts

P.J. Walker, H.A. Channon, S.R. Baud and R.D. Warner

Department of Primary Industries, Werribee, Vic. 3030.

Food service companies compete in a market characterised by a broad range of price and quality requirements for restaurants, cafes, hospitality trade, institutional and event catering. Pork wholesalers and food service sector buyers currently operate within a dynamic market-driven system. Specific cuts of pork are purchased on the basis of price ranges that conform to the budget allowance for meal ingredients. The food service sector also requires meat cuts that perform consistently well with regard to eating quality. The lack of clear objective criteria to differentiate pork on its eating quality attributes has contributed to a low demand for pork in the food service sector.

The aim of this study was to investigate the effects of tender-stretch hanging and brine enhancement of pork primals on three boneless roast cuts of pork (rolled shoulder, loin and leg roasts) that are commonly used by the domestic food service sector. Twelve Large White x Landrace female pig carcass (67-73 kg hot carcass weight, P2 of 9-10 mm) were used in a 2 (H: hanging method) x 2 (E: brine enhancement) x 3 (C: cut), factorial design. Carcasses were allocated randomly to hanging method (tender-stretched from aitchbone or Achilles tendon (normal)) within 10 min of reaching the chiller. At 24 h after slaughter, carcasses were boned and boneless, skin-on primal portions (n = 72) of silverside, caudal section of the boneless loin and shoulder blade were obtained. One primal per carcass of each cut was then either left untreated (conventional) or enhanced: 10% extension using a brine containing 1.5% NaCl, 3% sodium tripolyphosphate (Tari P22, BK Guilini) and 30% sodium L-lactate (Purasal S SP60, Purac) using a Fomaco Model FGM2040 needle injector). The primals were then rolled, netted, vacuum packaged and frozen at minus 20°C. Roasts were thawed overnight and then cooked in fan forced ovens set at 180°C to an internal temperature of 71°C and then rested for 20 min before presentation to consumers. A total of 120 panellists were involved with three consumer sessions held with 24 primals per session (8 primals per session). Each panellist evaluated six 3 mm slices from two different roasts per primal (n=720 individual tastings). Consumers rated each of the roast slices on a line scale of juiciness: 0 = very dry to 100 = very juicy; tenderness: 0 = very tough to 100 = very tender; overall liking: 0 = dislike extremely to 100 = like extremely.

Table 1. Consumer scores for juiciness (Juicy), tenderness (Tender) and overall liking (Liking) for boneless leg, loin and shoulder pork roasts derived from carcasses either normally hung (A) or tender-stretched (T) and either enhanced (EN) or not enhanced (Normal).

Cut	Silverside				Loin				Shoulder					
Pump	Normal		EN		Normal		EN		Normal		EN			
Hang	A	T	A	T	A	T	A	T	A	T	A	T	Significance	sed
Tender	26.6	44.2	50.4	66.2	37.5	47.0	75.9	72.1	61.9	63.9	67.3	76.7	H*, E***,C***	6.10
Juicy	26.7	36.3	47.7	63.5	27.9	34.8	53.2	65.8	53.8	59.9	56.7	69.9	H**,E***,C***	6.69
Liking	36.6	51.8	56.1	69.2	35.5	47.9	67.7	72.9	62.7	64.0	63.9	73.5	H*, E***,C***	5.64

*P<0.05; **P<0.01; ***P<0.001, H=hanging method; E=brine enhancement; C=cut:

Tender-stretching from the aitchbone improved consumer scores for tenderness of all roasts by 8.4 points over roasts derived from Achilles-hung carcasses (53.3 *vs.* 61.7; P<0.05). Silverside and loin roasts from carcasses hung using tender-stretching also had higher scores for juiciness and overall liking (P<0.05) than those from Achilles-hung carcasses (Table 1). Brine enhancement improved tenderness of all roasts with this improvement being greater in magnitude than tender-stretching alone. However this effect differed between cuts, with consumer tenderness scores increasing by 32 and 22 points for the loin and silverside, respectively. Less improvement in eating quality resulted from brine enhancement of pork shoulder roasts. This may reflect the higher content of inter- and intramuscular fat of shoulder roasts, resulting in greater juiciness and perceived tenderness after cooking, even when the roasts have not been brine enhanced. Brine enhancement offers the Australian pork industry the greatest potential for improving the eating quality traits of pork. Pork processors and different sectors of the food service industry have provided positive feedback of brine-enhanced pork cuts. Brine enhancement of pork could improve the likelihood of repeat purchases of pork meals from food service menus, provided pork that meets consumer expectations for high eating quality is consistently available.

Supported in part by Australian Pork Limited.

Using a gas mixture of nitrous oxide and carbon dioxide during stunning provides only small improvements to pig welfare

H.A. Channon, P.J. Walker, M.G. Kerr and P.A. Weston

Department of Primary Industries, Werribee, Vic. 3030.

Despite the acceptance of using carbon dioxide (CO_2) as a humane and safe method for stunning pigs, concerns about the animal welfare implications of the practice remain. Nitrous oxide (N_2O) is commonly used in dentistry, paediatrics and obstetrics as a mild sedative and analgesic for humans. Limited investigations indicate N_2O can improve the tenderness of lamb meat (e.g. Cottrell, 2003). Incorporating N_2O into the CO_2 gas mix used to stun pigs could be a simple and effective way to donate N_2O into the musculature and improve pork quality and pig welfare during stunning.

The aim of this experiment was to determine the effects of including N_2O in the CO_2 stunning gas used for pigs on pig welfare during stunning and meat quality, using a 2 x 2 factorial design. The treatments were: stunning gas treatment (S) - 90% CO_2 in air (control) or 90% CO_2: 10% N_2O and exposure time (T) – 2 or 4 min. Forty Large White x Landrace female pigs of 70-90 kg live weight were handled minimally before slaughter and then slaughtered, after overnight lairage, over two slaughter days. All pigs` were stunned using the Butina Dip-Lift CO_2 stunning unit. Adrenaline and noradrenaline analyses were carried out using an ELISA kit (IBL-Hamburg CatCombi ELISA) using blood plasma samples taken at exsanguination and frozen at –80°C. Pigs were videoed as they were lowered into the stunning pit to determine time to loss of posture; time taken to commence the excitation (kicking) phase; duration of kicking; and pig movement following the excitation phase for each animal. Drip loss was determined on the M. *longissimus lumborum* at 24 h after slaughter.

Table 1. Effect of stunning gas treatment on pig welfare during stunning and on the concentrations of adrenaline and noradrenaline in blood plasma.

Stunning treatment	90% CO_2	90% CO_2: 10% N_2O	s.e.d.[1]	P-value
Time to commencement of excitation phase[2]	4.0	0.5	2.52	0.17
Duration of excitation phase[2]	8.65	6.10	1.53	0.10
Pig movement after excitation phase[3]	1.18	0.85	0.141	0.027
Adrenaline (ng/ml)	143.5	76.0	16.36	<0.001
Noradrenaline (ng/ml)	226.9	183.2	13.32	0.002

1. Standard error of difference
2. Time (sec) determined once cage reached the bottom of the stunning pit
3. Intensity scale (0=none; 1=slight; 2=mild; 3=extreme)

Stunning gas treatment did not influence time to loss of posture, with pigs losing posture about 2 sec after the stunning cage reached the bottom of the pit. Although pigs in the 90% CO_2: 10% N_2O treatment entered into the excitation phase about 3.5 sec earlier than pigs stunned with 90% CO_2 in air (0.5 sec and 4.0 sec, respectively), this was not significant (P=0.17) (Table 1). Exposure time did not influence the responses of pigs to the stun gas (data not presented). Pigs stunned with 90% CO_2 in air had higher concentrations of both adrenaline (P<0.001) and noradrenaline (P=0.002) in blood plasma collected immediately after exsanguination than pigs stunned with 90% CO_2: 10% N_2O. These results suggest that adding N_2O into the stunning gas mixture for pigs may have some beneficial welfare effects, albeit small. Drip loss was not influenced by the method of stunning gas treatment (P=0.94), however pigs exposed to stun gas for 4 min had a lower (P=0.084) drip loss than pigs stunned for 2 min (2.57% and 2.04%, respectively).

There were no significant benefits to pork quality when N_2O was included in the stunning gas mixture. Therefore, the commercial adoption of N_2O will ultimately depend on continuing consumer and customer concerns regarding welfare issues of pigs during stunning.

Supported in part by Australian Pork Limited.

References

COTTRELL, J.J. (2003). PhD Thesis. Victoria University, Australia.

Australian Pork

Pig housing affects the fatty acid profile of back fat and belly fat in growing pigs

M. Trezona*, B.P. Mullan*, J.R. Pluske**, D.W. Pethick**, F.R. Dunshea*** and D.N. D'Souza*

*Department of Agriculture, South Perth, WA 6151. **Murdoch University, Murdoch, WA 6150. ***Department of Primary Industries, Werribee, Vic. 3030.

The fatty acid composition of pig tissue is largely a reflection of the fatty acid pattern of the diet, however age and ambient temperature can also have an effect. Meat quality attributes are influenced by the fatty acid composition of subcutaneous, intermuscular and intramuscular fat. Fatty acid concentration influences the firmness of the fat, which in turn affects the appearance and cutting of fresh and processed pork (Tume and D'Souza, 1999). In addition, fat colour and flavour can be affected by the fatty acid profile. Lambooij *et al.* (2004) investigated the effects of housing conditions on pork quality characteristics and concluded that differences in pork quality can be substantial when differences in housing conditions are large. In this study we hypothesised that the environmental differences between conventional and deep litter housing would affect the fatty acid profile of pig fat tissue and that these differences may influence carcass quality and eating quality.

One hundred and fifty two Large White x Landrace female pigs were stratified at weaning by weight into two housing treatments, conventional or deep-litter. Within each treatment, eight pigs were selected randomly as sample pigs. Pigs were phase-fed the same commercial, cereal-based diets *ad libitum*. At 24 weeks of age, pigs were slaughtered in a commercial abattoir. Fat was collected from the hot carcass at the dorsal midline in line with the last rib (subcutaneous back fat) and from the ventral midline in line with the last rib (belly) and stored at minus 80°C until fatty acid profiles were determined via gas chromatography. Data were analysed by ANOVA using Genstat v6.

Table 1. Effect of housing on fatty acid proportions in subcutaneous and belly fat of 24-week old gilts.

	Subcutaneous back fat				Belly			
	C	DL	lsd[1]	P	C	DL	lsd[1]	P
C12:0 (%)	0.090	0.104	0.001	0.019	0.108	0.120	0.013	0.062
C14:0 (%)	1.49	1.67	0.152	0.022	1.72	1.90	0.177	0.048
C16:0 (%)	24.51	25.08	1.121	0.298	25.86	26.75	0.900	0.052
C16:1 (%)	2.41	2.98	0.636	0.074	3.28	3.61	0.722	0.350
C17:0 (%)	0.573	0.432	0.067	<.001	0.459	0.383	0.095	0.108
C18:0 (%)	13.22	12.78	1.592	0.568	12.38	12.64	1.29	0.676
C18:3n3 (%)	1.320	1.392	0.191	0.432	1.115	1.191	0.271	0.559
Saturated (%)	40.71	40.93	2.57	0.853	41.35	42.66	1.904	0.161
Unsaturated (%)	59.29	59.07	2.576	0.853	58.65	57.34	1.904	0.161
Sat:Unsat	0.688	0.696	0.073	0.831	0.707	0.745	0.056	0.173

[1]LSD = Least significant difference

Housing treatment did not affect the percentage of saturated and unsaturated fatty acids or their ratio (Table 1). Belly fat had higher ($P<0.005$) levels of 12:0, 14:0, 16:0, 16:1 and 17:0 than back fat, however the overall percentage of saturated and unsaturated fatty acids, as well as the ratio between the two, did not differ between sites ($P>0.05$). The back fat and belly fat of pigs housed conventionally, had significantly lower percentages of 12:0 (lauric), 14:0 (myristic), 16:0 (palmitic) and 16:1 (palmitoleic) than pigs housed on deep litter and higher levels of 17:0 (margaric). Myristic, palmitic and palmitoleic acids have been positively associated with firmer fat and palmitoleic acid has also been positively associated with pork flavour, flavour liking and overall acceptability. Increased fat yellowness has been associated with reduced levels of palmitic and palmitoleic acids (Maw *et al.*, 2003). The results indicate that there is a difference in the fatty acid profiles of the back fat and belly fat of pigs raised either conventionally or in deep-litter systems. The fatty acids that differ contribute to aspects of fat quality such as firmness, flavour and colour. The results suggest that the effect of housing on fat characteristics may result in differences in carcass fat quality for pigs raised conventionally or on deep litter.

References

LAMBOOIJ, E., HULSEGGE, B., KLONT, R.E., WINKELMAN-GOEDHART, H.A., REIMERT, H.G.M. and KRANEN, R.W. (2004). *Meat Science* **66**: 855-862.

MAW, S.J., FOWLER, V.R., HAMILTON, M. and PETCHEY, A.M. (2003). *Meat Science* **63**: 185-190.

TUME, R. and D'SOUZA, DN. (1999). A review of the impact of carcass fat properties on pork quality. Pig Research Report. A final report prepared for the Pig R&D Corporation, Australia. FSA 4/1377.

Dietary olive oil decreases plasma triglyceride and lipoprotein oxidation and increases bone density in pigs

E. Ostrowska*, N. K. Gabler*, D. Ridley**, D. Suster*, B. G. Tatham*, D. R. Eagling*** and F. R. Dunshea*

*Department of Primary Industries, Werrinee, Vic. 3030, ** Rutherglen, Vic. 3685, *** Knoxfield, Vic. 3176.

Epidemiological studies indicate that in Mediterranean countries there is longer life expectancy and lower incidence of chronic diseases such as cardiovascular disease (CVD) and cancer, despite high dietary fat consumption (Roche *et al.,* 1998). These findings have been attributed to high dietary olive oil consumption, which is high in monounsaturated fatty acids (MUFA). Olive oil also contains at least 30 phenolic compounds (Tuck and Hayball, 2002) and it is unclear whether the higher MUFA or the polyphenol content contributes to the health of the Mediterranean population. Another major health issue is osteoporosis, a bone degenerative disease associated with an ageing population. Dietary lipids play an important part in bone health through both cartilage mineralisation and mediating cellular signalling pathways. Indeed, diets supplemented with olive oil have been shown to enhance phosphorus absorption, retention and bone mineral density (BMD). Therefore, the present study was designed to compare the effects of MUFA and polyphenols in olive oil on risk factors for CVD and bone density, using the growing pig as a model.

Thirty-two crossbred pigs (24.1 kg live weight) were penned individually, allocated to one of four dietary treatments and fed *ad libitum* for 28 days. The diets consisted of a wheat-based basal diet supplemented with either 12% tallow and 7% sunflower oil (TSO); 12% tallow and 7% extra virgin olive oil (TEVO); 19% extra virgin olive oil (EVO); or 19% refined olive oil (RO). The two sources of olive oil had similar fatty acid profiles but the EVO contained twice the phenolic compounds (335 *vs.* 150 ppm caffeic acid units) and less oxidised products (5 *vs.* 9 mEq O_2/kg) than the RO. Blood samples were obtained on days 7, 14 and 28 after a 16 hour fast (Fast) and then three hours after feeding (Fed). Plasma was analysed for triglycerides (TAG) and copper-induced plasma lipid oxidation was used to assess antioxidant status (Gabler *et al.,* 2005). Body composition and bone density were measured at the beginning and end of the study using dual energy X-ray absorptiometry. Data were analysed by ANOVA with pig within treatment fitted as a random effect for plasma data.

Table 1. Effect of dietary lipid and nutritional state on plasma TAG and oxidation and bone density.

	State	TSO	TEVO	EVO	RO	s.e.d [a]	State	Diet	State.Diet
TAG[c] (mmol/L)	Fed	0.50	0.46	0.40	0.43	0.091	<0.001	0.003	0.006
	Fast	1.32	1.16	0.91	0.89				
ET50[d] (mins)	Fed	206	277	236	253	85.1	0.08	0.18	0.50
	Fast	84	247	241	90				
Rmax[e] (abs_{234})	Fed	0.224	0.229	0.156	0.226	0.0246	<0.001	0.09	0.002
	Fast	0.201	0.145	0.155	0.144				
ΔBMD[f] (mg/cm^2)/d	-	1.2	2.5	6.3	5.2	1.87 [b]	-	0.05	-

[a]SED for interaction between diet and nutritional state. [b] SED for effect of diet. [c]Triglyceride. [d] Effective dose to give 50% of response maximum. [e]Absorbance response maximum. Change in bone mineral density.

Plasma TAG were lower (P=0.003) in pigs fed MUFA-rich diets, while the cholesterol profile was not significantly different between the diets (data not shown). Serum obtained after meals from pigs fed EVO diets was more resistant to *in vitro* copper-induced lipid peroxidation. Daily gain, feed intake and lean and fat deposition were not significantly different between the treatments. However, the daily change in bone mass density was higher in pigs fed diets containing olive oil. In conclusion, these data demonstrate that both extra virgin and refined olive oils reduce after meal hypertriglyceridemia, improve oxidation susceptibility and increase bone mineral density in growing pigs.

References

GABLER, N.K., OSTROWSKA, E., STERLING, S.J., JONES, R.B., TATHAM, B.G., EAGLING, D.R., JOIS, M. and DUNSHEA, F.R. (2005). *Journal of the Science of Food and Agriculture* **85**: 154-60

TUCK, K.L. and HAYBALL, P.J. (2002). *Journal of Nutritional Biochemistry* **13***:* 636-644.

ROCHE, M., ZAMPELAS, A., KNAPPER, J.M.E., KAFATOS, A., WEBB, D., BROOKS, C., JACKSON, K.G., GIBNEY, M.J. and WILLIAMS, C.M. (1998). *American Journal of Clinical Nutrition* **68:** 552-560.

Electrolyte balance in grower diets to increase the lean percentage of pigs

D.J. Henman

QAF Meat Industries, Corowa, NSW 2646.

Dourmad and Lebret (2000) showed that including sodium bicarbonate at 0.5-1% and reducing the salt content in a barley/wheat/corn/soy diet improved the calculated lean percentage of the pig carcass from 59-61%. The control diet contained a salt level of 0.45% and had an electrolyte balance (as measured by the MONGIN equation) of 150 milliequivalents (mEq)/kg.

This experiment was designed to test the hypothesis that adding 1% sodium bicarbonate to a standard wheat-based commercial grower (150 mEq/kg) and finisher diet (110 mEq/kg) would reduce the amount of fat deposited and increase the amount of lean in the pig carcass. Salt was removed from the diet and the added sodium bicarbonate lifted the electrolyte balance to 250 mEq/kg. Treatment diets contained twice the sodium content and half the chloride level of the control diet. Eighty commercial crossbred male pigs and 80 female pigs at 10 weeks of age were assigned equally to a control or treatment dietary regime in pens of 20. The pigs were weighed at the start, middle and end of the 98-day experiment. Feed was offered *ad libitum* and audited every seven weeks. Fat depth at the P2- and midline-leg sites was measured using real time ultrasound. The animals were processed at the QAF Meat Industries' abattoir where carcass weight and 'Henessy Chong' P2 fat depth were measured. Dressing percentage was calculated from the live weight and carcass weight measurements of individual pigs.

Table 1. The performance of grower pigs fed a diet containing 0% or 1% sodium bicarbonate.

		Control		Treatment (T)					
		Females	Males	Females	Males	s.e.m.	Sex (S)	T	SxT
Start weight	kg	27.8	26.0	27.8	26.0				
Final weight	kg	91.7	102.2	93.3	100.4	0.91	p<0.05	ns	ns
Rate of gain	g/day	719	817	739	801	18	p<0.05	ns	ns
Feed:Gain	kg/kg	2.61	2.51	2.63	2.38	0.043	p<0.05	ns	ns
Daily feed intake	kg/d	1.875	2.049	1.942	1.903	0.031	ns	ns	ns
Carcass weight	kg	71.1	78.4	73.7	76.5	0.7	p<0.05	ns	ns
P2 fat depth	mm	8.47	9.94	9.89	9.23	0.18	ns	ns	p<0.05
Leg fat depth	mm	11.37	11.63	12.54	10.85	0.24	ns	ns	p<0.05
Fat %		15.33	13.56	15.89	12.91	0.22	p<0.05	ns	ns
Dressing %		77.6	76.7	78.9	76.2	0.18	ns	ns	ns

ns – not significant

There was no difference in performance or fat cover of pigs fed diets containing sodium bicarbonate. There appeared to be some interaction between sex and sodium bicarbonate levels with male pigs having a lower fat deposition when sodium bicarbonate was added to the diet, however there was also a reduction in carcass weight and this may have influenced the fat effects. The results indicate that adding sodium bicarbonate will not increase the amount of lean from pigs produced for markets targeting a 75 kg carcass weight with average P2 level around 10 mm. Further examination of sodium bicarbonate could be warranted for heavy pigs under high heat stress conditions.

DOURMAD, J.Y. and LEBRET, B. (2000*). Journees de la Recherche Porcine en France* **32**:163-168.

Influence of animal size and subregional analyses on the repeatability of pig body composition measurements using dual energy X-ray absorptiometry

D. Suster*, B.J. Leury**, D.J. Kerton* and F.R. Dunshea*

*Department of Primary Industries, Werribee, Vic. 3030. ** The University of Melbourne, Parkville, Vic. 3052.

Dual energy x-ray absorptiometry (DXA) is a reliable and convenient method for determining total and regional fat, lean tissue and bone composition. Previous studies have demonstrated DXA delivers high degrees of accuracy compared to the use of chemical analysis in pigs (Suster *et al*., 2003). Repeatability of measurements is in the range of 2% for fat and 1% for lean tissue mass (Mazess *et al*., 1990). However, animal size (increasing tissue thickness can distort x-ray penetration) and sub-region analysis (sub-regions contain different in-built algorithms) are expected to affect scan repeatability. The aim of this experiment was to investigate the influence of animal size and regional grid placement on the repeatability of DXA measurements.

Fifteen Large White × Landrace male pigs were scanned in triplicate with a Hologic QDR4500A DXA at the beginning of the study (3 weeks of age, live weight 5-10 kg) and then every four weeks until 19 weeks of age. The QDR4500 software allows the scanned image to be divided into head, arms, legs and trunk using an in-built regional analysis grid that contains algorithms unique to each region. Different regional grid manipulations were performed at each scan to evaluate the effects of incorporating sub-regions into a whole body analysis over time. Repeatability in this experiment was measured statistically as the coefficient of variation (CV) of a triplicate set of scans. The CV of DXA measurements was analysed using ANOVA to determine differences between regional grid placements and across different scan times.

Table 1. Coefficient of variation for dual energy X-ray absorptiometry determined body composition of pig of varying age and analysed with different regional grid placements.

Grid placement[a]	1	2	3	s.e.d	Significance
CV (%) of lean tissue mass measurement					
3 weeks (5-10 kg)	1.09	0.60	1.15	0.11	<0.001
7 weeks (10-25 kg)	0.83	0.44	1.01	0.05	<0.001
11 weeks (35-45 kg)	0.32	0.28	0.64	0.03	<0.001
15 weeks (55-75 kg)	0.37	0.47	0.58	0.01	0.007
19 weeks (80-110 kg)	0.60	0.54	0.67	0.04	0.004
CV (%) of fat tissue mass measurement					
3 weeks (5-10 kg)	5.23	3.42	4.79	0.49	0.004
7 weeks (10-25 kg)	5.03	2.59	5.68	0.37	<0.001
11 weeks (35-45 kg)	2.03	1.64	2.68	0.18	<0.001
15 weeks (55-75 kg)	1.67	1.21	1.88	0.15	<0.001
19 weeks (80-110 kg)	1.55	1.45	1.38	0.17	0.632

[a]Grid placement 1, scan image was analysed as closely as possible to the human model using all available sub-regions. Grid placement 2, body in the scan image was placed in the arm region and head in the head region. Grid placement 3, body in the scan image was placed in the trunk region and head in the head region.

The DXA measurements were highly repeatable and were affected by both animal size and regional grid placement (Table 1). When averaged across grid placement, the CV of measurements decreased with increasing animal size for measurements of lean tissue (s.e.d. = 0.04, P<0.001) and more so for fat tissue (s.e.d. = 0.19, P<0.001). The placement of the regional analysis grid influenced the CV of both measurements, however this declined with increasing animal size. Animal placement into the trunk region or using the full regional analysis option as specified by the manufacturer provided less repeatable results than using the arm region. It is recommended that the arm region be used when measuring whole body composition, and particularly fat composition, to obtain the most precise measurements using the Hologic QDR4500 DXA. A degree of caution needs to be expressed in younger animals.

References

MAZESS, R.B., BARDEN, H.S., BISEK, J.P. and HANSON, J. (1990). *American Journal of Clinical Nutrition* **51**:1106-1112.

SUSTER, D., LEURY, B.J., OSTROWSKA, E., BUTLER, K.L., KERTON, D.J., WARK, J.D. and DUNSHEA, F.R. (2003). *Livestock Production Science* **84**:231-242.

Australian Pork

Type of rice fed to pigs after weaning influences apparent digestibility of starch at the ileum but not in the rectum

J.R. Pluske*, L. Montagne**, F.S. Cavaney*, B.P. Mullan***, D.W. Pethick* and D.J. Hampson*

*Murdoch University, Murdoch, WA 6150. **UMRVP, INRA, Rennes, 35042 France. ***Department of Agriculture, South Perth, WA 6151.

Cooked rice is being used commercially in some countries as an alternative cereal to wheat and (or) maize, particularly where dietary growth-promoting antibiotics are no longer permissible (Mateos *et al.*, 2001; Vicente *et al.*, 2004). Many varieties of rice are grown and, as expected, they differ in chemical characteristics such as the amylose: amylopectin ratio and resistant starch (RS) levels. The hypothesis tested in this study was that rice types having a lower amylose:amylopectin and (or) lower RS levels would have a higher apparent digestibility of starch when measured at the ileum but any differences would disappear when ascertained in the distal large intestine.

Forty-eight male pigs aged 19-24 days and weighing 6.7 ± 0.24 kg (mean ± SE) were used in a completely randomised block design having four experimental treatments, with 12 pigs allocated to each. Three rice-based diets differed only in the type of cooked rice fed, which were (i) medium-grain (cv. Amaroo; AM), (ii) long-grain (cv. Doongara; DOON) and (iii) waxy (WAXY). All diets were fortified with a supplement consisting predominately of animal protein sources. A fourth diet comprised a weaner diet based on wheat, barley and lupins (WBL). All diets contained titanium dioxide (TiO_2) as an indigestible marker. Each rice type was cooked in an autoclave at 121°C for 20 minutes using a ratio of 2:1 water:dry rice and was left to cool overnight in a cool room (4°C) before feeding the following day. Diet WBL was fed as a meal. Pigs were fed the experimental diets on an *ad libitum* basis for 14 days, at which time they were euthanased for sample collection. Diets and digesta were measured for starch (Megazyme Total Starch Kit), dry matter and TiO_2 using established procedures. The ANOVA analysis of Statview 5.0 for Windows (SAS Inc.) was used for statistical analysis.

Table 1. Apparent digestibility of starch in the digesta of pigs fed different diets after weaning.

	Diet					
	AM[1]	DOON	WAXY	WBL	s.e.ds[2]	*P*=
Starch digestibility (%)						
Ileum	96.2 [a]	88.6 [b]	99.1 [a]	88.5 [b]	5.78	0.004
Rectum	99.8 [a]	99.8 [a]	99.9 [a]	97.6 [b]	0.55	<0.001

[1]Refer to text for details of diets.
[2]SED: standard error of difference between treatment means.
[abc]Means in the same row lacking a common superscript are significantly different (P<0.05).

The amylose contents of rice types AM, DOON and WAXY were 182, 238 and 61 g/kg and the RS contents (after cooking) were 0.6, 1.42 and 0.75 g/kg dry matter, respectively. Rice types AM and WAXY having the lowest amylose to amylopectin ratio showed superior (P=0.004) digestibility of starch at the terminal ileum than the long-grain rice DOON and the commercial diet (WBL). Differences in apparent starch digestibility between rice types disappeared in the rectum, although pigs fed diet WBL showed a lower digestibility (average of 99.8 % versus 97.6%, P<0.001). Starch digestibility in the ileum between rice types depended on the amylose to amylopectin ratio and the amount of RS but microbial fermentation in the hindgut caused total disappearance of rice starch. Digestibility of starch in pigs fed WBL was incomplete in the rectum, reflecting differences in ingredient composition and physical and chemical properties between diets.

Supported by the Rice Sub-Program of the Rural Industries Research and Development Corporation.

References

MATEOS, G.G., ALCANTARILLA, A., LATORRE, M.A., LAZARO, R., GOMEZ, E. and LASO, N. (2001). *Journal of Animal Science* **79** (Suppl. 1):106-107.

VICENTE, B., VALENCIA, D.G., LAZARO, R., LATORRE, M.A. and MATEOS, G.G. (2004). *Journal of Animal Science* **87** (Suppl. 1):465.

Stress gene expression in growing pigs following respiratory disease challenge

C.A. Kerr*, S.J. Briscoe*, G.J. Eamens** and L.R. Giles**

*CSIRO Livestock Industries, Queensland Biosciences Precinct, St Lucia, Qld. 4067. **NSW Department of Primary Industries, Camden, NSW 2570.

Respiratory disease is common in commercial pig farming, with the most frequent respiratory disease affecting pigs being enzootic pneumonia (EP). *Mycoplasma hyopneumoniae* (Mhp) is the primary agent for EP and secondary bacterial infection particularly by *Pasteurella multocida* (Pm) complicates the infection and contributes to lost production. Determining the expression of key stress genes can be used to assess whole gene networks responding to disease. Genes of interest are those encoding: 1) Proteins that act as suppressors of cytokine signalling (SOCS), including SOCS-1 and SOCS-3 that regulate innate immune responses and SOCS-2, which is associated with growth responses; 2) Heat Shock protein 70 (HSP-70), which is associated with cellular tolerance to temperature; and3) '29a', a novel, short, interspersed element (SINE)-like fragment up-regulated in the cell by physiological stressors. This study aimed to better understand the impact of the two respiratory pathogens on the stress response as measured by gene expression (qRT-PCR). Disease challenge models were developed using Australian field isolates of Mhp (strain Hillcrest) and Pm (strain PM508, supplied by R. Bowles, QDPI). Sixty-four female pigs (hybrid, mainly Large White x Landrace) were allocated at 24.1 ± 4.3 kg (mean ± sd live weight to individual pens in four rooms maintained at 22°C from day - 7. The experiment consisted of four treatments: Control, Pm, Mhp and Mhp followed by Pm. Each treatment group was housed in a separate room. Procedures were undertaken to minimise the risk of cross-infection between rooms. Inoculation with Mhp occurred on day 0 and Pm on day 21. Blood samples were collected on days -1, 23 and 41 for gene expression (Kerr *et al.,* 2005). Data were analysed for ANOVA.

Table 1. Mean gene expression (10^{-3}) and standard error (SE) of SOCS-1, HSP70 and 29a in 64 growing female pigs in no challenge (control), *Pasteurella multocida* (Pm) and *Mycoplasma hyopneumoniae* (Mhp) and combined challenges bled at days –1, 23 and 41.

Gene	Treatment	Day -1 Expression	SE	Day 23 Expression	SE	Day 41 Expression	SE
SOCS-1^	Control	9[c]	3	1	0	63[a]	52
	Pm	7	1	20	10	644	614
	Mhp	6	2	260	257	1,649[a]	760
	Pm+Mhp	20[c]	2	66	34	0	0
HSP-70*	Control	0	0	0	0	11[a]	6
	Pm	1	0	1	0	89	47
	Mhp	1	0	0	0	106[a]	30
	Pm+Mhp	1	0	0	0	0	0
29a*	Control	34[c]	4	85[c]	16	941 [b]	152
	Pm	72	7	459[c]	495	1,960	495
	Mhp	86	6	152	327	1,230	327
	Pm+Mhp	102[c]	18	612[c]	252	189[b]	129

[abc] Results in the same sub-column with superscripts differ ([a]P<0.05, [b]P<0.01,[c]P<0.001).
^ Normalised to ribosomal protein, *normalised to 18S rRNA.

Respiratory disease up-regulated gene expression of SOCS-1, HSP70 and 29a (Table 1). These genes also increased in expression as control animals grew. The combined Mhp + Pm treatment had an additive effect at day 23 for 29a. However, there was no additive effect for HSP70 and SOCS-1. SOCS-2 and SOCS-3 were not expressed in this experiment. The expression of some key stress related genes is indicative of respiratory challenge of at least individual pathogens.

Supported by Australian Pork Limited and a Department of Agriculture, Fisheries and Forestry science award.

References

KERR, C. A., BUNTER, K.L., SEYMOUR, R., SHEN, B. and REVERTER, A. (2005). *Journal of Animal Science* **83:**1753-65.

2 Health and antimicrobial resistance

Symposium- health strategies for the modern pork industry

P.M. Mitchell

"Broad Acres", Prairie, Vic. 3572.

In the past, pig production in many countries has been based on continuous flow, farrow-to-finish management systems usually on one site. Even with careful management, the prevalence of pig pathogens tends to increase over time, reducing efficiency and increasing production costs. Diseases on farms do not occur as a result of a single risk factor in the production cycle but rather as the result of multiple interactions that occur between the pigs, their environment, and the pathogens. Our understanding of the risk factors that influence this intricate system of interactions has increased greatly over the last few years. It is now possible to eradicate specific disease from individual herds by using a combination of management practices, strategic and judicious antibiotic therapy and/or vaccination protocols.

The control of traditional and emerging pathogens in modern pork production systems challenging the pork industry worldwide. The very nature of pig production makes the management of herd health an ever-changing process, and a variety of tools should be used to ensure the health of the pigs. Nevertheless, in the past, antibiotics have tended to be used as the basis of health management with the majority of pharmacological intervention being used as prophylactic treatment (to prevent a disease from occurring) rather than therapeutic treatment (used in the face of a disease outbreak). Consumer and human health advocates increasingly are opposed to this imprudent prophylaxis with pharmaceuticals.

Additionally, antibiotic therapy for some pathogens may be used too late to prevent losses and continued prophylactic use may lead to problems of resistance. Management of *Haemophilus parasuis*, the causative pathogen of Glasser's disease, has been more efficacious through vaccination rather than sole reliance on antibiotics. Moreover, the haemolytic *Escherichia coli* (K88:O149) once isolated only from weaner pigs in Australia, but now unfortunately isolated as often from sucker piglets, is a prime example of a pathogen that on many farms is resistant to all registered antibiotics for use in pigs. Oral vaccination of the dams on most farms offers the most effective control of this insidious pathogen. Increasingly, researchers and producers are seeking to control porcine pathogens through vaccination. Effective vaccines that aid in the control of a number of respiratory and enteric pathogens have been available for several years. In addition to vaccination there are a number of other tools, which can be used as longer-term solutions in the management of herd health. In recent years various production strategies including 'segregated early weaning' (SEW), Swiss depopulation, multisite rearing, all-in/all-out rearing (by room, shed or site) and segregation of the breeding herd by parity have been promoted as a management tool to control respiratory disease. But, pig production operations can differ greatly between different countries and also between farms in the same geographical area. The challenges for our industry regarding the health management of pig herds are substantial.

This symposium will examine what the future may hold with regards to pig herd health. Opportunities for vaccine use in the future are also discussed. In addition, the current health management strategies that are available to assist commercial pig producers to control the spread and the impact of pig pathogens are reviewed.

Disease control challenges for the pig industry – what will the future bring?

R.S. Morris

Massey University EpiCentre, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, Palmerston North, New Zealand.

Abstract

As recent history shows, diseases will continue to evolve in the future at a rate that will stretch our capacity to respond effectively. To understand and respond to these continuing challenges, we must think of disease more in terms of ecological and evolutionary processes. Because of its place in global agricultural ecosystems, the pig will occupy a prominent place in new diseases of public health and food safety importance. In addition, there will be new diseases affecting pigs but not man and all of these developments will be due to emerging and evolving infectious disease agents. Smallholder pig herds will be the main entry point of new diseases, which will be a mix of wildlife-derived agents and apparently novel RNA viruses. The public profile of the new diseases will, in some cases, be out of proportion to their true impact on human health and pig production. However a number of these new diseases will be transferred at some point into the commercial pig industry and will then be disseminated rapidly around the world, circumventing the enhanced biosecurity measures which have been highly successful in reducing past disease problems. As well as novel diseases, we will face new manifestations of familiar diseases, due to changing features of production systems generating unexpected and adverse effects and the impact of external factors, such as climate change. We need to improve the speed and effectiveness with which we identify the true causal factors in emerging diseases because in recent times important new diseases have become global in distribution before we have identified the agent and developed effective control methods.

To control infectious diseases we will make increasing use of vaccines and reduced use of antibiotics, with only slow growth in the range of novel immunological interventions available. However there will continue to be rapid growth in the availability of novel diagnostic methods for specific diseases, which will be valuable tools in control. The use of physical and epidemiological barriers to control and eradicate diseases from individual herds and sub-herds will continue to grow in importance. A major growth area will be the manipulation of risk factors to control multi-factorial diseases, which will increasingly rely on enhanced real-time methods of monitoring the environment, health and productivity of the pig.

There will be some unpleasant surprises in the disease problems we face, which will catch us unawares.

Disease as a dynamic ecological and evolutionary process

If we look back over the past one hundred years, the diseases that are currently of greatest concern to the pig industry have changed substantially, during phases that have typically been in the order of twenty years. During this time, each group of top priority diseases has faded into the background and has been replaced by new challenges. Similarly, the tools that we have used to control diseases have come and gone on a somewhat longer time scale, around forty years. Over this time we have also moved from a situation where many diseases were limited to one region of the world or spread only slowly from one area to another, to the current situation where new diseases disseminate rapidly throughout most of the world in as little as five to ten years.

This is just as true of diseases in man as it is of pigs. For example, in the early twentieth century there were less than 40 diseases in the American Public Health Association handbook on communicable diseases, there are now around 300 and their order of importance is very different.

If we look forward into the future, can we make any predictions about the disease challenges we are likely to face and the tools we can hope to have available to control them? To look deep into our crystal ball we first need to explore the forces that are shaping the evolution of diseases.

All diseases are the result of a continuing process of interaction within what has traditionally been called the agent-host-environment triad, although this is now a rather outdated conceptual framework. Even the newer causal web viewpoint is limited since it considers only risk factors and because diseases evolve in space as well as time. The way we view disease is affected by the way we capture our particular image of it. The pathologist sees only a 'still photograph' of the disease at the instant when the animal died, the clinician sees a 'photo opportunity' over a brief period, and the epidemiologist seeks to reconstruct the complete movie film, with the full cast of players and the entire script - acting out short term (ecological) processes and long term (evolutionary) processes.

The key to understanding the evolution of diseases is to understand the underlying ecological processes that cause changes in the measurable features of disease in pig populations. We therefore need to examine the causal web in a dynamic way, by examining the interaction of space, time and the spectrum of risk factors on the interplay between host, agent and environment in producing disease and also the various ways in which subtle changes in the relationships can cause diseases to evolve. The literature in this area is an incomplete mosaic and what we know comes from many diseases in a variety of species. I will first describe the evolutionary processes which underlie the changing patterns of disease, illustrating with pig disease examples where possible.

There are surprisingly few papers in the scientific literature on the fundamental reasons why these changes have occurred, as distinct from the voluminous literature which examines (disease by disease rather than holistically across the spectrum of diseases) how it has occurred and provides many pieces of the jigsaw but not the complete picture. This paper examines the various factors that have caused the evolution of diseases and draws out implications for what we can expect to happen in the future.

The evolution of disease agents and emergence of new infectious diseases

There is a steady flow of diseases in pigs that are either completely novel or emerging in importance or in various other ways represent a new disease challenge to the pig population. All three of these mechanisms have been influential in recent decades.

Evolution of new diseases and their agents

In disease syndromes for which the evidence suggests that a novel agent has entered the animal population, wildlife species are now the major recognised source, just as they are also for human diseases (Murphy 1998; Weiss 2001). As human settlements encroach more and more on wildlife habitats such transfers can be expected to continue. There is a high risk that diseases endemic in one or more wildlife species will establish in pigs as the normal spatial separation between the species breaks down. In Malaysia, bats have been identified as the source of the Nipah virus, which is infectious for both pigs and man and causes serious and often fatal disease in both species (Chua *et al.*, 2000; Field *et al.,* 2001). Bats are also the source of the related Tioman virus (which was also isolated in Malaysia during investigations into the epidemiology of Nipah) and of the Menangle virus that caused disease in one herd of pigs in Australia and a case in man. Blue eye pig disease in Mexico is another paramyxovirus that occurs sporadically and its possible links to bats deserve exploration. The growing list of bat viruses that have been found to be pathogenic for man and domestic animals makes this group of mammals of special interest as a source of future pig diseases of potential zoonotic importance, although so far we can only speculate on why bats are such a rich source of pathogens for other species.

Diseases could also come from other source species, including species more closely related to the pig (such as the warthog and bush pig in the case of African swine fever), but also species for which exchange of agents seems as unlikely as for bats (for example, vesicular exanthema calicivirus from marine pinnipeds (Neill and Meyer, 1995). In some cases, such as encephalomyocarditis virus, the agent occurs in a range of species and it remains unclear which are the reservoir hosts and which are the spill-over hosts, although in at least some outbreaks rodents appear to be the source for pigs.

Influenza A viruses are currently receiving a high level of international attention, especially the H5N1 epidemic in Asia. Wild birds are the reservoir of these viruses, especially web-footed waterbirds such as ducks and seashore birds. When the viruses are transmitted to domestic poultry, especially chickens, they can increase in virulence and cause very serious disease. Pigs can also become infected and suffer disease and in some cases subsequently put man at high risk (see below).

For most of the known diseases derived from wildlife, pigs have fortunately remained only a 'spill-over host' in which infection relies on continuing transfer from the reservoir species and cannot be maintained within the pig population. In a few cases, such as African swine fever (Sanchez-Vizcaino, 1999), a disease has undergone sufficient epidemiological change that it can now persist in pigs in the long term, without any need for contact with a wildlife reservoir host. When this occurs it presents more serious control problems and efforts to prevent such changes could be very beneficial. When a disease infects a new species and becomes endemic in that species, there is almost always some key triggering epidemiological event or process that allows the new host species to switch from a 'spill-over' to a maintenance host and preventing this could be critically important. Typically this has to do with a switch in transmission processes, as described below.

While we generally are generally only concerned with the possibility of pig diseases infecting man, now that pigs are kept in single-species farms of high health status (where people are the only other mammalian species allowed), it

is likely that some agents of human infections will transfer into pig herds, and perhaps even lead to serious breakdown in the health status of the pig herd. Swine vesicular disease could well have arisen by evolutionary divergence from a human virus (Brocchi *et al.,* 1997; Zhang *et al*., 1999) and other agents that affect pigs now or in the future could either have man as the reservoir host or as the source of pig infection. Hepatitis E virus in pigs and man is an example for which there is a high degree of homology between the viruses isolated from the two species and cross-infection could occur in one or both directions, but the evolutionary history of the agent is not clearly resolved (van der Poel *et al*., 2001).

Having identified some novel pig diseases as coming from wildlife and perhaps rarely from man, it is noteworthy that almost all the other totally new disease agents identified in recent years are of completely unknown origin with no hint of a precursor virus of lower pathogenicity. It is also noteworthy that these agents are typically ones that show high genomic plasticity, principally RNA viruses. Because of the error-prone form of their replication process, RNA viruses form what are termed 'quasi-species' swarms of viral particles that almost all differ slightly from each other. Those that have higher evolutionary or Darwinian fitness continue to replicate (Domingo *et al*., 1998; Lederberg 1998), while others die out. If these viruses get a foothold in a new species, they can rapidly become host-adapted and initiate a new disease. The fact that such totally new agents with no identifiable history continue to arise suggests that they may mostly arise by inter-species transfer, but we have not recognised the source species yet.

Porcine reproductive and respiratory syndrome (PRRS) is an example of an RNA virus which remains an enigma with regard to its origin, made even more intriguing by the fact that two substantially different strains of the agent appear to have evolved independently and the second strain was found to be circulating simultaneously in Europe shortly after the introduction of a single strain from North America (Nelsen *et al*., 1999; Rowland *et al*., 1999; Meng, 2000). Subsequently, a wider range of strains of this virus has become established in various populations. Intriguingly in Europe the disease was only of importance for a few years, whereas in North America it has remained a serious yet gradually changing disease ever since it was first identified during the 1980s. PRRS is one of a series of diseases for which the causal agent took considerable time to identify accurately, with a range of false leads being followed before the true causal virus was found.

The emergence of post-weaning multi-systemic wasting syndrome (PMWS) occurred only a few years after PRRS and spread around the world rapidly just as PRRS had done. It has reached New Zealand, but was detected and controlled before it had spread outside a small initial cluster of herds. PMWS has brought considerable controversy with it both in this region and worldwide, which remains unresolved. Although there is general agreement that porcine circovirus type 2 (PCV2) contributes to the disease, ten years after the disease was first identified there is still intense debate about whether there is an underlying necessary causal agent, yet to be identified. It is difficult to reconcile the findings and interpretation of laboratory studies of the disease with field experience and epidemiological evidence, especially since the disease has shown the features of a spatially spreading epidemic consistent with a novel agent reaching susceptible populations. I will return to the issue of identifying true causal agents for new diseases later in this paper. Perhaps we have to look for the source of such agents outside the farmed domestic animal species and explore more widely the potential mechanisms by which infections with other novel agents are transferred and how transfer could be prevented.

Changes in known disease agents

Some of the notable disease epidemics of recent times have involved evolution of a novel variant of a disease agent from a known disease into a disease with modified characteristics. For example, the emergence of a very highly pig-adapted strain of foot-and-mouth disease (FMD) led to a series of epidemics of the disease in Taiwan that, most unusually, were largely limited to pigs and thereby changed the character of the epidemic and the options for control. Conversely, the O1 Asia pandemic strain that has spread so widely in the past few years appears, from British experience, to involve pigs less readily than ruminants and to be less effective in generating infectious aerosols from pigs. This reduced the likelihood of windborne spread in that epidemic and hence changed the priorities for surveillance activities. FMD is renowned for these epidemiological variations, which can produce very divergent disease patterns due to the continuing evolution of this exceptionally labile RNA virus.

In the past we have tended to equate differences in antigenic characteristics of agents with different patterns and severities of disease. This is true of some agents - for example different serotypes of *Actinobacillus pleuropneumoniae* show differences in pathogenicity and other epidemiological characteristics, as do different enterovirus sero-groups. Usually such differences are linked to the capacity of bacterial cells and viral particles with a different antigenic structure to lock on to attachment sites on cells in particular tissues to counteract host defences or to cause cell damage.

But effects may be subtler than these fairly clear-cut cases. Increasingly, factors such as quantitative variation in numbers of organisms disseminated through different routes of excretion (representing perhaps predilection for multiplication in different mucosal surfaces and different tissue tropisms) could determine the pattern of transmission.

This can happen by raising the size of the dose to which a susceptible host is challenged up to a level that allows an alternative route of infection to come into operation. This can substantially change the character of a disease, because it can push a disease across a threshold to the point where a new mode of transmission becomes dominant and a new disease or new form of a disease is generated. The original single strain of African swine fever virus circulated in warthogs and bush pigs, with the soft ticks of the genus *Ornithodoros* acting as a reservoir and a vector of infection to wild and domestic species of pigs (Sanchez-Vizcaino, 1999). This strain was highly virulent for domestic pigs, but as pigs were exposed to infection by the oral route, the virus began to transmit directly and now has evolved into multiple strains with a full spectrum of virulence, ranging from peracute to mild chronic disease. Carrier pigs rather than ticks have now become the most important source of the virus - a new possibility once the agent did not have such a high case fatality rate.

Influenza A viruses provide an example of even more complex interactions (Ludwig *et al*., 1995; Alexander and Brown 2000; Brown 2000; Guan *et al.,* 2000). Because of their segmented genome, these viruses undergo extensive genetic re-assortment when human and avian viruses infect the same host, leading to new re-assortments with substantially changed character that can have markedly different pathogenicity, host range and principal routes of transmission. The pig is considered the leading species in which much of this re-assortment takes place, because it has cell receptors for both avian and human influenza viruses. This process has produced many new strains over past decades - notably the 1918 pandemic strain that was highly pathogenic for man and also (apparently for the first time) pathogenic for pigs - although pig disease was limited to the United States while human disease was worldwide. Most of the strains that shift between species are re-assortments, but on much less frequent occasions a complete virus can become infectious for a second species, without re-assortment. It appears that once some particular influenza viruses are circulating in pig populations, they become genetically very unstable and this leads to the generation of many variants and increases the risk of a second species jump occurring, notably to man. A feature of the H5N1 Asian virus is that it appears to be poorly infectious for pigs, but more infectious for a range of other mammals. This could be helping to protect against the emergence of a virus capable of causing a human pandemic.

Evolution in the agent-host relationship and the disease process within the host

Changing relationships between the host and the disease agent may induce changes in the agent itself (as described above) or in the disease process and the transmission of infectious agents between hosts.

As a general rule, when a host is exposed only rarely to a particular agent the lack of population selection for innate (genetically determined) resistance and the naïve state of the host animals' immune state produces the most severe form of the disease possible and a wide range of age groups is affected. If exposure is more common, immunological responses are activated in most animals and the age range of susceptibility to the disease narrows and the severity of the disease typically declines. There may be a degree of natural or managerial selection of hosts for innate resistance, particularly in the most severe or troublesome diseases.

Influence of the host on evolution of agents and diseases

For agents with a very stable genotype (such as porcine parvovirus), the disease largely disappears in an endemically stable situation but infection becomes ubiquitous and sporadic incidents occur in pockets of naïve animals. For agents that show moderate potential for evolution of new variants, a wider spectrum of milder strains that can co-exist within the population tends to emerge - often suppressing the most pathogenic strains from clinical expression or leading them to show more cyclic patterns of occurrence as herd immunity waxes and wanes. African swine fever and erysipelas broadly fit this pattern. Where the agent is genetically labile, such as PRRS, influenza A, vesicular stomatitis or FMD (all of which are RNA viruses), the agent tends to evolve new strains (Domingo *et al*., 1998; Lederberg, 1998; Rodriguez *et al.,* 2000) that have reduced immunological cross-protection with the older strains. This means that, as new strains emerge, the host is again susceptible. Such diseases will tend to be among the most troublesome to the producer because they vary capriciously in severity and pattern of occurrence. In addition, vaccines and other control methods can be quite variable and unpredictable in their effectiveness.

Transmission mechanisms

The routes of excretion of disease agents (portals of exit), and the quantitative scale of excretion by each route comprise another important element in the evolution of diseases, which depends principally but not entirely on tissue tropism (Shenk, 1993). Most agents can be excreted by more than one route, but frequently one route predominates under typical circumstances. For example, an agent may principally be excreted by the faecal route, but there may be a low level of respiratory route excretion. However, changes in the antigenic structure may change this so that the volume of infectious material excreted as an aerosol may increase and change the pattern of disease. Respiratory

coronavirus is an example of where a relatively minor change in the transmissible gastroenteritis (TGE) virus produced a variant agent that was transmitted principally by the respiratory route in place of the faecal route and the agent consequently became much less pathogenic. Influenza A viruses provide a good example of an agent where different strains show varied mixes of aerosol and faecal and fomite transmission, with different patterns determined by quite small differences in the hemagglutinin and neuraminidase genes.

The application of some control measures or management changes may reduce one transmission mechanism substantially while leaving another unaltered, perhaps reducing the disease overall but favouring the selection of strains that are more capable (for example) of aerosol transmission. This may cause the disease to rebound in incidence while becoming resistant to control measures that are ineffective against this route of transmission. A good example of the impact of evolutionary changes in industry practice on disease transmission routes is the introduction of widespread artificial insemination into the pig industry in recent years. Classical swine fever is a disease for which the epidemiology had been well characterised, yet transmission in semen had never been identified as a significant route. However the 1997-1998 epidemic in The Netherlands demonstrated that spread in semen distributed from an artificial insemination centre could be a potent new way of seeding infection into a range of herds over a very short period (Elbers *et al.,* 1999).

Transmission dynamics

Diseases can also evolve due to changes in management and housing, which can alter the dynamics of transmission between animals. For diseases in which transmission via the respiratory route is important, the capacity of particular agents to form infectious aerosols is an influential factor in the evolutionary process of the disease. Some agents, such as FMD virus, are capable of forming infectious aerosols that can be carried up to tens of kilometres over land and substantially further over water. For reasons that do not appear to be fully resolved, there are large differences in the capacity of FMD strains to be transmitted by aerosol. This is partly due to the quantitative scale of respiratory route excretion, which determines whether an infective dose reaches a susceptible animal, and also to apparent differences in the capacity of strains to form aerosols that reflect the biological properties of the virus – whether it readily forms infectious droplets with water vapour; whether virus particles coalesce to form infectious packets that exceed the minimum infectious dose; and the distance that droplets formed by the agent can travel as an aerosol. Part of the continuing debate over whether some diseases such as PRRS can be transmitted by wind may be the result of strain variation, with some strains being transmitted effectively between farms while others are not. Over time there is likely to be a selection in favour of strains of an agent that can more readily be transmitted by aerosol since they will spread more widely and be more difficult to control and these will therefore become the dominant strains in the overall pig population.

There are also issues surrounding the distance that various agents can travel but still initiate infection, and this will affect the evolution of diseases on a larger scale. A range of bacteria can be transmitted by aerosol, with the most studied being tuberculosis, but transmission distances are short, and a few metres at most. In general, bacteria are transmitted in dried down particles of proteinaceous material, and these can float in the air for considerable time, allowing transmission without immediate contact. How readily such organisms establish in a susceptible host depends on the aerodynamics of organisms within the respiratory tract, and the size of the infectious dose that is required to establish infection through various entry points (for example, tonsils or alveolus). This can be further complicated by interactions among organisms - for example, prior infection by another agent or exposure to mildly toxic airborne chemicals such as ammonia can reduce the level of activity of respiratory tract cilia and allow small numbers of bacteria to establish an infection in a host that would normally control such challenges. Some viruses (and to a lesser extent Mycoplasmas) can be carried in aerosols within a piggery and to its immediate surrounding area under most conditions, with a more limited range of viruses able to be carried significant distances if meteorological conditions are favourable for creation of a virus plume. The viral plume is then transmitted downwind and may infect pigs or other species that are exposed to infectious doses in the air. This has been extensively documented for the FMD virus, for which pigs typically act as the source and ruminants as the recipients because of their susceptibility to respiratory route exposure and their respiratory tidal volumes, which allow them to 'vacuum clean' small quantities of the virus from the air. Thus while birds were previously thought to be the carrier of the virus, it is now recognised that the virus is spread over long distances in the air. Knowing this, it is now possible to make quite accurate predictions of which farms and which species on these farms are at risk from a particular pig farm in an FMD outbreak. There is anecdotal, and in some cases research evidence, that pseudo-rabies, PRRS and *Mycoplasma hyopneumoniae* can be transmitted by aerosol, probably between as well as within farms, but the epidemiological features of transmission remain surprisingly unclear. It is also unclear why other viruses, which by analogy might be expected to be transmitted in aerosols, are apparently not.

Transmission route interacts with the size of the challenge dose to determine whether a disease is transmitted successfully and whether the infective dose depends on the route of exposure. Infection by the respiratory route may require as little as one bacterial cell or possibly just a few viral particles in some diseases, while for other diseases a

much higher challenge is needed. For the oral route, infective doses are typically orders of magnitude higher than for the respiratory route.

Complex causal webs at animal level

The interaction between dose, route(s) of exposure, strain of disease agent and other factors means that changes in management practices or in environmental conditions in a piggery (air flows, effluent disposal, temperature and humidity), may cause diseases to evolve to a new form. The derivation of respiratory coronavirus from TGE is probably such an example.

It is not just classical infectious diseases that evolve within the world's pig population. Genetic selection practices can also produce an increased tendency toward non-infectious diseases as a side effect, with porcine stress syndrome representing a classic example. In the quest for lean muscle growth, the Hal gene (associated with porcine stress syndrome) was inadvertently selected for but was rapidly eliminated from most breeding pyramids once the nature of the inheritance and a suitable diagnostic system were discovered. Other diseases typically involve more complex interactions between genotype, nutrition and management practices and are more difficult to control – with muscular-skeletal disorders, such as leg weakness syndrome, being a good example of this. Conformation changes may also increase susceptibility to some infectious diseases and it is likely that this has occurred to some degree with pyelonephritis caused by *Actinobaculum suis* (formerly *Eubacterium suis*).

We may also see true biological evolution occurring in a range of other non-infectious diseases, such as mycotoxicoses and diseases caused by physiologically inappropriate feeds or feed compositions. This could arise where feed ingredients are used that are novel for pigs or where changing climatic conditions lead to the appearance of new diseases because plants and their associated fungi are exposed to new situations.

Evolution of diseases at herd level

The evolution of a disease is influenced, as described above, by the interplay between the individual host and disease agents. But the nature and speed of evolutionary change is influenced far more by what happens in the herd and the larger population.

Extensive production systems

Traditional forms of pig production practised across most of the world involve pigs being kept outdoors in small groups and in contact with several other domestic species and also, increasingly, with wild species of birds and mammals. This facilitates the inter-species transfer of diseases, as discussed earlier. In addition, because these outdoor pigs typically recycle food wastes to some degree and commonly scavenge additional feed, they can suffer a range of diseases, such as trichinosis, that are rarely seen in modern commercial production systems. In countries where the majority of commercial pig production is from large-scale intensive operations, these 'smallholder' systems still commonly exist and may threaten national disease status through the persistence or entry of diseases that are exotic to the commercial industry. Some diseases of this type may persist very successfully in slowly mixing populations of pigs that are under limited supervision and that may have very slow population turnover with few, if any, animals subject to meat inspection or veterinary care. In such systems, boars are commonly used across multiple herds and can be responsible for the maintenance of various disease agents.

Herds of this kind co-exist geographically with, and often outnumber, intensive production systems in all pig-producing countries and yet produce only a tiny fraction of pork entering the marketing chain. Such systems typically practise minimal biosecurity and as such represent an ongoing risk for commercial units as sources of disease agents, such as *Mycoplasma hyopneumoniae,* from which the commercial units were previously free. Even if there is no transfer of infection, the acceptance of a country or a herd as free of a particular pathogen may be put in jeopardy by the existence of that pathogen in a discrete but interspersed non-commercial population. Although the merits of outdoor extensive pig production have been promoted considerably, this system carries with it a much greater overall disease threat to other pigs and, based on historical experience, to the emergence of serious, zoonotic disease hazards. This phenomenon is not just limited to contagious diseases, but can include, for example, Clostridial infections, which are far more common in outdoor units.

Feral pig populations also exist in most countries, and it is not unusual for feral pigs to either have unintended contact with 'free-range' pigs or to be domesticated into such units, thus adding to the interchange of diseases between feral populations and smallholder pigs.

Intensive production systems

Typically, large-scale intensive pig operations have a much narrower range of pathogens than do outdoor extensive units. Moreover, the range of pathogens in large-scale units is decreasing as biosecurity is enhanced, age groups are separated at different locations and agent-specific eradication is undertaken by methods apart from herd depopulation, such as Swiss depopulation and mange eradication by herd treatment. However the very substantial gains that have been achieved via large-scale units have also induced various new means of disease evolution. The spread of PRRS and PMWS through such units, despite intensive health precautions, demonstrates that microbes abhor a vacuum and agents and transmission pathways will tend to evolve to take advantage of the special circumstances existing in herds of higher health status.

Several factors enhance the evolutionary potential for new diseases to emerge in such systems. The first is large group sizes and large herd sizes. In small herds, pathogens that produce strong immunity (in survivors) after infection will die out because the number of susceptible hosts falls below the threshold required to maintain infection. For the disease to be maintained in the population, the infection must be re-introduced periodically from other herds (Haggett 2000). As herds, and particularly groups of pigs within a herd, which have effective epidemiological contact get larger, more and more diseases can maintain within the population because there are always sufficient susceptible hosts. This maintenance threshold can be pushed higher by separating age groups, but this tends to increase group size again and consequently the new maintenance threshold is exceeded. Not only does this process ensure maintenance of agents that are epidemiologically adapted to this situation, but it encourages the emergence of variant strains of agents, which can co-exist better with whatever management enhancement we introduce in large herds. These herds are certainly of much higher health status, but they have their own particular pathogen flora that has adapted to the new circumstances. In some cases these flora may induce increased incidence of some diseases, such as *Streptococcus suis* Type 2, but in other cases it may be beneficial. The emergence of respiratory coronavirus appears to have provided protection against the closely related transmissible gastroenteritis virus and hence has reduced the severity of this disease in herds where the respiratory coronavirus is endemic.

High health status systems

Systems with high health status raise the biosecurity stakes considerably. While animals in herds with lower health status are exposed to most pathogens in the herd while they still have waning colostral immunity, animals in high health status herds are in specific age groups that are fully susceptible to a pathogen well beyond the normal age of exposure. If these animals are then exposed to the pathogen, the disease can be far more serious than in the partially protected younger animal.

While very advantageous, the removal of particular pathogens from the pig environment can cause remaining diseases to evolve into new forms or to new levels of severity because the competitive exclusion process that previously operated has been reduced or eliminated. It has been proposed that the emergence of *Salmonella enteritidis* as an egg-borne pathogen of principally zoonotic importance in commercial poultry may have been an unintended consequence of removing the poultry pathogen *Salmonella gallinarum* from commercial flocks (Rabsch *et al.,* 2000). In addition, it seems likely (although unproven) that the growth in importance of *Strep. suis* Type 2 in growing pigs is a consequence of the decline or elimination of commensal organisms such as other streptococci, which previously induced cross-protection or occupied an ecological niche in the animal that shielded it from exposure to more pathogenic competitors. Now that we know that the commensal *Erysipelothrix tonsillarum* is different from *E. rhusiopathiae*, it is tempting to suggest that the answer to the frustrating outbreaks of erysipelas that have occurred in adult sows in various countries in recent years could be associated with the changing ecological balance between these two organisms.

Layers upon layers of diseases

It could be that the apparent change in disease importance in high health status herds could result from the uncovering of less serious, but also less tractable problems, once the larger disease issues have been cleared away. For example, while the decline in incidence and clinical severity of swine dysentery (*Brachyspira hyodysenteriae*) over recent decades has removed a large problem, it has also allowed the less pathogenic Brachyspiras (*B. pilosicoli*) to be identified as the cause of residual diarrhoeic diseases or as simple commensals (*B. murdochii*) and has uncovered *Lawsonia intracellularis* as a new disease agent. It is likely that these organisms were always present but were not identified until the incidence of swine dysentery was reduced. It is not unexpected that these residual organisms are difficult to grow or differentiate, and occur in multiple host species rather than being pig-specific, nor that they are difficult to eliminate from pig herds by standard hygiene or disease control methods. This phenomenon would also be expected for other diseases and it could be that some of the confusing findings of recent years about whether or not *Mycoplasma hyopneumoniae* is present in particular herds may relate to the emergence of related but different Mycoplasmas that

mimic some aspects of enzootic pneumonia, but do not produce the pathogenic form of the disease. Similarly, the periodic outbreaks of abortions and stillbirths in herds, which fit the classic temporal pattern of a mini-epidemic but cannot be explained by any of the known pathogens, are probably due to multiple agents that are as difficult and frustrating to investigate as *Lawsonia* was for many years. We can only hope that they will one day be characterised adequately, just as the enteric pathogens have gradually been. In these cases what is evolving is not so much the disease but the diagnostic insights of veterinarians.

Complex causal webs at herd level

Most of the diseases we deal with in intensive production systems have multiple component causes (Rothman and Greenland 1998). Some of these may be necessary causes, in that the disease cannot occur without them being present, but they are not sufficient causes when acting alone. Koch's postulates, therefore, have only limited relevance to current examinations of disease causation. Diseases for which the presence of the agent alone is both a necessary and sufficient cause for expression of a disease are usually the easiest to control. These diseases have been removed from intensive units, leaving only those with more complex causal webs as our current challenge. One reason why veterinarians become confused by apparently conflicting research and field data about disease causation is that there are often multiple component causes of disease expression. In addition, various mixes of these may create sufficient cause - making it seem as if the disease occurrence is capricious and is producing apparently inconsistent results between studies - simply because different component causes have varied in the different investigations. Commonly (but not uniformly) one or more of the component causes are necessary causes and several more are contributory causes, which in various mixes with the necessary cause(s) will add up to sufficient cause and result in disease. The issue is further clouded by the difficulty in studying causes directly and needing to find indirect ways of measuring them (known as risk factors). Risk factors are proxies for causes that vary in strength of association with the true causal factor and their relationship to these true causes has sensitivity and specificity, just like a diagnostic test. They can also be biased, which complicates interpretation, but the objective of epidemiology is to choose the correct control strategy despite all of these difficulties. When we run out of readily measurable risk factors to explain a disease, we throw in 'stress' as the wildcard factor, which although valid is one of the most difficult factors to measure and is often used as an escape hatch rather than seeking explanatory variables that will represent specific stressors. Furthermore, attempts to measure stress may be stressful in themselves.

Enzootic pneumonia provides a good example of complex causality. *Mycoplasma hyopneumoniae* is a necessary cause and the disease does not exist in the absence of this agent. But many herds in which the agent is present show no expression of clinical disease, while in others clinical disease is a constant problem. Many risk factors have been identified (Stark 2000), with some of these inconsistent with each other and while some of these are undoubtedly causal others are close or distant proxies for the true causal factors. Manipulating some risk factors singly may control the disease (especially if they are necessary causes), but commonly it is necessary to modify multiple factors together because of biological interactions among the factors. Complex causality is not limited to infectious diseases, with tail biting in growing pigs an ideal example of a non-infectious condition that has a complex and sometimes very confusing cascade of causal influences.

As diseases evolve and some easier diseases are eradicated, we can expect that these more complex disease control strategies will be the only way to make progress and we will need a much better understanding of causal complexes if we are to make genuine progress.

Evolution of diseases at the ecosystem level and global level

When most people think of disease evolution, they think only of large scale trends and new diseases sweeping the world. In this paper I have shown how such headline grabbing events are the outcome of deeper epidemiological processes that are occurring continuously but are widely recognised only when they surface as an apparently new disease or an epidemic in a new location.

Interacting populations

Large-scale evolutionary trends in disease occurrence are, however, influenced by additional factors that manifest only at an ecosystem level and above. Some diseases undergo changes in expression because of links between different animal populations, as has occurred over recent decades with classical swine fever (CSF) and hog cholera in Europe. Changes in management systems for pigs, trends in populations of wild boar and community attitudes towards them (Laddomada, 2000) have all exerted an influence. Varied interactions between wild and domestic populations have produced regular spill-over of CSF from wild boar populations into domestic pigs and wide dissemination of infection through animal transport, producing events such as the 1997/98 CSF epidemic in The Netherlands, and many smaller

outbreaks in various other European countries. This is just one of many examples of ecosystem level processes in disease evolution and it is clear from the number of major disease epidemics in recent years that although we have protected some parts of the pig population from diseases to which they were previously exposed, pigs still play a prominent part in epidemics of animal disease around the world, which in some cases spill over into human populations. This is likely to continue into the future, although we will see further evolution in the particular diseases that make their presence widely felt and in the epidemiological patterns they express.

Trade influences

The single most important factor in the large-scale disease trends of recent years has been the expansion of trade in animals, animal products and feed materials. Previously, pigs used to be traded extensively only at local level and this ensured pathogens were mixed only between farms in a particular area. However, more recently such local mixing of pathogens through saleyards and other mechanisms has become limited to the smallholder production systems that represent a declining proportion of total pig production. Now, diseases move long distances quite quickly via both legal trade and the illicit movement of animals and their products. This has produced new patterns of disease movement so that diseases like PRRS and PMWS can start in one small area and then over a few years become widely travelled. While in some cases a disease may arise independently in multiple locations due to the wide, simultaneous adoption of a new practice, most of the major expansion of diseases in recent times have been caused by increased trade rather than because of the intensification of production - as is often suggested by critics of current production systems.

Changing global population structure

An issue requiring further epidemiological investigation is the degree to which interchange of genetic material and hence some of the diseases present within particular breeding pyramids occurs between modern global breeding companies. While each company is geographically distinct and protects against the possibility of diseases being distributed via pigs and semen to the herds of their customers, this can only be done for known diseases. In the future we can expect to see new diseases arising and we may well see examples where genetic improvement practices in the industry result in wide dissemination of disease agents which are not recognised as important until it is too late to stop their spread. It will be important to develop risk management strategies that can minimise the damage and that can create protected sub-populations within pyramids, which are likely to remain free of such novel agents even if they are discovered in the main population.

Climatic effects

If global warming changes climatic conditions to a significant extent in most parts of the world, then this will, without doubt, have a substantial impact on the distribution of pig diseases. The most obvious effect will be on insect-borne diseases (Gubler, 1998), which are likely to become evident in areas previously disease-free. This has already occurred with Japanese B encephalitis, which has extended its range to the northern tip of Australia. Because animal populations in previously disease-free areas are fully susceptible, disease expression will initially be more extreme than in areas where the agent has long been endemic. In addition, for zoonotic diseases, such as Japanese B encephalitis, we will see human cases of infection. Extended disease range will not just be to contiguous areas since movement via aircraft and other means may well produce islands of insect vectors that are long distances from the disease source. These insects may then find a favourable environment and spread outwards. West Nile virus in North America is an example of this phenomenon, and it is likely we will see more examples arise in the future.

However, it is not just insect-borne diseases that will be affected by climate change. Many other diseases are influenced by climate and we can expect the geographical distribution of many diseases to shift if temperature and rainfall patterns change. In any particular area some diseases will decline in importance while others will grow. Because changes are likely to be spatially complex and occur in steps rather than slow progressions, it is not easy to make specific predictions about the likely changes - it will, however, be important to observe, interpret and respond as they occur.

Changes in the practical significance of diseases

In considering the evolution of diseases, we cannot exclude an examination of likely changes in the economic and social significance of diseases, since this will influence how veterinary effort is directed. Previously, the impact of disease on animal productivity has been the dominant driver of veterinary effort for the pig industry. More recently, this driver has been subsumed into the larger issue of net economic benefit from animal health interventions, which represents a significantly different criterion for decision-making.

It is now very clear that the risk to human health through food safety (both real and 'perceived but unsubstantiated') is progressively taking over and will become the principal driver in coming years, with animal impacts being secondary despite the adverse implications of this change for animal welfare. This will change the ranking with which diseases are acted upon and in many cases will shift the emphasis from clinical issues to monitoring and management of microbiological and chemical hazards. This will cause a significant evolution in the approach to health care of pig herds. With greater international movement of pigs and pig products we will also see diseases that affect this trade (or are perceived to do so) being given higher priority.

We can expect further evolution in how diseases and their agents are seen as important, depending on a mix of scientifically valid considerations and socio-political pressures. It might be expected that the animal welfare impact of diseases would receive increasing attention, but experience so far suggests that disease is a blind spot in the priority setting of animal welfare activists, despite its substantial importance to the true welfare of animals.

Changes in available control methods

Presently, control of infectious diseases of pigs relies heavily on vaccines and antibiotics. But the use of antibiotics is steadily declining and there is continuing pressure to reduce their use in pig production and to be more selective in their application. This pressure is based in part on valid concerns about emergence of resistant strains of human and animal pathogens, but it is also overlaid with issues of public perception that are not scientifically sound. In consequence, we are in danger of having an excessively restricted access to antibiotics in the future, which will force changes in the approach to disease control. In Denmark, restrictions are already extremely tight and the industry has responded successfully by applying a wider range of control techniques, although in some cases it has been necessary to accept higher levels of disease than would otherwise be the case. Anti-viral drugs will increasingly become available in the future, but I consider it unlikely that they will find substantial use in pig production.

The range and effectiveness of vaccines for pig diseases have grown rapidly in recent years. For example, vaccines against *Mycoplasma hyopneumoniae* have enabled the control method for enzootic pneumonia to be changed from one based on antibiotic use and housing modification to vaccination. However, despite intensive efforts, several other diseases have not yet had successful vaccines developed and we can expect a gradual expansion of the range of highly protective vaccines, rather than rapid breakthroughs. Genetically engineered vaccines have been touted for many years as the way of the future, but the number of such vaccines that have been market successes has been small. Such vaccines will, at best, only slowly replace or augment the existing range of vaccines. Several other molecular immunological techniques have also been proposed for future disease control but, as yet, there is little sign that the promise will be achieved in the foreseeable future.

Molecular techniques have, however, been adopted rapidly for disease diagnosis, ranging from new serological tests through to rapid methods of agent identification through to procedures based on strain characterisation for epidemiological investigations. These methods have changed disease diagnosis substantially for the better and made possible control strategies that were previously impossible. I therefore see such techniques playing a growing and beneficial role in the development of more refined methods for disease control and eradication at the herd level.

In recent years, the single biggest health gain in the pig industry has been the adoption of a range of what I call 'barrier methods'. Barrier methods raise the health status of a herd or sub-groups within a herd by shielding animals from exposure to disease agents. By eliminating one or more agents from the group and maintaining this freedom by separating age groups or by otherwise modifying exposure, animals remain free of certain diseases. Traditionally, depopulating an entire herd and replacing it with breeding stock from a herd of higher status has enhanced the health status of a herd. However, because of down time, this is a very costly approach. Techniques such as Swiss depopulation are much less expensive and use a more epidemiologically precise approach to eliminate agents. 'All-in-all-out' and multi-site production systems have also become increasingly common, especially in North America where PRRS has caused major shocks.

For multi-factorial diseases, where elimination of the agent is not a practical possibility, managing risk factors to minimise diseases has become the preferred approach. In the past, such diseases were often treated with antibiotics – and often with only limited success due to their multifactorial nature. As better information has become available on risk factors and their interaction in causing disease, manipulation of the environment and management practices has become the preferred control strategy.

As the pig industry placed increasing reliance on a small number of sources of genetic material, genetic disorders such as malignant hyperthermia and leg weakness syndrome, grew in importance and appeared much more widely than before. In part this was due to the narrow focus of genetic selection programs on rapid growth in market pigs. However, as these problems have been recognised and selection criteria widened to take them into account,

genetic disorders have become less prominent. Nevertheless, the recognition and management of emerging genetic disorders remains an important task for breeding companies.

A dynamic systems view of diseases and their relationship to the production ecosystem

To understand adequately the past and current evolution of diseases in the world's pig industry and make useful predictions of future trends we need to broaden the focus of our investigational approaches. An appraisal of the pig health literature suggests that there has been excessive emphasis on laboratory investigation of disease using methods that fall well short of what is required to understand the evolution of diseases and the significance of the various epidemiological factors involved. Diseases are very dynamic processes on the small ecological and larger evolutionary scales, especially in an industry that is changing as fast as the pig industry. We need to think of pig units as dynamic ecosystems and analyse them accordingly (Pahl-Wostl, 1995).

Diseases fall into a limited number of groups with each group using different ecological methods to maintain itself in the global pig population. Some diseases have relatively low Darwinian fitness in modern pig production systems and are relatively easy to eliminate. But the diseases of importance for the future have high Darwinian fitness and consitute those dieases that have multiple transmission pathways; effective methods of survival in the environment or the pig; and a tenacity for adaptation to changes in pig management and all the disease control strategies we can apply to them.

Such diseases are almost all epidemiologically complex and display a complex cascade of causality, rather than the linear model we find easier to deal with. They are complex dynamic systems and we need to look at them in this way. To investigators of pig diseases, PRRS (and proliferative enteropathy before it) should have been a timely warning that traditional methods of disease investigation were failing us and that our approaches were not fast or creative enough to deal effectively with the disease challenge.

PMWS provides us with a current challenge for which laboratory research findings cannot be reconciled at all well with the field reality. And what about the next syndrome? In each of these cases, a range of laboratory researchers have claimed success in identifying the causal agent (often identifying different agents!), when field experience indicated otherwise and the answer has turned out to be quite different from these premature claims.

It is striking that, despite greatly enhanced biosecurity in the commercial pig industry, we have seen rapid spread of new diseases such as PRRS and PMWS around the world within a few years and in a much shorter period than was taken to identify the true causal agent of each syndrome. We need more appropriate strategies for investigating the evolution of new diseases.

A perspective on the likely future evolution of diseases in the world pig industry

We can expect to face a continuing supply of epidemiological challenges in protecting the health of the global pig industry, as new diseases arise and old ones change their character to meet the challenges of surviving in a world populated by people who are forever trying to eliminate or control them. This is a battle that neither side will win and for which we can only maintain an acceptable equilibrium. We need to think about disease in terms of all the component processes that enable diseases to evolve and to survive efforts to shift the balance our way.

This paper has considered the factors that influence the man(ager)-pig-disease triad, which together with the host-agent-environment triad, operates from the molecular through to the global level to result in the disease processes we have to deal with.

There is only one safe prediction that can be made about future disease patterns – that we will always be wrong in our predictions of the diseases that will be our main problems in the future. However, by looking deeper at evolutionary pressures and epidemiological processes, we can foretell what we will *need to know* if we are to be better prepared to unravel the next pestilence than we were for the last twenty we have faced.

What lies ahead?

- I expect the pig to figure prominently on the international scene in new diseases of human health importance, including those that affect food safety. This is a direct consequence of the place of pigs within agricultural ecosystems. Linked to this, I see smallholder systems wrongly tainting commercial pig production with disease hazards, since smallholder systems are where these issues principally occur and their impact on public perception far outweighs their contribution to the pork on supermarket shelves.

- I see a need to develop enhanced risk management strategies for diseases that may be spread through movement of genetic material.
- I see diseases changing faster than our capacity to respond and hence I also see a constant supply of new challenges.
- I see slow growth in the availability of 'quick fix' solutions to disease problems, such as new vaccines and medications, and hence I see control methods moving increasingly towards management of subtler risk factors as a control strategy. This will be linked to the use of more refined methods of herd monitoring of productivity and health, including environmental monitoring. Real-time data processing will become increasingly commo and real-time analysis and interpretation of data will become increasingly important.
- And finally I expect that there will be some unpleasant surprises, which will catch us unawares.

Vaccines - what are the future options for health and disease control?

S.J. Driesen, V.A. Fahy and P.M. Mitchell

Department of Primary Industries, Epsom, Vic. 3551.

Summary

Vaccines offer pig producers a cost-effective way to protect their animals against the onset and progression of infectious diseases. The future development of animal vaccines will centre principally on the adoption of technologies that better address epidemic and pandemic diseases confronting the human race. Safer and more effective vaccines will stem from a better understanding of the biological mechanisms pathogens use to cause disease and how the immune system responds to these. In addition, the complex immune system that protects the body must be targeted correctly to ensure that the correct protective defensive response is activated.

Vaccines differ in the way they target pathogens, and also in their mode of action and the immune response they elicit. Protection against bacterial pathogens has usually been achieved by targeting virulence factors such as adhesion or toxins using either whole cell, subunit, recombinant or toxoid vaccines. Whole cell vaccines (WCV), both inactivated and live, have proven they offer a low cost and effective means of preventing respiratory, enteric and systemic infections with minimal side effects. A distinct advantage of WCV is that they express many antigens (not all protective), some of which have not even been recognised.

Mucosal vaccination offers several benefits over the parenteral route, including ease of administration. While vaccines delivered orally or nasally have the potential to stimulate both mucosal and systemic immunity, they must also overcome the natural defence mechanisms of the body. Mucosal delivery of live vectors, especially those expressing multiple foreign surface antigens that can infect host cells, provide the greatest promise. It is fairly well established that soluble antigens given by the mucosal route are poorly immunogenic. Accordingly, strong mucosal adjuvants and improved delivery systems need to be considered to overcome the need for live vaccines, the dilution effect and vaccine degradation. Oral delivery of inactivated whole cell/purified proteins/DNA vaccines may be a future option not only for enteric pathogens but also for respiratory pathogens, particularly as the M cells of the mucosal lymphoid tissue are highly efficient in the uptake of particulate antigens and microparticles.

Technologies that better define protective antigens the expression of protective antigens in live vectors, more effective adjuvants and delivery systems, mucosal vaccines that target specific lymphoid tissue, and the co-administration of animals with more than one antigen, will provide the industry with the enhanced products of the future. In addition to vaccine development, scientists and veterinarians alike must continue to undertake concurrent investigations into disease management and treatment. Above all, vaccines improve animal welfare and reduce the cost of production through improved growth rates and feed conversion efficiency.

Introduction

We have adopted one essential philosophy in our investigations, that eventually the most successful immunisation schedules may well be attained if the natural aspects of immunity are adhered to. In this respect it is as important to know as much about the immune response to the live pathogen as it is to any model antigen, and that this should provide the frame work within which immunisation schedules should be tested. (Porter *et al.*, 1980).

The introduction of vaccines has been one of the most significant technological advances, and has led to a reduction in the incidence of many diseases. Vaccines are unique in that they offer effective protection against the onset and progression of infectious diseases. Most other medications are therapeutic and are used not to prevent the disease but to treat a disease and/or its symptoms (Pastoret and Jones, 2004). The development and use of safe, effective vaccines has and will continue to contribute significantly to improving production efficiency and sustainability of the pig industry, and allow us to compete globally against other pig producing countries.

Pathogens have several distinct effects on the body. The most noticeable effect is overt illness resulting in clinical symptoms such as depression, fever, vomiting, diarrhoea etc. The second and more insidious effect of pathogens is subclinical disease (such as enzootic pneumonia), which results in reduced growth rates and feed conversion efficiencies. A less outwardly obvious impact of pathogens is the immune response they elicit in the infected host. When a pathogen infects an animal host, the animal's immune response increases in strength over time, reducing the number

of infecting pathogens until symptoms disappear and the host generally recovers from the infection. However, by the time this has occurred there will invariably have been significant production losses and, in continuous flow herds, many more animals will have become infected.

Mechanisms of immunity

An individual's natural immunity is like a library that keeps a record of every pathogen that its immune system has ever encountered. If the immune system comes in to contact with a particular infectious agent again, it can destroy the pathogen quickly as it seeks to enter the body. In most cases, the pathogen is killed before it has had a chance to multiply and produce any symptoms of illness. This explains why (in most cases) diseases are only caught once, even though exposure to the pathogen may occur many times. An exception to this is the influenza virus, which undergoes antigenic shift and/or drift at least every seven years.

Vaccines are designed to stimulate the immune system to protect against potential pathogens without the need for disease to occur. However, in contrast to natural infection, it will take several doses of vaccine for immunity to develop following vaccination. A variation on this is when the vaccine is composed of a live avirulent strain or vector. An effective vaccine will stimulate the immune system to develop antibodies and/or activated cells that will effectively kill or neutralise the pathogen before disease occurs.

Role of vaccines

Many issues, some of which have been hotly debated for some time, impact on the decision to use vaccines and the future role they will play in improving pig health. First and foremost is the phasing out or reduced use of growth promotants in everyday pig production. This phasing out is an attempt to reduce any possible environmental risk and the emergence of antibiotic resistant 'super bugs', which have significant human health implications (Dean, 1989). Lobby groups opposed to the widespread use of antibiotic growth promotants have raised consumer awareness of these issues, and because of this there will be a future requirement to produce products perceived as 'clean and green'. Often the mechanism of action of these growth promotants has been to mask the effects of subclinical disease, such as ileitis or swine dysentery, and once the growth promotants are no longer available vaccines will be needed to control such diseases.

Other factors that will fashion the future direction of vaccines include: market demand; cost-benefit to the producer; profit for pharmaceutical companies; the type and effectiveness of vaccines; regulatory requirements; and adverse public perceptions of genetically manipulated vaccines. The relatively poor potential for profit from vaccines compared with pharmaceutical products has previously impacted on product development for the industry. However in the current consumer climate it is likely that we will witness the development of new vaccines, especially through improvements in current technologies and a better understanding of the pathogen-host relationship. It would be naïve to expect that a vaccine that protected against say, ileitis, would come on to the market for less than the current cost of chemical control. However, even at the same cost, vaccines hold the advantage of being environmentally friendly as they improve the welfare of animals by reducing death and illness from disease and they can give significant gains in average daily gain and feed conversion efficiency (Molitor *et al.*, 2005).

Regulation of pharmaceutical compounds

In Australia, all agricultural and veterinary products must be registered by the Australian Pesticides and Veterinary Medicines Authority (APVMA - www.apvma.gov.au) under the National Registration Scheme. Vaccines are assessed and required to pass stringent quality, safety (both human and animal) and efficacy requirements before permits are issued for their use. Efficacy data especially have to be generated in pen and field trials carried out within varying climactic regions in Australia. Safety data from overseas studies are accepted in registration dossiers. However, the registration administrators have never forgotten the loss in average daily gain on many poultry farms caused after registering a coccidostat for chickens based on safety data generated using northern hemisphere genotypes. After this incident the use of local safety data is vital to support the registration process.

In New Zealand, the Agricultural Compounds and Veterinary Medicines Group (ACVM) oversees regulatory control of agricultural compounds and their importation, manufacture, sale and use on behalf of the New Zealand Food Safety Authority (NZFSA - www.nzfsa.govt.nz/acvm).

In recent years, the APVMA has allowed minor use permits for the production of autogenous vaccines in facilities registered for Good Manufacturing Procedure (GMP). Justification for use of autogenous vaccines includes failure of protection by registered products or the absence of a registered product. Applications for autogenous

vaccines must also include information on methods of vaccine, production and formulation, and information on efficacy where it is available. All other requirements and procedures for autogenous vaccines are as stringent as those required for registered commercial vaccines.

Defence mechanisms

The immune system is a collection of special cells and chemicals that respond in a co-ordinated way to defend the body against various pathogens. The body can gain immunity against certain diseases either naturally (by becoming infected and surviving the illness) or through vaccination.

One of the remarkable things about the immune system is its ability to recognise many millions of foreign molecules and to respond by producing antibodies and cells that can match and counteract each one of the foreign molecules. Any substance capable of triggering an immune response is known as an antigen. An antigen can be a bacterium or a virus, or even a portion or product of one of these organisms.

When responding to a natural infection, the immune system homes in on antigens (usually proteins or carbohydrates) that are produced by the causative organism and recognised as being non-self. The immune system can be divided into two lines of defence; non-specific (or innate) and specific (or acquired).

Non-specific immunity

Non-specific immunity is not reliant on specific antigen recognition and is provided by anatomical and physiological factors as well as passively derived immunity. Non-specific protective mechanisms provide a barrier to prevent harmful substances from entering the body or, if the external barriers are breached, can eliminate them before the onset of disease. The anatomical features that provide initial defence and prevent foreign materials from entry into the body are: the skin (a waterproof barrier that secretes oil with bacteria-killing properties); the mucous membranes (mucous traps particles and bronchial cilia wave the mucous upwards so it can be coughed out); natural secretions (tears, saliva, acids); gut pH; and the natural competitive flora in the gut and urogenital tract (Roth, 1999; Thacker, 2003).

The physiological components of the non-specific immune system are complement proteins, phagocytic cells, natural killer cells, interleukins and interferon. Complement proteins and phagocytes exist mainly in the blood and require an inflammatory response to recruit them to the extracellular fluid at the site of invasion (Roth, 1999; Thacker, 2003).

Passive immunity is provided by antibodies that are transferred in colostrum to the piglet and are specific to antigens encountered by the sow (Brandrick and Molitor, 2005). Piglets are born without antibodies (agammaglobuli nanaemic). For the first 24 hours after birth the small intestine is capable of absorbing many large protein molecules non-selectively and in doing so absorbs maternal antibodies from the colostrum to achieve levels of passive immunity, which equal or exceed that of the dam (Driesen, 1989; Brandrick and Molitor, 2005). However, passive immunity does not produce any antigenic memory and once the antibodies have been metabolised (the half-life of maternal antibodies for most species is 21 days) the individual is again susceptible to infections that it was temporarily protected against. It is therefore important that animals are exposed to potential pathogens while still protected by maternal antibodies. Maternal antibodies slowly disappear from the blood stream with most protection gone by 8-10 weeks of age.

Complement proteins

The complement system is an enzyme cascade system similar to the blood's coagulation system and is composed of at least 20 serum proteins (Roth, 1999). Once activated, the complement system triggers a cascade of reactions that destroys invading bacterial organisms by rupturing their cell membranes and upsetting their osmotic balance. The complement system is extremely important in controlling both protection and host damage. The complement system also produces components that attract phagocytes and aid opsonisation and phagocytosis. Opsonisation is the process by which plasma proteins coat the invading organisms to stimulate or attract phagocytes.

Phagocytosis

Phagocytic cells (macrophages, neutrophils and eosinophils) are responsible for engulfing, ingesting and killing invading organisms. They are aided in this task by complement proteins and antibodies. An important function of the phagocytic cells is antigen processing and their presentation to the lymphocytes. They are also essential for initiating the cell mediated immune response.

Natural killer cells

Natural killer cells are lymphoid cells that can kill virus-infected body cells without previous antigenic exposure and stimulation. The activity of these cells is increased in the presence of interferon-γ and interleukin-2 (Roth, 1999; Olin and Molitor, 2005).

Soluble antimicrobial proteins

Soluble antimicrobial proteins, which include acute phase proteins and cytokines, are important in non-specific defence against bacteria and viruses. These antimicrobial proteins promote maturation and differentiation of cells and aid innate immune function, which controls the spread of infection. However, they can also increase disease severity if over produced.

Specific immunity

Specific immunity is directed at infectious agents to which the animal has been exposed through either natural infection or vaccination. By active, we mean that the body's immune system is educated on how to produce the specific antibodies itself as opposed to the mother's passive preformed antibody. Once recovery is made from a pathogen the immune system will recognise it and immediately produce the antibodies needed to protect the individual should the organism be encountered again.

Systemic immunity

Systemic immunity protects against pathogens that manage to break through an animal's external barriers, such as the skin and mucous membranes and is stimulated through natural infection or parenteral vaccines. Systemic immunity results from B lymphocyte cells producing IgG antibodies or T lymphocytes, which destroy cells harbouring intracellular pathogens. IgG antibodies are confined to the extracellular compartment of the body and neutralise viruses and bacterial toxins as well as blocking micro-organisms from attaching to host cells and mediating their uptake by phagocytic cells. Unfortunately, systemic immunity is ineffective against pathogens located on the mucous membranes or the intracellular compartments (Ryan *et al*., 2001; Seegers, 2002).

Mucosal immunity

Most pathogens enter the body through the mucosal surfaces and mucosal immunity has evolved to prevent pathogens from invading through this route. At the mucosal surface the main class of antibody is secretory IgA, which is produced by plasma cells lying below the mucous membranes. IgA responses are initiated following antigen transport by specialised phagocytic cells called M cells. M cells overlay the mucosal lymphoid tissue in structures such as Peyers patches and nasal-associated lymphoid tissues as well as diffuse effector tissues, such as gut lamina propria and nasal mucosa. Mucosal immunity can be primed by natural exposure or vaccines designed for delivery to the mucous membranes. To date these have been live vaccines capable of colonisation (Ermak and Giannasca, 1998; Mor, 1998; Seegers, 2002; Fagarasan and Honjo, 2004; Lycke, 2004).

Lymphocytes normally recirculate from the blood through the tissues and lymph nodes where they screen for their complementary antigen before returning to the bloodstream via efferent lymphatics and then to the thoracic duct, which empties into the anterior vena cava (Herbert, 1974). The lymphatic system evolved as a means of returning large proteins back into the cardiovascular system and the immune component has piggy-backed on this system of capillaries.

When a lymphocyte contacts the surface antibody of a complimentary antigen it is stimulated to expand or divide clonally so that one cell becomes many. On repeated exposure in the form of natural infection or booster vaccination the originally stimulated clone (many of which have become circulating memory cells) are clonally expanded once again and multiplied many times to produce high levels of circulating antibodies (Thacker, 2004b).

When the immune response has waned, many of the clonally expanded cells live on as memory cells and become part of the mobile circulating pool of lymphocytes. Thus, regardless of where the original antigenic invasion occurred, this mobile pool can respond to invasion at any other site in the future. The cells within the mobile pool are long lived, which is why humans only require a booster for tetanus toxoid about once every eight years.

The mucosal and systemic systems each have their own independent pool of recirculating lymphocytes. They each have different receptors on their surface that recognise either systemic lymphoid tissue or mucosal lymphoid tissue. This makes sense, as the different antibodies would differ in their effectiveness at different sites. For example

IgG is of little value in preventing invasion through the mucosal tissue whereas IgA has the capacity to bind with the secretory component and be transported across mucous membranes and neutralise pathogens before they can establish themselves. The secretory component is a membrane protein expressed on the basolateral surface of secretory epithelial cells and is responsible for the transportation of immunoglobulin (particularly IgA) across these cells into external secretions.

Cellular basis of immune response

Activation of the immune system involves two main cell types: B lymphocyte cells (processed by bone marrow - called B cells because they were originally found in the Bursa of Fabricus in chickens) and T lymphocyte cells (processed by the thymus) both of which receive critical help from a sub-population of T cells known as helper T lymphocytes. Helper T cells organise the immune response. The B-lymphocytes in the blood stream respond to the foreign antigen by producing predominantly IgG antibodies - the major immunoglobulin class in the extracellular fluid and blood. These antibodies bind to the antigen to 'neutralise' or inactivate it and thereby prevent the pathogen from invading the systemic circulation and in the case of viruses prevent the organism from invading host cells. However, should a pathogen gain entry to the host cells the cellular response, spearheaded by cytotoxic (killer) T lymphocytes (known as CTLs), recognise the invaded host cells as being foreign and attack and destroy the cells and invading organism. Each individual T cell or B cell will only recognise and respond to its individual 'destiny antigen' (Roth, 1999; Ryan *et al*., 2001; Thacker, 2003; Olin and Molitor, 2005).

Vaccines

Systemic vaccines are composed of antigens, which are usually but not necessarily proteins that are injected into the muscle or subcutaneous tissue to elicit an immune response. Two vaccinations of a killed antigen about four weeks apart are usually required to adequately prime the immune system. In the case of live modified viral vaccines one vaccination will usually suffice. The purpose of vaccines is to induce a "primed" state and create a population of circulating memory cells within the immune system of a vaccinated subject. Following natural exposure to a pathogen, a rapid immune response is generated providing protective immunity from the pathogen and a greatly reduced disease risk. Success depends on the generation of memory T and B cells and the presence in the serum of neutralising antibody (van Dijk, 1999).

Standard vaccines vary in their type and the duration of protection they provide. Those based on killed pathogens or on antigens isolated from disease-causing agents (subunit vaccine) cannot make their way into cells. They therefore give rise primarily to a humoral response, which relies upon circulating antibody to nullify the pathogen, as they are not capable of generating killer T cells. These humoral responses are ineffective against many micro organisms that infiltrate cells once antibody levels fall below a certain threshold. The human flu vaccine is an example of a killed vaccine that stimulates circulating antibody levels, which neutralise the virus before it can enter the cells and replicate.

Vaccines are designed according to how the pathogen causes disease. For example, the vaccine for *Haemophilus parasuis* (Glasser's disease) is based on the whole organism, as it causes disease by systemic invasion of the serosal surfaces. However, with pleuropneumonia, produced by *Actinobacillus pleuropneumoniae* (APP), the damage is caused by invasion of the lung and secretion of necrotising toxins (Apx) and not just to *Actinobacillus pleuropneumoniae* itself. Therefore, for any systemic vaccine to be effective it needs to contain not only the cell antigen but also the complete toxin repertoire. This is also a good example of a vaccine that relies on good levels of circulating antibody at the time of infection, as the onset of disease is so rapid the B memory cells do not have time to become activated and animals can sometimes die within two to four of infection. The memory response of A and B cells will take 48-72 hrs before sufficient protective antibodies have been produced.

The ability of an inactivated vaccine to stimulate a protective response is enhanced when it is combined with an adjuvant. Adjuvants bind the antigen at the site of injection and also attract inflammatory cells to the vaccination site, stimulating them to release more and different cytokines and prolong the duration of the immune response. Because viruses and other intracellular pathogens have unique properties, vaccines against these infectious agents should ideally elicit both antibody and cell mediated responses. Effective vaccines stimulate the production of antibodies that destroy the pathogen before it can enter cells. In addition, they elicit cytotoxic T cells that can destroy the cells in which the pathogen resides. The mechanism by which cytotoxic T cells are generated depends on the presence of foreign antigens generated by the intracellular pathogens and excreted onto the surface of the cell. These cells are no longer recognised as self-cells and stimulate the generation of cytotoxic T cells, which then destroy the infected cells (Ryan *et al*., 2001; Brennan and Dougan, 2005). It is thought that this mechanism evolved to recognise and destroy cancer cells.

Why vaccinate?

Vaccines are one of the most effective ways to protect animals against infectious diseases. Examples include the vaccines given to gestating sows to control *E. coli* and *Haemophilus parasuis* (Glasser's disease). These vaccines provide protection to offspring by conferring passive immunity against the target pathogen. Before the advent of these vaccines producers used antibiotics and were always playing catch up with the bacteria, which were constantly developing resistance to the antibiotics. The sequence of events is slightly different with viral vaccines. Compared to bacteria, viruses have a different genetic structure and method of replication and these enable them to mutate more easily. Consequently, vaccine manufacturers are forced to change the antigenic composition of viral vaccines, such as the flu vaccine, because antigenic drift and shift alters the genetic composition of the virus and renders the current viral vaccine ineffective. Because of this antigenic drift humans become fully susceptible to the flu virus about every seven years.

Although vaccines are safe there are some possible risks, such as the possibility of growth suppression following the use of some vaccines. Despite ongoing research towards safer and more effective vaccines, adverse reactions do still occur. It is important to be aware of the risks and to comply with the manufacturer recommendations to reduce the chance of these occurring. However, the majority of these reactions are usually mild and in reality the benefits of preventing disease far outweigh the risk of vaccination side effects. It is important to remember that failing to use a vaccine is in itself not a risk-free decision.

Vaccine discovery and development

In the main, vaccine development has relied on our knowledge and understanding of the pathogenesis of the disease, portal of entry, the identification of virulence mechanisms and the effect of vaccination on the immune response (Ellis, 2001; Meinke *et al*., 2004; Scarselli *et al*., 2005).

Gene technology has provided scientists with greater research opportunities to explore options for production of safe and effective vaccines. More recently we have moved from whole cell preparations to new generation sub-unit vaccines (Brennan and Dougan, 2005). The idea that genes might serve as vaccines grew in part out of research that began almost half a century ago. During the 1950s and 1960s experiments unrelated to vaccine development showed that delivering genetic material into an animal's cells could trigger some synthesis of the encoded proteins as well as antibodies targeted against those proteins (Weiner and Kennedy, 1999).

By the early 1990s, research studies demonstrated that DNA (DNA vaccines) delivered into cells could stimulate the immune system of rodents and primates to generate B cell, cytotoxic T cell and helper T cell responses against many different pathogens. The research also showed that immune responses and disease protection could be elicited when different routes of administration were used. In addition, the responses could be enhanced by a variety of methods for facilitating DNA uptake by cells (Weiner and Kennedy, 1999).

Combination vaccines have also played a role for many years and clinical testing has shown them to be safe and effective. Although the introduction of additional antigens into combination vaccines could further reduce the number of inoculations required, their development, licensing and manufacture is complex.

Proteomics

Proteomics is the identification and analysis of all the proteins (the proteome) expressed within an organism, tissue or cell. Most studies, with the aid of two-dimensional gel electrophoresis (2D-PAGE) and mass spectrometry analysis, have identified and separated the protein components. Combining this with serological analysis of convalescent animals, scientists can identify protective novel antigens as potential vaccine candidates (Green and Baker, 2002; Meinke *et al*., 2004; Scarselli *et al*., 2005). More recently Western blotting, in combination with 2D-PAGE, has provided additional information on microbial protein expression and selection of possible vaccine antigens (Nilsson, 2002). A common problem encountered when designing a vaccine is identifying the protective antigen and proteomics can help determine what animals respond to during natural infections.

Bioinformatics and in silico modelling

Bioinformatics is a computerised data management system that enables useful information to be extracted from databases associated with the structure and function of gene and protein sequences. The next step from here is *in silico* biology, which uses computer-generated models to explore the processes that take place within a cell or organ (Droit *et al*., 2005). These combined technologies will enable scientists to build computer models that can test quickly and reliably for a range of suitable vaccine candidates (Scarselli *et al*., 2005). Two examples of products developed using *in silico* modelling to predict an interaction with a protein are - Relenza™ (Glaxo) and Tamiflu™ (Roche). These

neuraminidase inhibitors are the first in a new class of drugs developed against the influenza virus and work by neutralising the viral surface proteins that help the virus emerge from the lung cells to replicate.

Novel strategies

Other new and emerging developments related to vaccine administration include the potential use of inhaled aerosol or intranasal vaccines. Because many diseases result from inhaling pathogens, vaccines delivered to the mucosal surface could produce a strong immune response in the lungs and upper respiratory tract and provide highly effective protection against disease (Illum *et al.*, 2001; Lesinski and Westerink, 2001).

Additional needle-free approaches to vaccination include the use of skin patches similar in design to those used to prevent motion sickness, jet injectors using compressed air to propel microscopic vaccine-coated particles painlessly into the skin, and incorporating vaccines into edible plants (Mor *et al.*, 1998; Ellis, 2001; Kersten and Hirschberg, 2004). These and other approaches are still under development, but offer hope that in the future syringes and needles will become obsolete.

Vaccine Design

Each vaccine is unique in terms of its composition and formulation. These variations reflect not only the different pathogens from which the vaccines are derived, but also how the vaccines are used and the mechanisms through which their effects are mediated. Vaccine potency is improved by using adjuvants that enhance antigenicity and a delivery system that ensures the antigen and adjuvant are presented optimally to the host to stimulate an immune response (Ulmer, 2004).

While live, virulent organisms would make ideal vaccines, they obviously could not be used because they would probably cause disease. Therefore, the initial step to making a vaccine is to separate the two effects of virulence and antigenicity. This means isolating or creating an organism, or part of one, that is unable to cause complete disease, yet still retains the antigens responsible for stimulating the host's immune response. There are two basic types of vaccine in use today, inactivated and live vaccines (Ellis, 2001).

Inactivated vaccines

Inactivated vaccines vary from whole bacteria or viruses (often referred to as whole cell vaccines - WCV) or components of them (sometimes referred to as subunit or fractional vaccines). A large number of WCV have been developed from normal infectious, pathogenic organisms (field strains) that have been killed, usually with a chemical treatment such as formaldehyde. These vaccines are not infectious, but still retain the antigens responsible for inducing the host's immune response. They are therefore relatively safe to use. However, as they are composed of whole organisms they retain molecules that are not involved in evoking protective immunity and therefore are usually of lower immunogenicity. Consequently, multiple doses may be needed to induce immunity. In addition, they may have serious side effects due to the release of endotoxins. Immunogenicity may be enhanced by the incorporation of adjuvants into the vaccine formulation (Pace, 1998; Ellis, 2001; Walker, 2005).

Subcellular fractions

When protective immunity is known to be directed against only one or two proteins of an organism, it may be possible to use a purified preparation of these proteins as a vaccine (Ellis, 2001). To achieve this, the organism is grown in culture and inactivated and the antigenic parts of the virus or bacteria (such as the capsule, the flagella or part of the protein cell wall) are purified and concentrated from the culture suspension (Samuelson *et al.*, 2002). While these vaccines are safe with fewer local reactions occurring at the injection site, sometimes the proteins are weakly antigenic and this requires adjuvants that provoke a severe reaction at the injection site.

Recombinant proteins

Researchers isolate the gene or genes that code for appropriate sub-units from the genome of the infectious agent. This genetic material is placed into an expression vector, such as *E. coli* or yeast host cells, which then produce large quantities of sub-unit molecules by transcribing and translating the inserted foreign DNA. By using only purified proteins extracted from the pathogen, these vaccines are safe and cannot cause disease. While sub-unit vaccines can induce strong protection, this protection offers only short-term immunity and booster vaccinations are required to ensure continued protection (Wahren, 1996; Ellis, 2001). A good example of this type of vaccine is the CSL *E. coli* vaccine, which is composed of purified fimbrial antigens.

Toxoids

Toxoid vaccines are made by treating bacterial toxins to destroy toxicity using heat or chemicals, such as formaldehyde (Ellis, 2001). The toxin is often treated with aluminium or adsorbed onto aluminium salts. After treatment the toxin is called a 'toxoid'. The best example of this is the Tetanus toxoid vaccine.

Including a stabilised form of pleuropneumonia toxin (Apx) in a vaccine has proven to be far more efficacious than using a whole cell preparation (Spicer *et al*., 1997; Hogg, 1998). For example, protection against *Pasteurella multocida* Type D (Atrophic rhinitis) has been achieved with the inclusion of dermonecrotic toxin (Hodgson, 1999). The advantages of inactivated vaccines are that they give sufficient humoral immunity if boosters are given and there is no mutation or reversion.

The disadvantages of inactivated vaccines are that:

- Some vaccines do not raise sufficient levels of immunity.
- The response is short-lived and multiple doses (boosters) are needed to lift circulating antibody levels at the time of challenge. For instance, the incubation period for APP is 4-6 hrs whereas for tetanus it is 10-21 days. With tetanus a booster dose is only required every 5-8 years while with APP a booster dose would be needed about every 2-4 weeks (before the expected onset of disease).
- There is little or no mucosal and local (IgA) or cell-mediated immunity generated by inactivated vaccines.
- They cost more but this is relative to the benefits from prevention of the disease.

Live vaccines

Live vaccines are made from live bacteria or virus preparations that colonise the vaccine recipient but do not cause disease because they have been altered (mutated) to a non-pathogenic form. The mutation can be a natural field mutation or an attenuated laboratory strain (Medina and Guzman, 2001). Live vaccines can be used in their own right, for example fimbrial positive but enterotoxin negative *E. coli*. Alternatively, they can be used as live vector vaccines with the ability to deliver antigens from other pathogens, for example *E. coli* into which the gene for a protective swine dysentery antigen has been inserted. Mutant strains of *Salmonella typhimurium* have been shown to be suitable candidates for live vaccines in farm animals (Mitov *et al*., 1992; Garmory *et al*., 2003). The advantage of these live vaccines is that they are able to colonise cells and/or mucosal surfaces, stimulating mucosal and/or cell-mediated immunity without causing disease.

Heterologous vaccines

Heterologous vaccines comprise a closely related organism of lesser virulence, which shares many antigens with the virulent organism. The vaccine strain replicates in the host and induces an immune response that cross-reacts with antigens of the virulent organism. Both cowpox virus and vaccinia virus are closely related to variola virus (the causative agent of smallpox). Vaccination with vaccinia virus has lead to the worldwide eradication of smallpox (Ellis, 2001).

The advantages of live vaccines are that:

- Both cell mediated and mucosal immunity can be elicited, depending on the target tissue.
- They can raise immune response to several protective antigens.
- Immunity is long lived.
- They can lead to elimination of wild type viruses.
- They only require a single dose.

The disadvantages of live vaccines are that:

- There is danger of reversion to virulence (mutation is a major disadvantage).
- They can spread to non-vaccinated animals (this could also be an advantage if not all animals are vaccinated, but is an economic negative for the vaccine manufacturer).
- Organisms in the vaccine must remain viable to infect and replicate in the host.
- Vaccine preparations are very sensitive to adverse storage conditions.

DNA vaccines

DNA vaccines offer advantages over conventional vaccines because they are easily constructed, stable and they can evoke both humoral and cell-mediated immunity (Ellis, 2001; Lesinski and Westerink, 2001; Daemen *et al*., 2005). Because of this, they could replace the need for live attenuated or killed virus vaccines (Wahren, 1996).

The vaccines are usually delivered by injection or by a device known as a gene gun. Injection is commonly into muscle and this puts genes directly into specific cells and also leads to cell uptake of the vaccine in the vicinity of the inserted needle. The gene gun propels plasmids into cells near the surface of the body, typically those of the skin or mucous membranes (Wahren, 1996; Ellis, 2001; Lesinski and Westerink, 2001). Within the host cells, the foreign gene can be expressed (synthesised) from the plasmid DNA and if sufficient amounts of the foreign protein are produced they will elicit an immune response (Ellis, 2001).

The advantages of genetic vaccines are that:

- They activate both arms of the immune system.
- They are unable to cause infection.
- They are easy to construct and manufacture in large quantities.
- DNA is stable and able to resist temperature extremes.
- They are relatively inexpensive to manufacture.
- They can potentially provide immunity against several strains at once because they can be engineered to carry genes from different strains of a pathogen.

Vaccine formulation

Vaccines are made from an antigen isolated or produced from the disease-causing organism - the pathogen.

The pathogen can be:

- Weakened or attenuated by passaging repeatedly until a strain which does not cause disease is produced.
- Inactivated via heating or using formaldehyde.
- The desired antigenic part of the pathogen can be extracted.

A vaccine formulation refers to the final product (a dose of vaccine), which is produced by combining the treated pathogen (antigen) with:

- A suspension fluid to carry the vaccine into the body.
- Preservatives (Thiomersal) and stabilisers so the vaccine can be stored safely.
- An adjuvant to improve the body's immune response.

Adjuvants

Adjuvants are substances that when administered with a specific antigen, enhance a humoral and/or cellular immune response to the antigen. Pure antigens are weakly immunogenic and the addition of an adjuvant induces a mild inflammation and thus attracts and activates macrophages and dendritic cells (Petrovsky and Aguilar, 2004; Thacker, 2004a; Brennan and Dougan, 2005). Adjuvants are now routinely included in nearly all inactivated or purified antigen vaccines. An effective adjuvant formulation provides optimum presentation and uptake of the antigen, generates heightened immune recognition and reaction and determines how long immunity will last (Cox and Coulter, 1997; Ellis, 2001; Sesardic and Dobbelaer, 2004; Thacker, 2004a). While researchers use several adjuvants that elicit a strong immune response, these are not always suitable for commercial vaccines because of adverse side effects and animal welfare concerns. For example, Freund's complete adjuvant causes significant reactions at the injection site (Petrovsky and Aguilar, 2004).

Modes of action

The most functional attribute of adjuvants is their adsorption and depot effect. Adjuvants cause a slow release of the vaccine from the site of injection into the tissues, and thus provide maximum exposure to macrophages, microphages and specific T and B-lymphocytes. They also activate antigen-presenting cells to secrete cytokines that enhance the recruitment of antigen-specific T and B cells to the site of inoculation (Petrovsky and Aguilar, 2004).

Classification of adjuvants

Some of the common veterinary adjuvants (Cox and Coulter, 1997; Kovarik and Siegrist, 1998; O'Hagan *et al*., 2001; Petrovsky and Aguilar, 2004; Sesardic and Dobbelaer, 2004; Thacker, 2004a; Vajdy *et al*., 2004; Brennan and Dougan, 2005) used in vaccines are:

- Mineral salts (aluminium hydroxide, aluminium or calcium phosphate):
  - Represent the first safe and effective adjuvant widely licensed for human vaccines.
  - Slow release antigen deposit.
  - Promote a good antibody response.
- Oil emulsions (w/o - incomplete Freund's adjuvant; o/w – Emulsigen, Montanide).
- Microbial – natural and synthetic (LPS of gram negative bacteria, Muramyl dipeptide, bacterial toxins).
- Surface active agents (Saponin -Quil A, Lysolecithin, detergents).
- Particulate (Immune-stimulating complexes - ISCOM's, liposomes, virosomes, nanoparticles, microspheres).
- Complex carbohydrates (polymers of mannose and glucose).

Cytokines have the potential to be used as an adjuvant in DNA vaccines where they can be expressed on the same vector as the antigen (Petrovsky and Aguilar, 2004).

Mucosal adjuvants

As IgA is produced predominantly in mucosal tissues to protect mucosal surfaces, pathogens can still colonise mucosal surfaces because, even though high levels of circulating blood IgG antibodies are present, they are unable to cross the mucosal barrier. This means animals cannot be protected from an enteric disease with an injectable vaccine. As nearly all pathogens enter the body via mucous membranes, it is logical to assume that antigen delivery systems to the mucosa are likely to provide the best protection (Lesinski and Westerink, 2001; Jagusztyn-Krynicka *et al*., 2004; Petrovsky and Aguilar, 2004). Protection correlates well with an active mucosal immune barrier and it would appear to be the most effective way to protect against intracellular organisms that must first breach the mucosal barrier to reach the host cell (Muniappa and Duhamel, 1997; Brandtzaeg, 2003; Guedes and Gebhart, 2003). A live attenuated *Lawsonia intracellularis* vaccine (Enterisol® Ileitis, Boehringer Ingelheim) is currently being registered in Australia to control this obligate intracellular organism, which infects intestinal epithelial cells.

It is difficult, if not impossible, to stimulate mucosal immunity with parenteral vaccination. Although mucosal immunity is generated through mucosal vaccination, almost without exception the antigen has to be alive to stimulate adequate mucosal immunity to micro organisms. Therefore, to accommodate killed or inactivated mucosal vaccines, mucosal adjuvants need to be further developed to ensure adequate uptake and immunogenicity of antigens that are delivered to mucosal sites (Lycke, 2004; Petrovsky and Aguilar, 2004; Vajdy *et al*., 2004). Some of the more mucosally-active adjuvants include Muramyl dipeptide, avridine or cytokines, ADP ribosylating bacterial toxins or sub-units of these toxins including cholera toxin (CT) and heat labile toxin (LT) of *E. coli* (Bowersock and Martin, 1999). These enterotoxins have boosted immune responses to unrelated antigens when co-administered by either the oral or nasal routes. Liposomes have also been formulated with antigens in mucosal vaccines and because of their stability in acidic and bile solutions are suited for oral delivery systems (Ryan *et al*., 2001). However, we consider there is only a slight chance in the near future of the pig industry having access to adjuvants that will combine with killed micro organisms to stimulate effective mucosal immunity.

Delivery systems

Delivery systems improve vaccine stability and sustain appropriate antigen exposure to the body to maximise immune stimulation (Ellis, 2001). Polymeric microsphere (micro-encapsulation) technology has been used to achieve sustained or pulsed release of antigen in injectable, oral and intranasal vaccines. This technology stimulates a

protective immune response via the mucosal route and is related to antigen transport into M cells of the gut and mucosa-associated lymphoid tissue (Mestecky *et al.*, 1997; Ermak and Giannasca, 1998; Ryan *et al.*, 2001).

Mucoadhesive polymers offer several improvements over microspheres including a reduced cost of production and improved preservation of the antigen (Mestecky *et al.*, 1997). Chitosan is a polysaccharide comprising co-polymers and is derived by the partial deacetylation of chitin, a material found in abundance in crustacea shells (prawns, crabs etc) as well as in yeast and fungi (Illum *et al.*, 2001). Chitosan, which has the ability to open tight intracellular junctions, has been used as an immunological adjuvant and carrier for a vaccine against atrophic rhinitis (Molitor *et al.*, 2005).

Administration

Some pioneering methods to deliver vaccines include, skin application and edible plant vaccines (Chin *et al.*, 1996; Mor *et al.*, 1998; Ellis, 2001; Partidos, 2003; Kersten and Hirschberg, 2004).

Parenteral

Most vaccines are given by intramuscular (parenteral) or subcutaneous injection. Parenteral vaccination is most effective for pathogens that enter via the systemic route (wound infection) or cause a septicaemia (Seegers, 2002). Researchers are continuing to seek new approaches to reducing the number of inoculations given and eliminating the use of needles for administering vaccines.

A number of disadvantages exist with parenteral vaccination (Molitor *et al.*, 2005). These include:

- needle stick injuries;
- needle disposal;
- multiple vaccinations with a single needle resulting in disease spread;
- stressful operation and procedure for animals and operators and;
- time taken to administer.

Nasal

Antigens delivered to the nasal cavity are believed to be taken up by M cells, which are very efficient at taking up particulate antigens and microparticles (Zhou and Neutra, 2002; Vajdy *et al.*, 2004). Microparticles, such as liposomes, have been given intranasally to stimulate mucosal immunity as they are small enough to be taken up by the mucosa-associated lymphoid tissue (MALT) (Mestecky *et al.*, 1997; Nechaeva, 2002; Zhou and Neutra, 2002; Vajdy *et al.*, 2004; Koping-Hoggard *et al.*, 2005).

Oral

Oral vaccination acts mainly in the gut-associated lymphoid tissue (GALT) with antigens being taken up by the M cells overlaying Peyers patches (Venkatesan and Vyas, 2000; Minato *et al.*, 2003). Large doses of antigen are usually required for oral vaccines due to poor stimulation of GALT (Venkatesan and Vyas, 2000). Additionally, antigens delivered orally need to be protected from the harsh environment of gastric acidity and proteolytic enzymes of the gastrointestinal tract. Microencapsulation also offers some promise to overcoming these barriers (Nechaeva, 2002; Zhou and Neutra, 2002; Minato *et al.*, 2003).

Mucosal vaccination has several benefits over parenteral vaccination. The most important of these is the induction of mucosal, and to a much lesser extent, systemic immunity (Minato *et al.*, 2003; Vajdy *et al.*, 2004). While mucosal immunity occurs at both local and distant mucosal surfaces, the strongest IgA response is recorded at the mucosal surface where the vaccine is delivered (Zhou and Neutra, 2002; Hyland *et al.*, 2004).

Live vector vaccines and bacterial ghosts (not infectious) provide avenues for stimulation of mucosal immunity. Bacterial ghosts are prepared by growing the bacteria under normal conditions and then inactivating with a bacteriophage. Using this method, aerosol vaccination has been investigated experimentally to prevent APP infection (Bowersock and Martin, 1999). In addition, porcine adenoviruses have been used experimentally as a species-specific delivery vector. Recombinant porcine adenovirus expressing the gene for classical swine fever virus (CSFV) provided protection when vaccinated pigs were exposed to CSFV (Hammond *et al.,* 2003).

Combination vaccines

Sometimes vaccines for different diseases are given together in one shot. These are called combination vaccines and work in the same way as individual vaccines. Combination vaccines include those such as *E. coli*, parvovirus, erysipelas and leptospirosis. Contrary to popular belief, efficacy is not reduced with combination vaccines as the immune system is designed to cope with multiple responses at the one time. In fact, combining antigens can actually enhance the immune response (Fahy unpublished).

However, all combination vaccines should be studied to determine quality, stability, safety and efficacy. Care must be taken to maintain the correct formulation, stability and compatibility between individual antigens and other components, including preservatives and adjuvants. Combination vaccines must be comparable in safety and efficacy with vaccines containing the individual antigens separately (van Oirschot, 1999). Unauthorised mixing of vaccines may decrease vaccine efficacy if the adjuvants or delivery systems are incompatible (Thacker, 2004a).

Attributes of a good vaccine

- Ability to elicit the appropriate immune response and prevent infection or clinical disease.
- Long-term or ideally life-long protection.
- Safety. The vaccine itself should not cause disease, loss of production and local site reactions should be minimal. The vaccine should also be safe for pregnant animals and foetuses.
- Stability. The vaccine should retain immunogenicity under a range of adverse storage conditions.
- Inexpensive to manufacture.

How a vaccination program works

When animals within a herd are not vaccinated against a disease, the number of animals contracting the disease will usually be high. The number of animals affected by a disease will decrease following initiation of a vaccination program. As more of the herd becomes vaccinated, the threat of disease is reduced and the disease effectively disappears. At this point, most people start to question if vaccination is still necessary and some actually stop vaccinating. However, if the pathogen is still present in the herd and the animals are no longer being vaccinated, the disease can flare-up and start to spread. It is then realised how severe the disease is and vaccination will be resumed. As the herd immunity increases, the disease will, hopefully, disappear again.

A successful vaccination program for pigs

To ensure a successful vaccination program:

- Vaccinate all pigs in the group.
- Vaccinate after maternal antibodies have disappeared.
- Vaccinate according to manufacturers recommendations and in line with the herd health status. If the vaccination is aimed to protect piglets with colostral antibodies, the booster dose for the sow should occur about three weeks before farrowing.
- All replacement breeders should be quarantined and vaccinated (for endemic diseases) before entering the herd.

Variability between similar vaccines

Vaccine variability can occur for a host of reasons and more notably, between manufacturers and in the production of autogenous vaccines (Millman and Millman, 1970). Other contributing factors include:

- Method of antigen collection.
- The organisms used and determination of pathogenic organisms versus commensals.
- Production methods - growing conditions can impact on antigen expression and consequently antigenicity.
- Formulation of a vaccine dose - concentration of organisms, antigen and adjuvant.

In conjunction with these factors, on-farm vaccine storage and handling, vaccination timing and the epidemiologic and health status difference between herds must also be considered (Brinsmead *et al*., 2004). For example, shelf life is reduced when vaccines are stored at room temperature as opposed to 4-8° C.

Vaccination timing

Vaccination timing plays a critical role in vaccine efficacy with maternally derived antibodies reducing active immunity in some vaccination programs (Molitor *et al*., 2005). For a disease such as Glassers (caused by *Haemophilus parasuis*), vaccinating the sow results in strong passive immunity in the piglet lasting until at least eight weeks of age. This provides vital protection to young pigs during the period of critical challenge (Hogg, 1999). In the case of *Actinobacillus pleuropneumoniae* maternal antibodies also last until about eight weeks of age. However, pleuropneumonia is usually an affliction of grower pigs older than eight weeks of age. Consequently, sow vaccination is of no benefit as maternal antibodies fail to provide the necessary protection to stem the onset of disease. In fact, maternal antibodies only serve to interfere with active vaccination of weaners during the first eight weeks of life. In most cases the priming dose of vaccine should be given after maternally derived antibodies have waned. The booster shot should then be given two to three weeks before infection occurs (Figure 1).

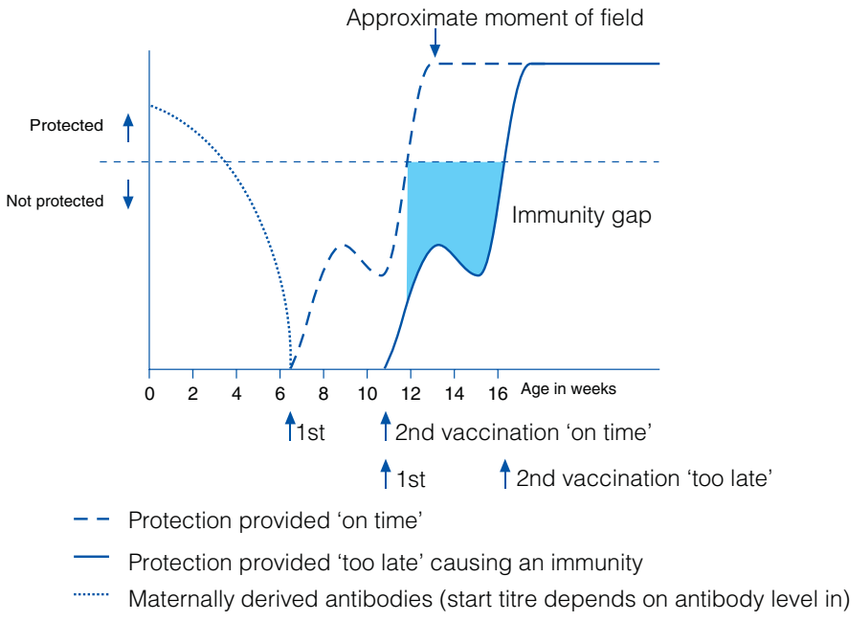


Figure 1. *Vaccination timing to prevent maternal interference and ensure protection from disease* **.**

(Modified from Strategic vaccine management (1999). Pig Progress. 6:18-21).

Herd immunity

Vaccines prevent disease in individuals and also in herds. For a disease to spread from one animal to another an infected animal must spread it and a susceptible animal must catch it. Herd immunity works by decreasing the number of susceptible animals. When the number of susceptible animals is reduced enough, the disease will disappear, as there are not enough animals to perpetuate the catch-and-infect cycle. This concept of herd immunity explains why piglets can be vaccinated at 5 and 21 days of age against Mycoplasma pneumonia (*M. hyo*) and disease incidence remains low on a herd basis. It is only gilt litters that have high levels of maternal antibodies to the *M. hyo* vaccine and these constitute about 20% of any weeks farrowing. Consequently, about 80% of the pigs will be protected by this early vaccination and because the disease does not kill animals, suboptimal performance of the 20% of animals that do not respond goes unnoticed. However for diseases such as Pleuropneumonia, which has a high mortality rate, a 20% mortality-rate would be noticed.

Some factors that influence herd immunity include:

- As vaccination rates increase, animal exposure to a disease decreases.
- Individual animals that are not vaccinated are less likely to get disease as the risk of exposure has been reduced. However, close contact and overcrowding could increase the risk of infection.
- Seasonal variation can result in increases in disease. For example, APP is more likely to occur in autumn when there are greater temperature fluctuations.
- Herd immunity does not guarantee that there will not be a disease outbreak.
- Transmission rates and infective doses are different for each disease and therefore different vaccination rates are required to produce herd immunity.

A small percentage of animals will not have an adequate immune response to a vaccine and it is important to remember that some animals may remain unprotected from a disease even though they have been vaccinated. However in a vaccinated herd the risk of infection will be minimal for these animals, as exposure to particular pathogens will be reduced greatly.

Vaccine myth

The *E. coli* vaccination scenario is a good example of how the systemic and mucosal systems work and also provides scientific proof to counteract the urban myth that: 'a booster dose of *E. coli* vaccine given to a sow at farrowing will protect against infection with haemolytic *E. coli* (HEC) O149:K88 in sucker pigs.'

Intramuscular vaccination with an *E. coli* vaccine into the neck of a sow deposits antigen and adjuvant in to the animal's tissue. The adjuvant then causes an inflammatory reaction and cytokines are released. This causes an influx of microphages and macrophages, which engulf the adjuvanted antigens and transport them to the draining lymph node in the afferent lymphatics. When the macrophages reach the draining node (which in this case is the prescapular lymph node), they home to the germinal centres of the lymph nodes where they present the antigen to its complementary recirculating lymphocyte. Germinal centres get their name from the fact that they are the sites of B-cell proliferation during an immune response and many dividing cells can be seen in germinal centres.

The cytokines cause a hyperaemia of the draining lymph node so that many more lymphocytes pass through it ensuring a maximum exposure of the antigen to the sow's repertoire of lymphocytes. Normally around about $5x10^6$ lymphocytes per hour transit a resting lymph node. At the peak of an immune response this increases to $5x10^8$ cells per hour. During the first 24 hrs following the arrival of antigen the lymph node shuts down and the efferent lymph is almost devoid of cells. This is interpreted as a mechanism to allow maximum exposure of antigen to lymphocytes.

Some 96 hrs after vaccination, proliferating lymphocytes called blast cells are seen in the lymph. Many of these produce specific antibodies. These cells are larger and paler than the normal lymphocyte as they are in the process of division and protein synthesis, hence their DNA and RNA are unravelled. If a booster dose is given some weeks later, the same process occurs again except that cells producing antibody appear in efferent lymph 48 hrs after challenge and the output of antibody is much higher than during the priming dose. Along with the cells that produce antibodies, there are also memory cells found in the efferent lymph from a responding node. These two populations of cells are in the process of migrating to all the systemic lymphoid tissue where a proportion of them will become resident memory cells that produce antibodies. Thus, an immune response initiated in one node is eventually disseminated throughout the whole sow.

Oral vaccination - mucosal associated lymphoid tissue (MALT)

Giving an oral dose of *E. coli* vaccine would stimulate a similar immune process to an intramuscular injection, however, the mesenteric lymph nodes (and not the prescapular lymph node) would be the draining nodes. Once again, the blast cells would leave the mesenteric node and be disseminated to all MALT. If the animal was pregnant during this dissemination process, blast cells would migrate to the mammary gland under the influence of progesterone and set up a population of antibody secreting cells (plasma cells). These cells would actively secrete IgA into the milk for the whole of lactation. This is known as the gut-mammary gland axis and is an evolutionary mechanism designed to protect offspring from any mucosal pathogens the mother has encountered in the environment.

The consequence of an *E. coli* booster at farrowing

While providing an *E. coli* booster at farrowing would result in high levels of circulating IgG in the dam, the IgG would not be available to the offspring because colostrum formation occurs only during the last 1-2 weeks of gestation. By day 112 of gestation colostrum has largely been formed and the cascade of hormones that initiate parturition switch off colostrum production. Thus, offspring cannot benefit from a booster dose at farrowing because the colostrum has already been formed.

Protection against *Colibacillosis*

Neonatal colibacillosis is caused by non-haemolytic *E. coli* (NHEC) and occurs during the first four days of life. For this disease, the colostrum is rich in anti-fimbrial (attachment) IgG that bathes the intestine and effectively neutralises the attachment antigens of the *E. coli*. In laboratory testing of scouring neonates the vaccine strains of *E. coli* from piglets whose dams have been vaccinated against *E. coli* are rarely isolated. However, the concentration of IgG antibodies declines (half-life of 6 hrs in the milk) so that by day four or five the levels of IgG are so low that they are no longer protective. The same situation occurs in other species and explains why scouring in calves occurs from day five onwards due to rotavirus, coronavirus, cryptsporidia and salmonella.

In pigs the onset of infection with haemolytic *E. coli* (HEC) O149:K88 occurs from five days onwards. There is no evidence that parenteral vaccination of sows at farrowing protects against this disease. In stark contrast, oral vaccination of sows with a live K88 strain at about week 11 of pregnancy is highly effective at stimulating lactogenic immunity and protecting piglets against the disease. This is an adaptation of the work carried out by Kohler (1974) who protected newborn piglets against neonatal *Colibacillosis* by oral vaccination of the dam with live *E. coli* cultures.

Protection against post-weaning *Colibacillosis* (PWCE)

This is an area of some controversy within our group as there are two opposing schools of thought. One group belives that by vaccinating sucker pigs parenterally, stimulation of circulating IgG antibodies will protect the piglets after weaning. The other group maintains that there should be sufficient circulating maternal antibody (half-life of 21 days) to protect the pigs (if IgG is indeed protective). Yet this maternal IgG fails to protect sucker or weaner pigs from HEC. This is evidenced by the fact that piglets of sows vaccinated with an *E. coli* vaccine still perish from K88 HEC from 10 days of age through to 14 days after weaning when maternal antibody is still present. In vaccine studies we have demonstrated that K88 antibodies continue to decline in sucker and weaner pigs in spite of parenteral vaccination of suckers with K88 one week before weaning and again 14 days later. Additionally, as a positive control we vaccinated them with an antigen that their dams had never been exposed to - horseradish peroxidase (HRP) - and induced a large secondary response to this antigen and none to the K88 antigen, suggesting that the maternal antibodies had neutralised the K88 antigen (Fahy, personal communication).

In our experience, oral vaccination of piglets one week before weaning, with the offending post-weaning strains, has proven highly effective in protecting against the enterotoxigenic and oedema disease strains of HEC.

Limitations to vaccine development

Manufacturer

Vaccine manufacturers are often unaware of which antigens provide best protection or how to best stimulate the immune system. However, with modern science and gene technology potential vaccine candidates can be assessed reasonably quickly and efficiently. In conjunction with the rapidly developing genomic and proteomic technologies, bioinformatics and in silico modelling will become the way of the future. In this way, manufacturers will be able to understand simultaneously the immune responses that are needed for protection and the antigens and other proteins that can generate them.

Vaccine manufacturers are also restricted through cost constraints of research, development, testing and manufacture of new vaccines (Offit, 2005). In the European Union, registration requirements include laboratory studies, extensive pharmacology and toxicology studies, clinical studies and field trials to establish conclusively product safety and efficacy. Once registration is approved, marketing surveillance and batch monitoring are required as ongoing evaluation. This particularly applies when new adjuvant and delivery systems are being used (Sesardic and Dobbelaer, 2004).

Another draw back in vaccine development relates to new technologies and intellectual property. Companies that spend a myriad of time and money into product research and development and product registration, are highly unlikely to part with any new or promising technologies to their competitors.

Regulation

In most countries, regulation includes a complete range of functions that covers the development and use of vaccines. These include safety, efficacy, toxicology, clinical trials, side effects, adverse reactions etc. In Australia efficacy is determined in pen trials and/or clinical field trials with overseas data only being accepted in support of product registration. Most product registration dossiers are prepared following the British or European Pharmacopoeia. There are also now greater requirements for GMP compliance (Milstein, 2004).

Minor use

In the past, manufacturers had no way to tailor-make their products easily and inexpensively. Autogenous vaccines are based on an antigen-strain specific vaccine for each individual herd where there is documented evidence of market failure (Tollis, 2004). While full marketing registration is not required, all aspects of production in a GMP approved facility plus limited safety and efficacy data are required before minor use permits are issued. In the near future, whole cell vaccines with improved adjuvants and delivery systems are likely to provide greater hope for bacterial therapies to prevent or minimise disease. Autogenous vaccines will, in some cases, provide the stopgap while manufacturers develop a vaccine produced conventionally. However, limited market size and hefty product development and marketing costs may prohibit manufacturers from doing so in some countries. While some vaccine manufacturers cannot justify the scheduling of small batch autogenous vaccines, there will always be a niche market for those that can.

Conclusion

Vaccines have assisted animal production industries by reducing the risk of infection in animals. This, in turn, has reduced the likelihood of disease transmission. While there are some vaccines that confer lifelong immunity on the animal, several vaccines must be given to the animal during each production cycle. The timing of vaccine administration is critical to avoid interference from maternal-colostral immunity and provide optimal protection to the animal during the time of greatest risk of infection. Not only have vaccines made a substantial contribution to the welfare of animals by reducing death, stress, pain and illness, they have also allowed animal industries to make significant cost savings by reducing the use of antibiotic medications. Additionally, reduction in disease and the improved health and welfare of the animals results in increased farm productivity and staff morale.

The future

Vaccinologists are very optimistic for the future development of vaccines. However, with few exceptions there has been little development of pig vaccines over the past 20 years. As we continue to develop technologies to identify and manufacture protective antigens and improved adjuvants and delivery systems, mucosal delivery systems offer the brightest future. In the future it will be possible to circumvent maternal antibody interference with active vaccination programs using live attenuated vaccines. Moreover, combination vaccines will be used more widely and with greater efficacy, while whole cell autogenous vaccines will continue to provide farm specific relief for some bacterial diseases.

Management for health – on-farm issues and cost benefits

E.B.M. Welch

PIC New Zealand, Christchurch, New Zealand.

Abstract

Ensuring that incoming breeding stock achieve a uniform and protective level of immunity to pathogens before mating is a major goal when managing for health within a breeding herd. To achieve this goal, practices such as isolation and acclimatisation of stock, introduction of weaner gilts and within-herd production of replacement gilts can be used. Rate of attrition of breeding animals is another indicator of the effectiveness of the operating health management system. Predisposing management factors that impact on sow losses include failure to feed sows to their nutrient requirements; inadequate water availability; inadequate gilt preparation; inappropriate temperature management and maintaining an unsuitable floor surface. The benefits of reducing sow loss can be illustrated by a cost: benefit analysis.

In the farrowing department, an important management task that affects sucker and sow health is how well piglets are managed and attended to in the first 24 hours after birth. Good management during this critical first 24 hours will help ensure: maximum colostrum intake; appropriate and effective ambient temperature for the litter and sow; that every pig has ownership of a teat; and that suitable hygiene standards are maintained. The cost effectiveness of such attention will depend on the labour cost, skill levels and the number of farrowing sows supervised by each person.

Changes in health status and production practices in the growing herd have resulted in an increasing set of challenges from re-emerging pathogenic organisms. In general, management practices such as age segregation have resulted in a lower level of disease challenge and significant improvements in health. On the other hand, age segregation of the grower herd has led to larger populations of, at times, partly infected but largely naïve pigs. Many of these can become acutely challenged by pathogens after protective levels of maternal immunity have waned. As a result, in many of these systems, disease outbreaks have been delayed and instead have appeared as more acute outbreaks in groups of pigs rather than the more chronic manifestations common in systems without age segregation. An effective weaning process compatible with the weaning age used and maintenance of a suitable environment are valuable management strategies to reduce the incidence of disease outbreaks in the grower herd. Increasing the weaning age of the piglet recoups many benefits in both post-weaning pig performance and subsequent sow fertility.

Introduction

Health outcomes in modern pig production facilities are influenced by many and varied interacting management decisions. This paper outlines some of the management factors affecting health, the relationships between these factors and the financial implications of some management approaches.

The breeding herd

Management for health in the breeding herd can be divided into two categories:

- Factors that directly or indirectly affect the health of slaughter progeny and the grower herd.
- Factors that impact on the health, survival and performance of breeding animals.

In both categories, one goal of breeding-herd management is to ensure a uniform immunity and disease status across the breeding herd. Ensuring a uniform disease status in the breeding herd:

- Minimises the likelihood of individual sows becoming infected with an agent that they were previously naïve to at a time when the effects of the disease or the immune response may have a negative effect on sow productivity.
- Minimises the likelihood of sows infecting other sows or their progeny (vertical transmission).
- Means a uniform level of passive immunity is transferred to progeny pigs. Lack of uniformity in passive immunity amongst grower pigs (or disparity in the age at which passive immunity ceases to become protective) can be a significant factor in the perpetuation of disease in the grower herd.

Strategies used to achieve uniform health status across the breeding herd include:

- Isolating and acclimatising gilts to ensure that gilts entering the herd have a solid immunity to herd pathogens by the time they come up for their first mating. This entails several different approaches depending on the disease agents being targeted. Brought-in gilts generally require a period of isolation lasting around four to six weeks followed by a period of acclimatisation when attempts are made to expose the gilts to disease agents endemic to the herd. Acclimatisation strategies include: vaccination; exposing gilts to mature herd animals; exposing gilts to organic wastes from other stages of production; and exposing gilts to air extracted from facilities bearing other pathogen-shedding pigs. While this approach has been successful for diseases like PRRS, it has not been so successful (particularly with short acclimatisation periods) with diseases such as *Mycoplasma hyopneumoniae*, which has a slower rate of transmission and a longer and more chronic disease course.
- Introducing replacement gilts at a younger age (10-14 weeks) and rearing these gilts with growing pigs in the destination herd while, if possible, using a dedicated gilt rearing diet. With some diseases, particularly those of viral origin and also respiratory diseases, the incoming naïve gilts may only serve to multiply the organism and increase infective dose to other pigs in the system. In contrast, in herds that are positive for *M. hyopneumoniae* and in which the growing animals have been vaccinated, this can be a very effective way of acclimatising the incoming gilts to the disease agent.
- Breeding replacement parent gilts in-house through closed herd multiplication.

Progeny of gilts can have a lower immune status than that of sows and vertical transmission of disease from gilts to their offspring occurs to a greater extent than with sows (Deen *et al.*, 2000; Pijoan *et al.*, 2004.) To overcome the inherent differences in health and immune status between the progeny of sows and gilts, some producers have raise gilt progeny separate from sow progeny. This system would be most appropriate in large multi-site production systems with significant health problems present in the grower herd.

There are many other areas of breeding herd management that impact on the health an immune status of animals. A significant indicator of the health status of a herd is the number of sows dropping out of the production herd. An examination of the factors affecting sow numbers can shed insight on the complex role management plays in maintaining the health of the breeding herd.

Some of the more common reasons for sow mortality, euthanasia and culling include (Irwin *et al.,* 2000; D'Allaire *et al.,* 1999):

- Locomotor problems (toe cracks, osteoarthritis, osteochondrosis, fractures, septic arthritis).
- Gastric problems (ulcers, strictured pars oesophagia, ruptured oesophagus, gastric dilatation, gastric rupture).
- Sub-optimal fertility or pregnancy failure.
- Farrowing problems (dystocia, periparturient pathology).
- Cystitis or pyelonephritis, Endometritis.
- Heart failure, heat stress.
- Gastro-intestinal accidents and torsions.
- Disease in the sow (e.g. respiratory, septicaemia).
- Milking problems.

While this list is not exhaustive, it does represent the major causes of sow mortality and attrition. All of the list items have predisposing factors in the area of management. Basic husbandry and stockmanship activities such as adequate preparation of incoming gilts and meeting the needs of animals for adequate nutrition, water, temperature, a suitable clean, dry and non-slip floor are important in the prevention of many of the above conditions. Some of these interactions are illustrated in Figure 1.

The important point in Figure 1 is that the seven basic management failures across the top predispose the herd (via a complex web of consequences) to the nine negative outcomes along the bottom. The nine negative outcomes represent most of the syndromes associated with attrition from a breeding herd. This representation is by no means exhaustive and does not assume complete cause and effect, but does illustrate how management factors can interact to predispose breeding animals to negative health outcomes. Management practices such as regular condition scoring or profiling the sow herd for back fat; continual review of feed intake and feeding practices during lactation; maintaining facilities to ensure appropriate environmental conditions and floor surfaces; and maintaining water systems to deliver adequate rates, volumes and quality of water are all valuable tools in the maintenance of a healthy, productive sow herd.

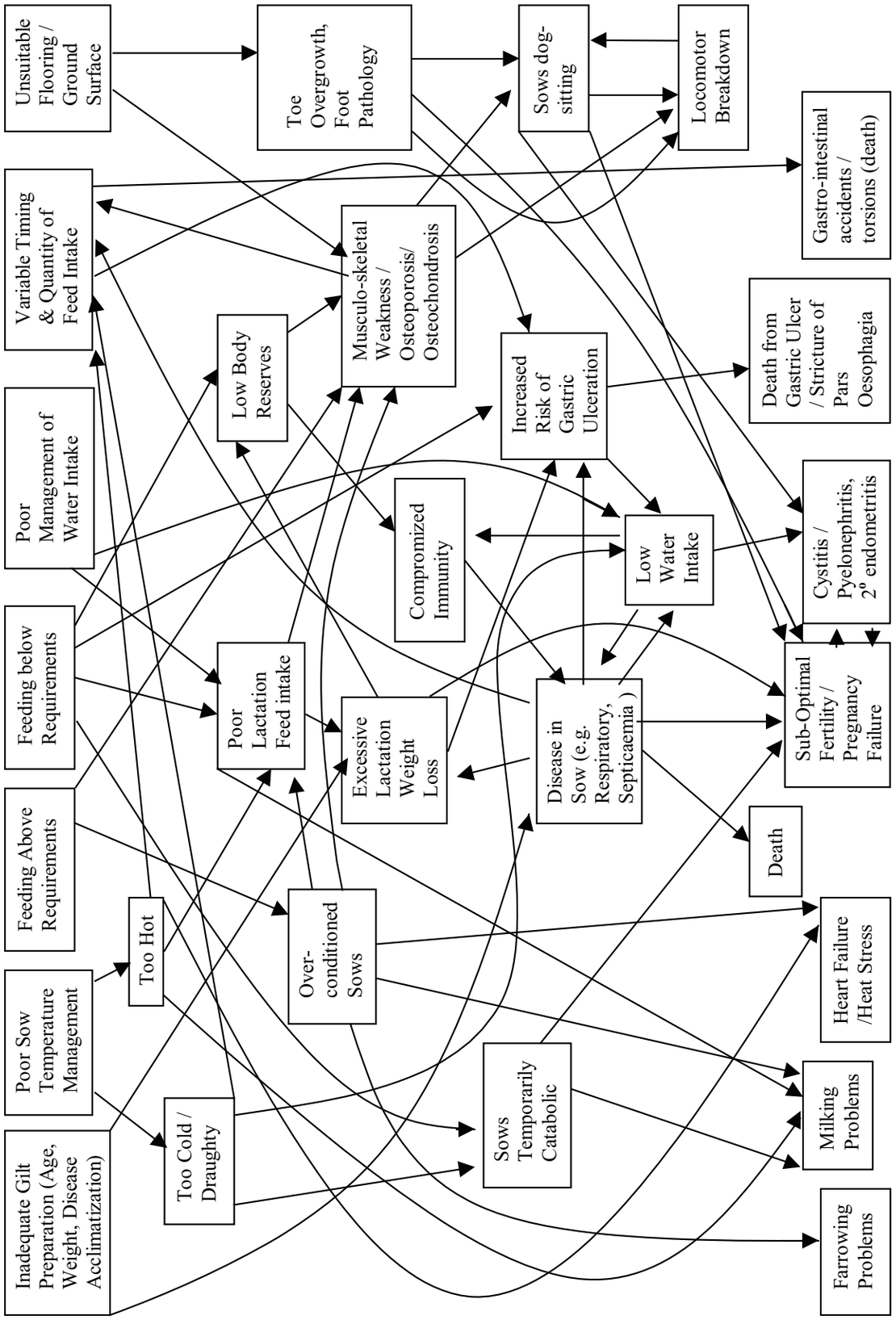


Figure 1. *Interactions between management and health outcomes in the sow.*

The influence of management on the health of a sow herd

An indoor unit in New Zealand with about 200 sows in which dry sow and farrowing space represented a production bottleneck. Over 12 months, the herd was running at a 59.4% annual replacement rate and 14% sow mortality. Mortality was due to a range of reasons including euthanasia related to lameness and locomotor problems; gastric ulceration and pathology; cystitis and pyelonephritis and; several sudden deaths of unknown aetiology.

The following changes were made:

- Condition scoring was carried out monthly and feed levels adjusted accordingly.
- Feeding equipment and feed levels were checked daily.
- Flow rates from all water nipples were checked weekly and a minimum flow rate of two litres per minute was targeted.
- Curtain opening on the building was reviewed twice each day.
- Feeding cards were instituted in the farrowing house to help staff maximise feed intake during lactation.
- The flooring surface under the sows was upgraded by replacing slats (many of which were worn and damaged) and resurfacing poor concrete surfaces, which were causing poor hoof wear and an uneven surface for sows' feet.

Eighteen months after the changes were implemented the replacement rate over a 12-month period had dropped to 39.6% and sow mortality and euthanasia to 5%. While these results are not necessarily 'cause and effect', a hypothetical cost-to-benefit ratio of these on-farm changes comparing the before (Scenario 1 – Table 1) and after (Scenario 2 – Table 2) scenarios is carried out below. Assumptions in the cost-to-benefit calculation included:

- To isolate the cost of the attrition rate, sow productivity changes over the trial period were not considered. Number of pigs weaned per sow per parity and litters per sow per year per parity were standardised across the comparison and were derived from a three-year average for these figures from seven herds with similar production systems with a total of about 2700 sows.
- The stage of the cycle when attrition occurred was not available for this farm so the timing quoted by Chagnon *et al.* (1991) was used in which 42.1% of mortality was peri-partum; 16.5% during lactation; 8% post-weaning and 35.4% during gestation.
- As dry sow and lactating sow space was a production bottleneck (restricted) all gestation deaths were assumed to result in a directly proportional drop in number of pigs weaned. Peri-partum deaths were assumed to result in the loss of 33% of pigs in these litters, lactation deaths were assumed to result in a 10% loss of pigs in these litters while post-weaning deaths had no effect on pigs weaned.
- Higher levels of sow attrition were assumed to be associated with reduced feed consumption.
- Feed intake was assumed to increase with parity and the amount of feed consumed by gilts between arriving in the breeding herd and service was considered.
- Fixed costs, facility costs, power costs and costs not varying between the two scenarios were not considered in the calculation.
- Labour input to Parity 1 females was assumed to be 30% above that for other parities due to additional time spent in their preparation, exposure to boars, acclimatisation and insemination.
- A purchase price of $400 for parent gilts and a cull sow price of $1.20 per kg dressed were used.
- Costs of producing a weaned pig were compared. Financial effects of the grower herd were not directly considered. The sale price of weaner pigs to the grower herd was assumed to be $67 to capture some of the grower herd profit in the calculation.

Table 1. Scenario 1 (before management changes).

	Parity 1	Parity 2	Parity 3	Parity 4	Parity 5	Parity 6
Dry sow feed (kg per day)	2.29	2.36	2.43	2.49	2.54	2.58
Gilt feed entry to service (kg per gilt)	189					
Dry sow feed cost ($ per tonne)	352.00	352.00	352.00	352.00	352.00	352.00
Lactating sow feed (kg per day)	4.90	5.40	5.80	5.90	6.00	6.00
Lactating sow feed cost ($ per tonne)	410.00	410.00	410.00	410.00	410.00	410.00
Variable costs ($ per sow per year)						
Feed cost	525.65	480.08	506.76	516.91	526.24	529.51
Labour	325.00	250.00	250.00	250.00	250.00	250.00
Veterinary costs	9.35	5.13	5.13	5.13	5.13	5.13
Semen	67.32	67.90	68.20	68.20	67.61	67.90
Total variable costs $ per sow	927.32	803.11	830.08	840.23	848.97	852.54
Pigs weaned per litter	9.20	9.61	9.94	9.96	9.66	9.07
Litters per mated sow per year	2.30	2.32	2.33	2.33	2.31	2.32
Parity distribution (%)	25.50	20.91	17.15	14.06	11.53	10.86
Gestation deaths % drop in pigs weaned	4.99	4.99	4.99	4.99	4.99	4.99
Peri-partum deaths % drop pigs weaned	1.95	1.95	1.95	1.95	1.95	1.95
Lactation deaths % drop in pigs weaned	0.23	0.23	0.23	0.23	0.23	0.23
Pigs weaned per sow per year	19.65	20.71	21.51	21.55	20.72	19.54
Variable costs per pig weaned ($)	47.19	38.79	38.59	38.99	40.97	43.63
Annual gilt cost less cull sow recovery after sow mortality ($/sow/year)	163.77					
Costs per pig all parities ($)	49.67					
Pigs weaned per sow per year all parities	20.57					
Total income per year ($)	275635.83					
Total variable costs per year ($)	204332.17					

Table 2. Scenario 2 (after management changes).

	Parity 1	Parity 2	Parity 3	Parity 4	Parity 5	Parity 6
Dry sow feed (kg per day)	2.32	2.39	2.46	2.52	2.57	2.61
Gilt feed entry to service (kg per gilt)	189					
Dry sow feed cost ($ per tonne)	352.00	352.00	352.00	352.00	352.00	352.00
Lactating sow feed (kg per day)	4.90	5.40	5.80	5.90	6.00	6.00
Lactating sow feed cost ($ per tonne)	410.00	410.00	410.00	410.00	410.00	410.00
Variable costs ($/sow/year)						
Feed cost	528.19	482.69	509.45	519.66	529.05	532.36
Labour	325.00	250.00	250.00	250.00	250.00	250.00
Veterinary costs	9.35	5.13	5.13	5.13	5.13	5.13
Semen	67.32	67.90	68.20	68.20	67.61	67.90
Total variable costs ($ per sow)	929.86	805.73	832.77	842.99	851.79	855.40
Pigs weaned per litter	9.20	9.61	9.94	9.96	9.66	9.07
Litters per mated sow per year	2.30	2.32	2.33	2.33	2.31	2.32
Parity distribution (%)	17.00	16.32	15.50	14.57	13.54	23.05
Gestation deaths (% drop in pigs weaned)	1.77	1.77	1.77	1.77	1.77	1.77
Peri-partum deaths (% drop pigs weaned)	0.69	0.69	0.69	0.69	0.69	0.69
Lactation deaths (% drop in pigs weaned)	0.08	0.08	0.08	0.08	0.08	0.08
Pigs weaned per sow per year	20.62	21.73	22.57	22.62	21.75	20.51
Variable costs per pig weaned ($)	45.09	37.08	36.90	37.27	39.17	41.71
Annual gilt cost less cull sow recovery after sow mortality ($/sow/year)	100.07					
Costs per pig all parities ($)	44.44					
Pigs weaned per sow per year all parities	21.52					
Total income per year ($)	288379.33					
Total variable costs per year ($)	191295.84					

Table 3. Cost of management changes.

Costs associated with management changes	Annual cost $
Monthly condition scoring labour cost ($)	360.00
Daily feed inspection and management labour cost ($)	1368.75
Water nipple checks / maintenance labour cost ($)	390.00
Twice daily curtain adjustment labour cost ($)	684.28
Interest (8 % per year) + depreciation on flooring surface upgrade ($)	4222.20
Total cost ($ per year)	7025.33
Profit difference between Scenario 1 and Scenario 2 considering weaned pig sale value and production cost:	$ 25769.84
Cost:Benefit ratio	1 to 3.67

The suckling pig

The first 24 hours of a pig's life represent a simple but significant management period during which various factors impact on the pig's long-term viability. Some of the more significant management issues during this period include:

- Farrowing supervision to assist in cases of dystocia and to minimise the stillbirth rate.
- Colostrum management for piglets including assisted suckling, colostrum sharing (split-suckling) and milking and administering colostrum from sows to disadvantaged pigs via stomach tube. Colostrum intake is positively correlated with pre-weaning survival and growth rate to 30 kg (Nielsen *et al.*, 2004.).
- Temperature management of the farrowing house environment and of the lying area of suckling pigs, and ensuring that the newly born pig can dry off after farrowing without being unduly chilled.
- Farrowing facility design, maintenance and operation.
- Fostering practices aimed at ensuring that every piglet has ownership of a functional teat within the first 24 hours of its life. These include fostering to even up number and size of pigs on each sow and shunt fostering when necessary to create additional available teats.
- Maintaining cleaning and hygiene practices between farrowing sows has a positive effect on the pre-weaning mortality, health and performance of suckling pigs (Bowman *et al.,* 1996). Ideally farrowing accommodation should be operated on an 'all-in all-out' (AIAO) basis.

Within modern farrowing facilities many of these tasks can be carried out over a short period of time with the assistance of practices such as batch farrowing and induction of farrowing - which increases the likelihood of sows farrowing during working hours.

The level of attention required to maximise piglet health and survival may be too great and some producers may question the economics of devoting labour and other resources to this period. However, it is possible to quantify the cost-to-benefit ratio of additional labour during the farrowing period and the first 24 hours following birth to maximise piglet survival.

The calculation assumes that:

- No additional costs are generated in rearing pigs that would otherwise die without management interventions from birth to weaning.
- Each pig at weaning is worth $ 63.
- Management intervention and special attention is provided over a period of 48 hours for each farrowing sow.
- Only 80% of sows are successfully induced to farrow during working hours.
- Eleven pigs are born alive per litter.

Table 4. Percentage improvement required in pre-weaning mortality to cover the labour cost of additional management intervention and supervision around farrowing.

Number of farrowing sows supervised by one person	Gross labour cost		
	$12 / hour	$18 / hour	$24 / hour
10	2.8%	4.2%	5.5%
20	1.4%	2.1%	2.8%
40	0.7%	1.0%	1.4%
60	0.5%	0.7%	0.9%

There is an interaction between labour cost per hour and the number of sows each person supervises (Table 4). This varies the required improvement in mortality to cover costs. For example, if the labour cost is $18 per hour (all inclusive) and each person supervises 20 farrowing sows with litters then they need to improve pre-weaning mortality by 2.1% of all pigs born to cover costs. Any improvement in pre-weaning mortality above this generates additional profit.

The growing herd – a constant challenge

The performance of growing pigs and the economics of production are affected profoundly by the presence of disease and the state of the immune system. Williams *et al.* (1997) placed pigs from the same source into two environments. Exposure to pathogens was encouraged in one environment and prevented or minimised in the other. The aim was to evaluate the performance of grower pigs in situations of high and low levels of immune activation. The researchers determined that between 6 and 112 kg immune activation had a significant impact on growth rate (GR) and feed conversion ratio (FCR.) Pigs exposed to low levels of pathogens (low immune activation) had a GR of 864 g/day and an FCR of 2.55. Those exposed to high levels of pathogens (high immune activation) had a GR of 720 g/day and an FCR of 2.91. With a feed cost of $300 per tonne, the difference generated in FCR alone would be worth about $11.45 per pig. With feed at $400 /tonne the difference would be $15.26 per pig.

Batista *et al.* (2002) compared two identical production systems with the same genetics but differing health status and reported a FCR of 2.54:1 for stock with a high health status compared to 2.8:1 for pigs with a low health status. Growth rates also varied considerably with the group with a high health status taking 145 days to reach 100 kg liveweight (672 g/day from birth) and the group with a low health status taking 180 days to reach 100 kg (555 g/day.) If average feed costs were $300 per tonne the FCR difference alone would be worth $7.80 per pig or $10.40 per pig with feed at $400 per tonne. The cost of poorer growth rates will be additional and will depend on the payment schedule, facility costs and whether or not grower space becomes a limiting factor and results in an inability to maximise slaughter weight of pigs within the payment matrix.

This and other evidence creates a compelling argument for removing disease as a depressor of performance from production systems. The number and proportion of commercial herds free of disease agents such as *Mycoplasma pneumonia*, pleuropneumonia, swine dysentery, mange, internal parasites and progressive atrophic rhinitis is on the rise, and this is delivering significant benefits in production performance. However, with the move away from systems based on a system of continuous flow towards practices based on age segregation, several new and significant health challenges have emerged. Some of these 'new' problem diseases include the 'Suiside diseases' (*Streptococcus suis, Actinobacillus suis, Haemophilus parasuis*), Porcine Proliferative Enteropathies and Erysipelas. The latter two diseases are difficult or impossible to keep out of a pig herd. Diseases such as *Streptococcus suis* and *Haemophilus parasuis* (Glasser's Disease) can be kept out of a closed herd or production pyramid of herds although there is still a risk they can be introduced via several avenues. Creating source herds from isolated herds and production pyramids could be one way to maintain pigs free of these diseases. However, pigs that are free of these diseases can create challenges when they are introduced into diseased herds.

Continuous flow versus 'all-in-all-out'

As production systems have become larger and more specialised there has been an increasing move away from systems based on continuous flow production to a system of AIAO production. An AIAO system can operate by room, by building or by site depending on the scale of the operation. In an AIAO production system the disease cycle is broken in the facility when it is de-populated of pigs and cleaned. Thus, when new pigs are put into a facility the disease does not affect the pigs because it has been eliminated. This principle of breaking the disease cycle in the grower phase has been largely successful in reducing immune system activation and thus increasing productivity and has for the most part been a positive step towards reducing costs of production.

However, the change from continuous flow to AIAO production has not been without its problems. The principles of AIAO and breaking the disease cycle are only effective if no additional disease is introduced with incoming pigs, such as vertical transmission from the dams of weaned pigs. In an AIAO system diseases that take a significant time to establish in a population, such as *Mycoplasma hyopneumoniae*, will potentially affect growing pigs at an older age but with a more severe and acute disease syndrome. This is in part due to the absence of passive immunity (maternal immunity received in colostrum), which wanes in older pigs. This contrasts to the situation present in a continuous flow system in which maternal immunity wanes gradually in the face of significant disease challenge at a younger age.

Streptococcus suis and *Haemophilus parasuis* (Glasser's Disease) have tended to be more problematic in AIAO systems than in continuous flow systems. This could be because weaned pigs in continuous flow systems are exposed to a higher infective dose of the disease sooner after weaning and at a time when passive (maternal) immunity is still at a protective or partially protective level. By contrast, in AIAO systems, the newly weaned group may constitute a group partly colonised by pathogens and with waning maternal immunity. The resultant infectious challenge consequently occurs to most of the animals at an older age when maternal immunity has waned.

A negative aspect of multi-site production has been the management and transport costs associated with moving pigs between sites and the inability to achieve efficient use of space at the end of the growing period. At this time a close out date for the whole group has to be set and faster growing individuals are sold earlier to avoid them going overweight.

Batch farrowing and age segregation

Smaller production units can achieve higher numbers of pigs weaned over longer intervals by farrowing sows in batches two, three, four and five weeks apart. This system can often achieve AIAO by room or building where this would not have been possible with weekly farrowing. In this way smaller units can capitalise on the benefits of age-segregated rearing and AIAO.

Weaning age

Over the past two decades there has been a progressive lowering of weaning age in various parts of the world, mainly driven by the desire to increase the output of weaned pigs by increasing the number of litters per sow per year and to decrease the amount of expensive farrowing accommodation required in new units.

There has also been a move in some parts of the world, particularly North America, to wean pigs while the majority still have protective levels of maternal immunity and to move these pigs to an age-segregated facility off site. These systems have mostly been referred to as segregated early weaning (SEW) systems but other names, such as Isowean, have also been used. This system has been used on a large scale in North America but not adopted as widely in Australia and New Zealand. The weaning age used has been low enough to prevent vertical transmission of diseases such as *Mycoplasma hyopneumoniae, Actinobacillus pleuropneumonia* and some viral diseases not currently present in Australia and New Zealand. The weaning age used has generally been below 19 days of age but has varied with the pathogen(s) for which prevention of vertical transmission is being attempted. The system is less effective when individual pigs receive inadequate colostrum or are weaned too old or are from dams that have poor immunity. These pigs can subsequently become infected at or before weaning with organisms that then infect the rest of the weaned pig population, which by then is a largely naïve and vulnerable population.

In Europe the trend has been to wean later (between 24 and 30 days), partly because of pressure from welfare laws and codes. These units often produce very good levels of productivity per sow.

Weaning at younger ages means pigs require improved diets and environments. If these requirements are not met pigs often become diseased following weaning, acquiring diseases such as post-weaning colibacillosis, streptococcal meningitis and Glasser's Disease. The profitability of weaning at younger ages has recently been brought into question with some data showing that performance of pigs following weaning is improved as weaning age is increased. Main *et al.* (2003) showed that progressively increasing the weaning age from 12 to 21 days resulted in an improved growth rate, survival, output and overall profitability of pigs following weaning. Himmelberg *et al.* (2004) showed that increasing lactation length between 15 and 21 days resulted in an increase in subsequent litter size of about 0.1 pigs born alive for every day of lactation. The author's experience would suggest that this relationship continues between 21 and 28 days lactation length. This relationship is likely to at least maintain, if not increase, pigs weaned per sow per year as lactation length increases, with added benefits in the ability of weaned pigs to adapt and perform following weaning.

A successful weaning process

A successful weaning process is characterised by a rapid and complete regeneration of intestinal villus structure, continued growth following weaning and no loss of protein and fat tissue in the post-weaning period. Close *et al.* (2003) quoted the work of Whittemore (1981) that demonstrated that when growth rates during the first seven days after weaning fall below an average of 200 g per day then the weaned pig will lose fat content. Whittemore (1981) proposed that this as a minimum growth target for a group of newly weaned pigs.

When group averages for growth rate in the first week after weaning are below expectations this is usually because there are several individuals in the group that have lost weight or gained very little. These will have lost fat reserves and are likely to be more susceptible to diseases such as post-weaning diarrhoea, Streptococcal meningitis and Glasser's disease.

Some management aspects that are important in achieving adequate growth rates following weaning include:

- Provision of an adequate and uniform temperature to the newly weaned pig.
- Provision of an adequate water supply in the form of either one drinking bowl per 15 pigs or one drinking nipple flowing at over 0.5 litres/minute per 10 pigs.
- Provision of adequate feeder space to accommodate the group feeding behaviour in the newly-weaned pig. Carr *et al.* (1998) suggested 7.5 cm per weaned pig in the first week after weaning followed by 3-4cm thereafter.

Air space and air quality

Air quality has a significant impact on the health and performance of growing pigs. Black *et al.* (2001) described a direct linear relationship between growth rate and the viable airborne bacteria count. In addition, ammonia levels from pig effluent have a negative effect on growth rate and the defence mechanisms of the pig's lung against pathogens.

Deep litter production systems

For the most part, finishing systems based on deep litter have resulted in improved respiratory and enteric health of pigs. There is, however, an interaction between stocking density, litter use and period of time spent in the pen. When pigs are stocked too heavily or kept too long in the same pen in which not enough litter is added then the dung and urine content of the deep litter encroaches on the lying area of the pigs. This causes pigs to lie on a moist surface, which indirectly impacts on their lower critical temperature and makes them more prone to chilling and other stress. This can lead to health problems such as outbreaks of respiratory and septicaemic disease.

Conclusion

As pig production systems have evolved, intensified and grown in size it has become clear that management practices and policies play a significant role in maintaining pig health. Many management practices have complex relationships with health outcomes and also complex interactions with each other. The key to a profitable and healthy production system is an understanding all of these relationships. Success of future systems will depend on what we learn from the shortcomings of past and present pig production systems.

Symposium- health strategies for the modern pork industry conclusion

P.M. Mitchell

"Broad Acres", Prairie, Vic. 3572.

Modern pork production is moving away from the use of antibiotics for the sole control of pathogens. For many this may be a daunting step as in the past our reliance on antibiotics for human and food animal treatments has been significant. Fleming's discovery of penicillin was a substantial breakthrough for researchers searching for tools that could be used in the battle against human and animal pathogens. But less than two decades after the discovery of penicillin, widespread resistance had already been reported for a number of bacteria. Moreover, unlike vaccines, antibiotics do not confer any measure of immunity upon the animal. Indeed, antibiotics tend to shield the immune system from the bacteria; such that the animal's natural and sophisticated defences are not engaged to protect the animal. Additionally, antibiotic treatment has become less effective for a number of disease complexes and modern animal production industries are under increasing pressure from consumer groups to reduce the use of antibiotics in food animal production.

We therefore must look for alternative tools to maintain the health status of our herds. Our other challenge is to anticipate what future health problems may impact on our herds. The purpose of this symposium is to:

- gain some insight into to the possible health challenges the pig industry may face in the future;
- acquire some understanding about the complexities of the porcine immune system and use this to determine how vaccines and vaccination technology can assist the pork industry;
- examine production management protocols that can be used to maintain or improve the health status and productivity of a herd.

As Morris (2005) points out, diseases are a dynamic ecological and evolutionary process. Evolving diseases and traditional pathogens will continue to manifest themselves in unexpected ways. Traditionally, the occurrence of disease has been examined under the simplistic agent-host-environment concept. But as disease is a dynamic occurrence rather than a snap-shot in time, this view is rather limited. As Morris correctly states, disease must be examined by taking into account interactions of space, time and risk factors on the host, the pathogen and the environment. These relationships must further be examined to explain how slight changes could cause diseases to occur. These associations must be examined at the animal level and also at the global, ecosystem and herd level. Novel diagnostic methods, vaccines and risk management strategies will become increasingly important tools in our attempts to maintain the health of our herds.

In the pig's battle against pathogens, resistance to infection is provided by both innate/natural immunity and specific/acquired immunity (Corbeil, 1991; Roth, 1992). Vaccines make use of the pig's natural immunological defence system by priming it and then providing the body with a record of a specific pathogen before the pathogens are able to colonise the pig. Vaccines vary greatly in composition and formulation but they are used to target the pathogens and also the pathogen's mode of attack and virulence factors (Driesen *et al.*, 2005). There are limitations to vaccine development that are brought about by lack of current knowledge regarding antigenic protection and stimulation of the immune system. In addition more, as explained by Driesen (2005), manufacture of new vaccines can be limited by the cost constraints that apply to research and development and safety and efficacy testing of a new product. Moreover, a company that has spent a significant portion of its research budget on developing and marketing a novel vaccine will protect it aggressively from competitors, with stringent intellectual property rulings. Nevertheless, vaccines have assisted the pork industry by greatly reducing the risk of infection and disease transmission. They also make substantial contributions to the welfare of animals by reducing morbidities and mortalities, which not only results in cost savings and improved herd productivity but also lifts staff morale.

New approaches to managing the challenges associated with maintaining the health status and productivity of pig are also reviewed in this symposium. Several strategies were described by Welch (2005) including:

- close monitoring of the body condition, feed intake and environmental and housing conditions of sows;
- management, intervention and supervision during the farrowing process;
- conversion to all-in all-out production;

- batch farrowing and age segregation;
- ensuring a successful weaning process;
- monitoring of the environment to ensure adequate air flow and quality for intensively housed pigs;
- deep litter production systems.

These strategies can be as challenging as the health problems faced by the herd. Producers should be able to develop successful approaches either by themselves or with the assistance of their consultants and veterinarians.

Novel management and immunological tools must continually be sought to control disease and improve the welfare of pigs. Physical and quarantine barriers - the main elements of biosecurity - will become increasingly important. These barriers, in combination with current and novel vaccination technology, as well as rigorous management protocols, will help ensure the welfare and health of the animals in our care, while we reduce our reliance on antibiotics.

References

Agricultural Compounds and Veterinary Medicines Group. www.nzfsa.govt.nz/acvm

ALEXANDER, D. and BROWN, I. (2000). 'Recent zoonoses caused by influenza-A viruses.' Revue Scientifique et Technique de L Office International Des Epizooties **19**(1):197-225.

ANONYMOUS, (1999). The Vaccination series Part 6: Strategic vaccine management. *Pig Progress* **15(4)**:14-17.

Australian Pesticides and Veterinary Medicines Authority. www.apvma.gov.au

BATISTA, L. and PIJOAN, C. (2002). Production and economic advantages of high health production. *Proceedings of the 17th IPVS Congress Ames* Iowa USA p166

BOWERSOCK, T.L. and MARTIN, S. (1999). Vaccine delivery to animals. *Advanced Drug Delivery Reviews* **38(2)**:167-194.

BOWMAN, G.L., OTT, S.L. and BUSH, E.J. (1996). Management effects on preweaning mortality: a report of the NAHMS National Swine Survey. *Swine Health and Production* **4**:25-32.

BRANDRICK, M. and MOLITOR, T.W. (2005). Role of maternal immunity and colostral immunity in designing vaccine programs. *Proceedings of the Australian Association of Pig Veterinarians Program* Australian Veterinary Association Conference Gold Coast Australia pp. 7-12.

BRANDTZAEG, P. (2003). Role of mucosal immunity in influenza. *Developmental Biology* **115**:39-48.

BRENNAN, F.R. and DOUGAN, G. (2005). Non-clinical safety evaluation of novel vaccines and adjuvants: new products, new strategies. *Vaccine* **23**:3210-3222.

BRINSMEAD, R., HILL, S. and WALKER, D. (2004). Are economic evaluations of vaccines useful to decision makers? Case study of *Haemolhilus influenzae* Type-B vaccines. *The Paediatric Infectious Disease Journal* **23**(1):32-37.

BROCCHI, E. and ZHANG G. (1997). Molecular epidemiology of recent outbreaks of swine vesicular disease: Two genetically and antigenically distinct variants in Europe, 1987-94. *Epidemiology and Infection* **118**(1):51-61.

BROWN, I. (2000). The epidemiology and evolution of influenza viruses in pigs. *Veterinary Microbiology* **74**(1-2):29-46.

CARR, J. (1998). 'Garth Pig Stockmanship Standards' p. 26 (5M Enterprises Ltd. United Kingdom, Sheffield.)

CARR, J., WALTON, J.R. and DONE, S.H. (1991). Cystitis and pyelonephritis in the Sow. *Pig Veterinary Journal* **27**:122-141

CHAGNON, M., D'ALLAIRE, S. and DROLET, R.,(1991). A prospective study of sow mortality in breeding herds. *Canadian Journal of Veterinary Research* **55**:180-184

CHIN, J., SAN GIL, F., NOVAK, M., EAMENS, G., DJORDJEVIC, S., SIMECKA, J., DUNCAN, J. and MULLBACHER, A. (1996). Manipulating systemic and mucosal immune responses with skin-deliverable adjuvants. *Journal of Biotechnology* **44**:13-19.

CHUA, K. and BELLINI, W. (2000). Nipah virus: A recently emergent deadly paramyxovirus. *Science* **288**(5470): 1432-1435.

CLOSE, W., JACQUES, K. and MULLAN, B. (2003). Alltech Pig Program 09/2002. Chapter Five - The Weaner Piglet. pp.5-21. Alltech Inc., 3031 Catnip Pke, Nicholasville, Kentucky 40356, USA.

CORBEIL, L.B. (1991). Immunity to infectious diseases In 'Microbiology of Animals and Animal Products' pp. 115-140. eds J.B. Woolcock.

D'ALLAIRE, S. and DROLET, R. (1999). Culling and mortality in breeding animals. In 'Diseases of Swine 8th Edition' pp. 1003-1014, eds. B.E. Straw, S. D'Allaire, W.E. Mengeling and D.J. Taylor (Iowa State University Press, Ames, Iowa).

DAEMEN, T., de MARE, A., BUNGENER, L., de JONGE, J., HUCKRIEDE, A. and WILSCHUT, J. (2005). Virosomes for antigen and DNA delivery. *Advanced Drug Delivery Reviews* **57**:451-463.

DEAN, S. (1996). Drugs, Europe and Legislation. *The Pig Journal* **37**:31-35.

DEEN, J. (1999). Why isolate incoming stock. *International Pigletter* **19**:19-21.

DEEN, J. and DEE, S. (2000). The problem of the guilty gilt. *International Pigletter* **19**: 70-71.

DOMINGO, E. and BARANOWSKI, E. (1998). Quasispecies structure and persistence of RNA viruses. *Emerging Infectious Diseases* **4**(4):521-527.

DRIESEN, S. (1989). Intensive care systems for weak piglets: Colostrum substitute and crib system. Final Report to Pig Research Council.

DRIESEN, S.J., FAHY, V.A. and MITCHELL, P.M. (2005). In 'Manipulating Pig Production X'. Vaccines - what are the future options for health and disease control? p. 35. ed. J.E. Paterson (Australasian Pig Science Association: Werribee).

DROIT, A., POIRIER, G.G. and HUNTER, J.M. (2005). Experimental and bioinformatic approaches for interrogating protein-protein interactions to determine protein function. *Journal of Molecular Endocrinology* **34**:263-280.

ELBERS, A., and STEGEMAN, A. (1999). The classical swine fever epidemic 1997-1998 in the Netherlands: descriptive epidemiology. *Preventive Veterinary Medicine* **42**:3-4.

ELLIS, R.W. (2001). Technologies for the design, discovery, formulation and administration of vaccines. *Vaccine* **19**: 681-2687.

ERMAK, T.H. and GIANNASCA, P.J. (1998). Microparticle targeting to M-cells. *Advanced Drug Delivery Reviews* **34**:261-283.

FAGARASAN, S. and HONJO, T. (2004). Regulation of IgA synthesis at mucosal surfaces. *Current Opinion in Microbiology* **16**(3): 277-283.

FIELD, H. and YOUNG P. (2001). The natural history of Hendra and Nipah viruses. *Microbes and Infection* **3**(4):307-314.

GARMORY, H.S., LEARY, S.E., GRIFFIN, K.F., WILLIAMSON, E.D., BROWN, K.A. and TITBALL, R.W. (2003). The use of live attenuated bacteria as a delivery system for heterologous antigens. *Journal of Drug Targeting* **11**:471-479.

GREEN, B.A. and BAKER, S.M. (2002). Recent advances and novel stategies in vaccine development. *Current Opinion in Microbiology* **5(5)**:483-488.

GUAN, Y. and K. SHORTRIDGE, (2000). 'H9N2 influenza viruses possessing H5N1-like internal genomes continue to circulate in poultry in southeastern China.' *Journal of Virology* **74**(20): 9372-9380.

GUBLER, D. (1998). Resurgent vector-borne diseases as a global health problem. *Emerging Infectious Diseases* **4**(3): 442-450.

GUEDES, R.M.C. and GEBHART, C.J. (2003). Onset and duration of fecal shedding, cell-mediated and humoral immune responses in pigs after challenge with a pathogenic isolate or attenuated strain of *Lawsonia intracellularis*. *Veterinary Microbiology* **91**: 35-145.

HAGGETT, P. (2000). The Geographical Structure of Epidemics. International Journal of Epidemiology. Oxford, Oxford University Press.

HAMMOND, J.F., JANSEN, E.S., MORRISSY, C.J., HODGSON, A.L.M., and JOHNSON, M.A. (2003). Protection of pigs against 'in contact' challenge with classical swine fever following oral or subcutaneous vaccination with a recombinant porcine adenovirus. *Virus Research* **97**(2):151-157.

HERBERT, W.J. (1974). 'Veterinary Immunology', 2nd edn, (Blackwell scientific Publications Ltd, Oxford).

HIMMELBERG, L. and RATHJE, T. (2004). Gilt development and previous lactation length influence on subsequent born alive. *American Association of Swine Veterinarians 35th Annual Meeting*, Des Moines, Iowa. pp. 217-218.

HODGSON, A.L.M. (1999). In 'Manipulating Pig Production VII'. pp. 40, ed. P.D. Cranwell (Australasian Pig Science Association: Werribee).

HOGG, A. (1998). Autogenous *Actinobacillus pleuropneumoniae* bacteria containing stabilized hemosylin: a field study. *Proceedings of the American Association of Swine Practitioners 29th Annual Meeting*, pp. 103-104, Des Moines, Iowa, USA.

HOGG, A. (1999). What every practitioner should know about swine health and immunization. *Large Animal Practice* **19**(6)**:**20-22.

HYLAND, K., FOSS, D.L., JOHNSON, C.R. and MURTAUGH, M.P. (2004). Oral immunization induces local and distant mucosal immunity in swine. *Veterinary Immunology and Immunopathology* **102**(3):329-338.

ILLUM, L., JABBAL-GILL, I., HINCHCLIFFE, M., Fisher, A.N. and DAVIS, S.S. (2001). Chitosan as a novel nasal delivery system for vaccines. *Advanced Drug Delivery Reviews* **51**:81-96.

IRWIN, C.K., GEIGER, J.O., PRETZER, S., and HENRY, S (2000). Identifying causes of sow mortality. *Proceedings of the 16th International Pig Veterinary Society Congress,* pp. 290, Melbourne, Australia,

JAGUSZTYN-KRYNICKA, E.K., WYSZYNSKA, A., and RACZKO, A. (2004). New approaches to development of mucosal vaccine against enteric bacterial pathogens; preventing campylobacteriosis. *Polish Journal of Microbiology* **53**:7-15

KERSTEN, G. and HIRSCHBERG, H. (2004). Antigen delivery systems. *Expert Review of Vaccines* **3**:453-462.

KOHLER, E.M. (1974). Protection of pigs against neonatal enteric colibacillosis with colostrum and milk from orally vaccinated sows. *American Journal of Veterinary Research* **35(3)**:331-338.

KOPING-HOGGARD, M., SANCHEZ, A. and ALONSO, M.J. (2005). Nanoparticles as carriers for nasal vaccine delivery. *Expert Review of Vaccines* **4(2)**:185-196.

KOVARIK, J. and SIEGRIST, C. (1998). Optimization of vaccine responses in early life: the role of delivery systems and immunomodulators. *Immunology and Cell Biology* **76(3)**:222-236.

LADDOMADA, A. (2000). Incidence and control of CSF in wild boar in Europe. *Veterinary Microbiology* **73**:121-130.

LEDERBERG, J. (1998). Emerging infections: An evolutionary perspective. *Emerging Infectious Diseases* **4**(3):366-371.

LESINSKI, G.B. and WESTERINK, M.A.J. (2001). Novel vaccine stategies to T-independent antigens. *Journal of Microbiological Methods* **47(2)**:135-149.

LUDWIG, S. and STITZ, L. (1995). European swine virus as a possible source for the next influenza pandemic. **212**(2):555-561.

LYCKE, N. (2004). From toxin to adjuvant: the rational design of a vaccine adjuvant vector, CTA1-DD/ISCOM. *Cellular Microbiology* **6(1)**:23-32.

MAES, D.G.D., JANSSENS, G.P.J., DELPUTTE, P., LAMMERTYN, A., and DE FRUIF, A. (2004). Backfat measurements in sows from three commercial pig herds: relationship with reproductive efficiency and correlation with visual body condition scores. *Livestock Production Science* **92**:57-67

MAIN, R.G., DRITZ, S.S., TOKACH, M.D., GOODBAND, R.D. and NELSSEN, J.L. (2003). Increasing weaning age improves pig growth performance and profitability in a three-site production system. *American Association of Swine Veterinarians 34th Annual Meeting* pp. 459 Orlando, Florida.

MEDINA, E. and GUZMAN, C.A. (2001). Use of live bacterial vaccine vectors for antigen delivery: potential and limitations. *Vaccine* **19**:1573-1580.

MEINKE, A., HENICS, T. and NAGY, E. (2004). Bacterial genomes pave the way to novel vaccines. *Current Opinion in Microbiology* **7(3)**:314-320.

MENG, X. (2000). Heterogeneity of procine reproductive and respiratory syndrome virus: Implications for current vaccine efficacy and future vaccine development. *Veterinary Microbiology* **74**(4):309-329.

MESTECKY, J., MOLDOVEANU, Z., MICHALEK, S.M., MORROW, C.D., COMPANS, R.W., SCHAFER, D.P. and RUSSELL, M.W. (1997). Current options for vaccine delivery systems by mucosal routes. *Journal of Controlled Release* **48**:243-257.

MILLMAN, S. and MILLMAN, M. (1970). Bacterial autogenous vaccines: preparation and identification of micro-organisms. *Annuls of Allergy* **28(6)**:267-275.

MILSTEIN, J.B. (2004). Regulation of vaccines: strengthening the science base. *Journal of Public Health Policy* **25(2)**:173-196.

MINATO, S., IWANGA, K., KAKEMI, M., YAMASHITA, S. and OKU, N. (2003). Application of polyethyleneglycol (PEG)-modified liposomes for oral vaccine: effect of lipid dose on systemic and mucosal immunity. *Journal of Controlled Release* **89**:189-197.

MITOV, I., DENCHEV, V. and LINDE, K. (1992). Humoral and cell-mediated immunity in mice after immunization with live oral vaccines of *Salmonella typhimurium*: auxotrophic mutants with two attenuating markers. *Vaccine* **10**:61-66.

MOLITOR, T.W., MURTAUGH, M. and DEE, S. (2005). Advances in vaccines/adjuvants. *Proceedings of the Australian Association of Pig Veterinarians Program* pp. 23-27, Australian Veterinary Association Conference, Gold Coast, Australia,.

MOR, T.S., GOMEZ-LIM, M.A. and PALMER, K.E. (1998). Perspective: edible vaccines-a concept of age. *Trends in Microbiology* **6(11)**:449-453.

MORRIS, R.S. (2005). In 'Manipulating Pig Production X'. Disease control challenges for the pig industry - what will the future bring? p.23 ed. J.E. Paterson (Australasian Pig Science Association: Werribee).

MUNIAPPA, N. and DUHAMEL, G.E. (1997). Outer membrane-associated serine protease of intestinal spirochetes. *FEMS Microbiology Letters* **154(1)**:159-164.

MURPHY, F. (1998). Emerging zoonoses. *Emerging Infectious Diseases* **4**(3): 429-435.

NECHAEVA, E. (2002). Development of oral microencapsulated forms for delivering vaccines. *Expert Review of Vaccines* **1(3)**:385-397.

NEILL, J. and MEYER, R. (1995). Genetic relatedness of the caliciviruses - San-Miguel sea-lion and vesicular exanthaema of swine viruses constitute a single geotype within the caliciviridae. *Journal of Virology* **69**(7):4484-4488.

NELSEN, C. and MURTAUGH, M. (1999). Porcine reproductive and respiratory syndrome virus comparison: Divergent evolution on two continents. *Journal of Virology* **73**(1):270-280.

NIELSEN, J.P., KJAERSGAARD, H.D. and KRISTENSEN, C.S. (2004). Colostrum uptake – effect on health and daily gain until slaughter. *Proceedings of the 18th IPVS Congress* Hamburg, Germany, Vol 2 pp. 722.

NILSSON, C.L. (2002). Bacterial proteomics and vaccine development. *American Journal of Pharmacogenomics* **2(1)**:59-65.

O'HAGAN, D.T., MACKICHAN, M.L. and SINGH, M. (2001). Recent developments in adjuvants for vaccines against infectious diseases. *Biomolecular Engineering* **18**:69-85.

OFFIT, P.A. (2005). Why are pharmaceutical companies gradually abandoning vaccines. *Health Affairs* **24(3)**:622-630.

OLIN, M. and MOLITOR, T.W. (2005). γδlymphocytes to intracellular pathogens in swine. *Proceedings of the Australian Association of Pig Veterinarians Program,* Australian Veterinary Association Conference, Gold Coast, Australia, pp. 205-210.

PACE, J.L., ROSSI, H.A., ESPOSITO, V.M., FREY, S.M., TUCKER, K.D. and WALKER, R.I. (1998). Inactivated whole-cell bacterial vaccines: current status and novel strategies. *Vaccine* **16(16)**:1563-1574.

PAHL-WOSTL, C. (1995). The Dynamic Nature of Ecosystems: Chaos and Order Entwined. New York, John Wiley & Sons.

PARTIDOS, C.D. (2003). Antigens onto bare skin: a 'painless' paradigm shift in vaccine delivery. *Expert Opinion on Biological Therapy* **3(6)**:895-902.

PASTORET, P.P. and JONES, P. (2004). Veterinary vaccines for animal and public health. *Developmental Biology* **119**:15-29.

PETROVSKY, N. and AGUILAR, J.C. (2004). Vaccine adjuvants: Current sate and future trends. *Immunology and Cell Biology* **82**:488-496.

PIJOAN, C.M., TORREMORELL, M., and DEE, S.A., (2004). Pig Health and Production. In *'Proceedings of the 18th IPVS Congress* Hamburg, Germany Vol 1 pp. 1-4.

PORTER, P., BLADES, A.J., ALLEN, W.D. and CHIDLOW, J.W. (1980). Live *vs.* dead *E. coli* vaccines: Effects on immunity health and production. *Proceedings of the International Pig Veterinary Society, Copenhagen Denmark*, pp. 163.

RABSCH, W. and HARGIS, B. (2000). Competitive exclusion of Salmonella enteritidus by *Salmonella gallinarum* in poultry. *Emerging Infectious Diseases* **6**(5): 443-448.

RODRIGUEZ, L. and BUNCH, T. (2000). Re-emergence of vesicular stomatitis in the western United States is associated with distinct viral genetic lineages. *Virology* **271**(1):171-181.

ROTH, J.A. (1992). Immune System. In A.D. Leman, B.E. Straw, W.L. Mengeling, S. D'Allaire, & D.J. Taylor (Eds.), *Diseases of Swine* (pp. 21-39). Ames, Iowa USA: Iowa State University Press.

ROTH, J.A. (1999). The immune system. In 'Diseases of Swine', 8th edn, pp. 799-820, eds B.E. Straw, S. D'Allaire, W.L. Mengeling and D.J. Taylor. (Iowa State University Press, Ames, Iowa).

ROTHMAN, K. and GREENLAND, S. (1998). Chapter 2: Causation and Causal Inference. Modern Epidemiology. K. Rothman and S. Greenland. Philadelphia, Lipincott-Raven**:** 7-28.

ROWLAND, R. S. and ACKERMAN, M.T. (1999). The evolution of porcine reproductive and respiratory syndrome virus: Quasispecies and emergence of a virus subpopulation during infection of pigs with VR-2332. *Virology* **259**(2):262-266.

RYAN, E.J., DALY, L.M. and MILLS, K.H.G. (2001). Immunomodulators and delivery systems for vaccination by mucosal routes. *Trends in Biotechnology* **19(8)**:293-304.

SAMUELSON, P., GUNNERIUSSON, E., NYGREN, P. and STAHL, S. (2002). Display of proteins on bacteria. *Journal of Biotechnology* **96(2)**:129-154.

SANCHEZ-VIZCAINO, J. (1999). Diseases of Swine. pp. 1230, Ed. B. Straw. Ames, Iowa State University Press**.**

SCARSELLI, M., GIULIANI, M.M., ADU-BOBIE, J., PIZZA, M. and RAPPUOLI, R. (2005). The impact of genomics on vaccine design. *Trends in Biotechnology* **23(2)**:84-91.

SEEGERS, J.F.M.L. (2002). *Lactobacilli* as live vaccine delivery vectors: progress and prospects. *Trends in Biotechnology* **20(12)**:504-515.

SESARDIC, D. and DOBBELAER, R. (2004). European union regulatory developments for new vaccine adjuvants and delivery systems. *Vaccine* **22(19)**:2452-2456.

SHENK, T. (1993). Virus and cell: determinants of tissue trophism. Chapter 8. In 'Emerging Viruses'. ed. S. Morse. New York, Oxford University Press.

SPICER, P.M., DRIESEN, S.J., CONNAUGHTON, I.D., HOLYOAKE, P.K. and CAPLE, I.W. (1997). Response of vaccinated pigs to challenge with *Actinobacillus pleuropneumoniae*. In 'Manipulating Pig Production VII', pp. 309, ed P.D. Cranwell. (Australasian Pig Science Association: Werribee).

STARK, K. (2000). Epidemiological investigation of the influence of environmental risk factors on respiratory diseases in swine - A literature review. *Veterinary Journal* **159**(1) 37-56.

THACKER, E. (2003). Porcine Immunology - The innate immune system. Part 1: Immunology. *The Pig Journal* **52**:111-123.

THACKER, E. (2004a). What you should know about vaccine adjuvants. *Pig International* **34**:10-13.

THACKER, E. (2004b). Porcine Immunology - The innate immune system. Part 2: Lymphocyte development and maturation. *The Pig Journal* **53**:75-91.

TOLLIS, M. (2004). Current concepts and future approaches to the development of autologous/autogenous vaccines for veterinary use. *Developmental Biology* **117**:55-60.

ULMER, J.B. (2004). Enhancement of vaccine potency through improved delivery. *Expert Opinion on Biological Therapy* **4(7)**: 045-1051.

VAJDY, M., SRIVASTAVA, I., Polo, J., DONNELLY, J., O'HAGAN, D. and SINGH, M. (2004). Mucosal adjuvants and delivery systems for protein-, DNA- and RNA-based vaccines. *Immunology and Cell Biology* **82(6)**:617-627.

VAN DER POEL and VERSCHOOR, W.F. (2001). Hepatitis E virus sequences in swine related to sequences in humans, the Netherlands. *Emerging Infectious Diseases* **7**(6):970-976.

VAN DIJK, P.M. (1999). The Vaccination series Part 1. Working with the immune system to be in control, not on the defence. *Pig Progress* **15(1)**:8-11.

VAN OIRSCHOT, J.T. (1999). The Vaccination series Part 4. How to select the best vaccine. *Pig Progress* **15(4)**:14-17.

VENKATESAN, N. and VYAS, S.P. (2000). Polysaccaridecoated liposomes for oral immunization-development and characterization. *International Journal of Pharmaceutics* **203**:169-177.

WAHREN, B. (1996). Gene Vaccines. *Immunotechnology* **2**:77-83.

WALKER, R.I. (2005). Considerations for development of whole cell bacterial vaccines to prevent diarrheal diseases in children in developing countries. *Vaccine* **23(26)**:3369-3385.

WEINER, D.B. and KENNEDY, R.C. (1999). Genetic vaccines. *Scientific American* **281(1):**50-57.

WEISS, R. (2001). The Leeuwenhoek Lecture 2001 - Animal origins of human infectious disease. *Philosophical transactions of the Royal Society of London*. Series B-Biological Sciences **356**(1410): 957-977.

WELCH, E.B.M. (2005) In 'Manipulating Pig Production X'. Management for health - on farm issueds and cost benefits. p.51 ed. J.E. Paterson (Australasian Pig Science Association: Werribee).

WILLIAMS, N.H., STAHLY, T.S. and ZIMMERMAN, D.R. (1997). Effect of level of chronic immune system activation on the growth and dietary lysine needs of pigs fed from 6 to 112 kg. *Journal of Animal Science* **75**:2481-2496.

ZHANG, G. and Haydon, D. (1999). Molecular evolution of swine vesicular disease virus. *Journal of General Virology* **80**:639-651.

ZHOU, F. and NEUTRA, M.R. (2002). Antigen delivery to mucosa-associated lymphoid tissues using liposomes as a carrier. *Biosciences Reports* **22**:355-369.

Health status of weaners can be identified by analysis of their population *Escherichia. coli* virulence gene profiles

T. Chapman, D. Jordan*, and J.C. Chin

Elizabeth Macarthur Agriculture Institute, Camden, NSW 2570, *School of Veterinary Science, St Lucia, Qld. 4072.

Escherichia coli isolated from healthy and pigs with post-weaning diarrhoea (PWD) display both common and unique combinations of virulence genes (Chapman *et al*., unpublished data). In addition, specific virulence gene combinations can also be used to identify common enterotoxigenic *E. coli* (ETEC) serogroups such as O141 and O149. We have used this information as a basis to test the hypothesis that the health status of a pig can be profiled by virulence gene analysis of entire populations of *E. coli* sampled from its faeces.

Rectal swab samples were collected from 12 healthy and 12 scouring weaner pigs (5-8 weeks old). Bacteria, released by vortexing the swab in 2 ml of brain-heart infusion broth containing 20% glycerol, were plated onto hydrophobic grid membrane filters (HGMF) and incubated overnight at 37°C on MacConkey agar.

The selectively grown *E. coli* population was dispersed into 5 ml of sterile MilliQ water and DNA was then extracted using the Promega DNA purification kit (A1120). A series of 18 multiplex and uniplex PCRs was used to detect 58 virulence genes associated with pathogenic *E. coli* from nine pathotypes of both enteric and extraintestinal origin.

Of 58 virulence genes, 40 were identified in the population samples (host profiles) with 21 genes (*iha*, *fyuA*, *traT*, *yjaA*, *fimH*, *eaeA*, *iutA*, *east1, iroN*$_{E.coli}$, *iss*, STa, STb, *chuA*, *cnf1*, *hlyA*, *paa*, *papG allele II, papG allele III, ireA, ompT* and *sfaS*) shared between healthy and clinical pigs. Chi-squared analysis revealed that only 13 of the 40 virulence genes played a significant ($P<0.05$) role in distinguishing between these two groups of animals. Six of these genes were only identified in scouring weaners (LT, *aah*, aidA, *papG allele I'*, *cdt* and F18), while four of the significant virulence genes were only found in healthy weaners (*cvaC*, *bmaE*, *papC* and *ehxA*).

Chi-squared analysis of a third group of weaners, considered to be subclinical because of previous contact with diseased animals, revealed an additional six virulence genes (*fyuA*, *fimH*, *iha*, *iss*, *yjaA* and *hlyA*) to the original 13 that could be used to differentiate between all three groups ($P<0.05$).

These results confirm that virulence gene profiling of *E. coli* populations from each animal can be used to cluster pigs into groupings that correlate with their enteric health status with respect to colibacillosis. Population microbial analysis or PoMA for virulence genes and also antibiotic resistance genes can provide invaluable information on efficacy of intensive pig farming management practices and should be adopted routinely because they are cost effective.

Supported in part by QAF Meat Industries and International Animal Health.

Production of a pig CD34 antibody for isolation of hematopoietic stem cells

D.S. Layton*,**, A.G.D. Bean*, G.S. Stephenson*,**, T.E. O'Neil*, M.M. Broadway* and A.D.G. Strom*

* CSIRO Australian Animal Health Laboratory, Geelong, Vic. 3220. ** Melbourne University, Melbourne, Vic. 3052.

Intensive growing conditions are often associated with a higher incidence of immune system stimulation, which can lead to a reduced growth rate. With this in mind by studying the functional development of leucocytes from pig bone marrow precursors, (hematopoietic stem cells [HSC]), we can further develop strategies directed toward enhancing the pig immune system. To date, the most effective method of HSC isolation has been through the use of a CD34 monoclonal antibody (mAb). However, as no such reagent is currently available in the pig (Hienz *et al.*, 2002) the aim of this project was to develop antibodies against porcine CD34.

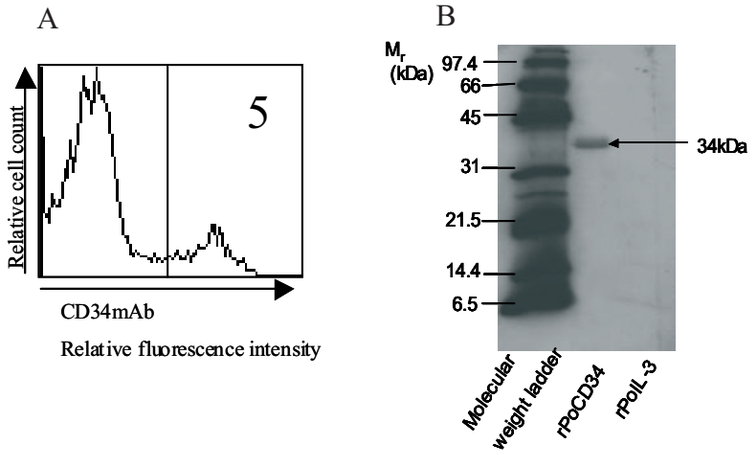


Figure 1. *(A) FACS analysis of porcine BM with CD34mAb (n=5) (B) Western blot analysis of CD34mAb on rPoCD34 and rPoIL-3 (negative control) with molecular weight markers.*

The extracellular region of the porcine CD34 gene was cloned and inserted into prokaryotic and eukaryotic expression vectors. The prokaryotic expression vector was used to produce a recombinant porcine CD34 protein (rPoCD34) in E. *coli*. Mice, chickens and rabbits were inoculated with the rPoCD34 and the eukaryotic expression vector as a DNA inoculation. Following DNA inoculation, a boost with either the recombinant protein or porcine bone marrow cells was administered. Sera was collected and tested for reactivity against porcine bone marrow. Spleens were taken from mice and hybridomas generated using Sp2 myeloma cells. Fluorescent Activated Cell Scanning (FACS) analysis was used to identify positive clones and polyclonal antibodies.

A positive hybridoma was identified that stained about 5% of the porcine bone marrow cells (Figure 1B) as well as a population of CD90+ cells, which correlated with previous observations in human and mouse CD34 studies. This mAb also bound the 34kDa rPoCD34 on western blot (Figure 1A), indicating that the antibody was against CD34. HSC were isolated using this mAb and showed stem cell enrichment in a methylcellulose assay, as seen with studies of human CD34+ cells (Verfaillie, *et al.*, 1990).

The pig CD34 antibody has enabled the detection and isolation of pig HSC, a step that will allow studies of hematopoiesis and potentially could identify ways to enhance the pig immune system and thus pig production.

References:

HEINZ, M., HUANG, C., EMERY, D., GIOVINO, M., LEGUERN, A., KURILLA-MAHON, B., THEODORE, P., ARN, J., SYKES, M., MULLIGAN, R., DOWN, J., SACHS, D. and GOODELLl, M. (2002). Use of CD9 expression to enrich for porcine hematopoietic progenitors. *Experimental Hematology* **30**:809-815.

VERFAILLIE, C., BLAKOLMER, K. and McGLAVE, P. (1990). Purified primitive human hematopoietic progenitor cells with long-term in vitro repopulating capacity adhere selectively to irradiated bone marrow stroma. *Journal of Experimental Medicine* **172**:509-520.

Antimicrobial resistance of *Escherichia coli* and *Enterococcus* spp. from an integrated, semi-closed population of humans and swine

R.B. Harvey*, H.M. Scott**, W.Q. Alali**, L.D. Highfield**, D. B. Lawhorn***, R.C. Anderson* and D.J. Nisbet*

*Agricultural Research Service, USDA, USA. **College of Veterinary Medicine, Texas A&M University, USA. ***College of Veterinary Medicine, Texas A&M University, USA.

Antimicrobial resistance (AMR) in human infections is generally perceived by the medical community to be a consequence of sub-therapeutic antibiotic usage in animal production (Anderson, 1999). However, since no definitive studies have confirmed this, controlled epidemiologic studies with stable human and animal populations are needed to identify the transmission dynamics of AMR (Khachatourians, 1998).

The present experiment continues a longitudinal study to examine the AMR profiles and potential AMR transmission dynamics of *Escherichia coli* (EC), *Enterococcus faecalis* (EF) and *E. faecium* in a semi-closed population of swine and humans (Scott *et al*., 2005). The study population consisted of humans (4,794 swine workers and 34,431 non-swine workers) and 51,742 swine in a multi-site, vertically integrated swine operation. There was little to moderate movement of humans into the system and little movement out of the system, whereas there was very little swine movement into the system and no movement out of the system. Composite swine faecal samples and human wastewater samples were collected each month between January and December 2004. Nine hundred and eighty seven samples of human wastewater and 931 samples of swine faeces were cultured for EC and EF.

EC were isolated by culturing onto CHROMagar-*E. coli* (DRG International, Mountainside, NJ). *M. enterococcus* agar (Becton Dickinson, Sparks, MD), with and without vancomycin (20 µg), was used to culture vancomycin-resistant *E. faecium* (VRE) and EF, respectively. Isolates were identified using API test kits (bioMerieux, Hazelwood, MO). Antibiotic sensitivity was tested using a micro-broth dilution (Sensititre®) system and Gram-negative (CMV1AG-NF) and Gram-positive (CMV1AG-PF) panels from the National Antimicrobial Resistance Monitoring System (NARMS).

Table 1. Proportion (%) of resistant microbial strains in a semi-closed population of humans and swine.

		Escherichia coli (EC)				*Enterococcus faecalis* (EF)		
		Number of antimicrobials						
		0	1	2 or more		0	1	2 or more
Human	(n = 829)	53%	26%	21%	(n = 345)	3%	9%	88%
Swine	(n = 857)	12%	37%	51%	(n = 279)	0%	1%	99%

EC were isolated from 84% of the human samples while 35% were positive for EF and 92% and 30% of swine samples were positive for EC and EF, respectively (Table 1). Swine EC and EF showed a greater multi-drug resistance than human EC and EF, which agreed with an earlier study in which swine EC displayed increased AMR compared to humans (Scott *et al*., 2005). EF from both populations was resistant to more antimicrobials than EC. During this study, 17 VRE were isolated from human wastewater from multiple geographic locations while no VRE were isolated from swine samples. These are believed to be the first non-clinical isolations of VRE in the U.S. Our preliminary results suggest that: swine EC and EF have increased AMR compared to human isolates; there was no apparent AMR transfer from swine to humans or vice versa; and VRE may be more common in the environment than previously perceived. Longitudinal studies in this population will continue until 2007 during which we will further characterise AMR by determining the prevalence of genetic factors such as integrons, plasmids and gene cassettes that encode for AMR.

References

ANDERSON, R.M. (1999). *Nature Medicine* **5**:147-149.

KHACHATOURIANS, G.G. (1998). *Canadian Medical Association Journal* **159**:1129-1136.

SCOTT, H.M., CAMPBELL, L.D. and HARVEY, R.B. (2005). *Foodborne Pathogens and Disease* **2**:24-37.

Weaning pigs without antimicrobial growth promotors – microbiological and immunological results from an integrated project

J. Kluess*, E. Sowa**, H.J. Rothkötter** and W.B. Souffrant***

*Schothorst Feed Research, The Netherlands. **Otto von Guericke University Magdeburg, Magdeburg, Germany. ***RU Nutritional Physiology, Dummerstorf, Germany.

The European-wide ban on antimicrobial growth promoters (AMGP) for weaning piglets has highlighted the need for research on gut physiology to develop alternative strategies. The resident microflora and the gastrointestinal immune system (GALT) are important entities in the porcine gut and their actions are thought to be closely connected. We hypothesised that different fibre contents in starter diets after weaning would exert a differential influence on resident microflora and GALT in the distal small intestine of piglets.

Piglets were weaned at 28 days (day 0) onto cereal-based, AMGP-free diets with low (3%, LF) or high (8 %, HF) crude fibre content. Differences in fibre content were obtained using wheat bran and sweet lupin. At -4, +1, +2, +5 and +8 days, pigs were sacrificed (eight pigs per day per diet) and digesta and tissue from the distal small intestine were taken for microbiological (digesta) and immunological (tissue) investigation. Selective agar plates (VRBD, SG, MRS, SB) were applied to determine microbial counts of enterobacteria, enterococci, lactic acid bacteria and yeast. The mRNA-expression of cytokines IL-2 and TGF-ß was measured by RT-PCR and respective PCR products were quantified densitometrically. Data were analysed by Tukey test in STATISTICA (6.0).

There were no differences in performance and no pig showed diarrhoea after weaning. The two starter diets exerted different levels of impact on microflora and cytokine expression after weaning. More significant changes were detected in the microflora of piglets fed HF than in piglets fed LF (Table 1). IL-2 and TGF-ß had a significantly higher up-regulation until +5 days, indicating a stronger immunological response to the HF diet. The more pronounced cytokine expression coincided with more changes in microflora for piglets fed HF, while the LF diet induced much weaker responses. This indicates a close relationship between both entities (Stokes, *et.al.,* 2004). The nature of this relationship (causal or not) remains to be elucidated.

Table 1. Microbiological and immunological data of piglets pre- and post-weaning (mean ± s.e.d.).

Microflora (log CFU / g)						
Parameter	-4 day	+1 day	+2 day	+5 day	+8 day	Diet
Entero-bacteria	7.2 ± 0.9[a]	8.4 ± 0.3[ab]	8.5 ± 0.6[b]	8.2 ± 0.4[ab]	7.9 ± 0.6[ab]	HF
		7.5 ± 0.3	6.8 ± 0.6	7.8 ± 0.4	8.2 ± 0.6	LF
Enterococci	6.8 ± 1.4[a]	6.6 ± 0.8[a]	6.5 ± 0.7[ab]	4.6 ± 0.9[ab]	3.6 ± 0.9[b]	HF
		6.6 ± 0.8[ab]	6.2 ± 0.7[ab]	4.5 ± 0.9[b]	4.3 ± 0.9[b]	LF
Lactic acid bacteria	8.6 ± 0.6[a]	6.9 ± 0.7[a]	8.3 ± 0.4[ab]	8.1 ± 0.4[ab]	8.7 ± 0.2b	HF
		7.6 ± 0.7	8.6 ± 0.4	8.5 ± 0.4	8.4 ± 0.2	LF
Yeast	2.0 ± 1.3	2.9 ± 0.8	2.3 ± 1.3	0.6 ± 0.4	3.1 ± 0.9	HF
		2.5 ± 0.8	3.2 ± 1.3	2.8 ± 0.4	3.1 ± 1.3	LF
Cytokines (relative units mRNA)						
	-4 day	+1 day	+2 day	+5 day	+8 day	Diet
IL-2	0.078 ± .050[a]	0.178 ± .059[a]	0.187 ± .108[a]	0.306 ± .068[b]*	0.137 ± .066[a]	HF
		0.108 ± .152	0.141 ± .154	0.120 ± .119*	0.143 ± .108	LF
TGF-ß	0.423 ± .077[a]	0.657 ± .087[b]*	0.792 ± .115[b]*	0.984 ± .121c*	0.790 ± .160[b]*	HF
		0.112 ± .052[a]*	0.078 ± .047[a]*	0.159 ± .067[a]*	0.183 ± .060[ab]*	LF

Rows [abc] and columns* (see cytokines) with unlike superscripts are significantly different (P<0.05)

This research was financially supported by the EC: project HEALTHYPIGUT (QLK5-LT2000-00522).

References

STOKES, C.R., BAILEY, M., HAVERSON, K., HARRIS, C., JONES, P., INMAN, C., PIE, S., OSWALD, I.P., WILLIAMS, B.A., AKKERMANS, A.D.L., SOWA, E., ROTHKÖTTER, H.J. and MILLER B.G. (2004). *Animal Research* **53**(4):325-343.

Antimicrobial resistance in enterotoxigenic *Escherichia coli* isolates from Australian pigs

M. Smith*, D.J. Trott*, D. Jordan**, M.D. Barton*** S.J. Driesen**** T. Chapman* and J.C. Chin*****

* The University of Queensland, St Lucia, Qld. 4072. ** NSW Department of Primary Industries, Wollangbar, NSW 2477. ***University of South Australia, Adelaide, SA 5000. ****Department of Primary Industries, Bendigo, Vic. 3554. *****Elizabeth MacArthur Agricultural Institute, Camden, NSW 2570.

Antimicrobial agents are used widely in the Australian pig industry to control enterotoxigenic *E. coli* (ETEC) and many other diseases. Antimicrobial resistance genes (ARGs) permit bacteria to escape or bypass the toxic effects of antimicrobial agents. ARGs are becoming widespread in isolates from animals and humans and are often encoded on multi-drug resistant (MDR) plasmids that can be readily transferred between pathogenic bacteria. MDR ETEC have been isolated from Australian piggeries (Stephens, 2003) and can often have a serious impact on pig production. It is not yet known which plasmid-mediated ARGs are present in these ETEC isolates and whether they represent a public health risk. We hypothesise that although Australian porcine ETEC isolates may contain ARGs on MDR plasmids, they are not of public health significance due to conservative antimicrobial registration and prudent usage.

One hundred and seventeen ETEC isolates from pigs with post-weaning diarrhoea (PWD) and 2-3 week scours that had been identified as MDR (resistant to >4 antimicrobials) were obtained from veterinary laboratories throughout Australia and tested for resistance to 14 antimicrobial agents (ceftiofur, gentamicin, apramycin, neomycin, spectinomycin, streptomycin, enrofloxacin, chloramphenicol, tetracycline, ampicillin, nitrofurantoin, sulphamethoxazole/trimethoprim, florfenicol and tylosin) as described by Stephens (2003).

Enrofloxacin and ceftiofur were the only antimicrobial agents for which resistance was not detected (Table 1). Resistance to florfenicol and chloramphenicol occurred in 5.3% and 27.2% of isolates, respectively. A variety of resistance profiles was demonstrated for the aminoglycoside antimicrobials, with gentamicin showing the least (17.4%) and streptomycin the most (77.4%) resistance. Higher proportions of isolates were resistant to sulphamethoxazole/trimethoprim (48.2%) and tetracycline (70.2%). Possible cross-resistance was observed between the aminoglycosides, gentamicin and apramycin. These resistance profiles suggest that MDR ETEC do not represent a major public health risk as a reservoir of significant ARGs. However, the range of registered antimicrobials available for treatment of PWD is diminishing rapidly, with several isolates already showing resistance to florfenicol.

Table 1. Resistance (%) of multi drug-resistant enterotoxigenic *E. coli* isolates [a].

	No	Ceft	Genta	Apra	Neo	Spect	Strep	Enr	Chlo	Tetra	Am,p	Nitro	Sul/ Tri	Flor	Tyl
Qld	52	0	12	12	12	32	41	0	10	31	14	6	19	0	52
SA	27	0	0	0	5	5	22	0	13	26	13	3	14	4	27
Vic	28	0	8	16	15	13	19	0	4	20	26	3	17	1	28
WA	8	0	0	0	1	0	6	0	3	3	3	1	5	1	8
NSW	2	0	0	0	0	0	1	0	0	0	1	1	1	0	2
Total	117	0	20	28	33	50	89	0	30	80	57	14	56	6	117
%		0.0	17.1	23.9	28.2	42.7	76.1	0.0	25.6	68.4	48.7	12.0	47.9	5.1	100.0

[a] Numbers in the table represent the number of isolates showing resistance to each antimicrobial.

Having defined the phenotypic characteristics of the ETEC collection, this work will now focus on genetic aspects. DNA primers targeting plasmid-mediated ARGs have been designed for this purpose and will be applied in multiplex polymerase chain reaction assays in unison with hydrophobic grid membrane filter technology. This approach is highly suited to detecting particular ARGs within a complex microbial community such as the gastrointestinal tract of pigs.

STEPHENS, C.P. (2003). *Commun Dis Intell* 2003 **27** Suppl: S127–S131.

Effect of Bio-Mos™ and Selplex™ on the performance of piglets

R. Huang*, W. Zhu*, S. Huang* and S. Chen**

*Nanjing Agricultural University, Nanjing, China, **Alltech Asia-Pacific Bioscience Center, Beijing, China.

Bio-Mos™ (Alltech Inc, USA) is a mannan-oligosaccharide derived from the cell wall of yeast (*Saccharomyces cerevisiae*[1026]) and has been shown to adsorb enteric pathogens and may also be involved in immuno-modulation (Davis, *et. al.*, 2004). Adequate selenium is necessary for basic processes such as growth and antioxidant defence (Edens, 2001). Sel-Plex™ (Alltech Inc, USA), a natural organic form of selenium, is believed to be superior to the chemically inorganic selenite form normally used in animal feed. The objective of the current trial was to evaluate the benefit of replacing conventional antibiotic growth promoters (AGP) with Bio-Mos™ alone and in combination with Sel-Plex™ replacing sodium selenite.

One hundred and forty four piglets (Duroc x Landrace x Large White) taken from litters of 12 sows were weaned at 21 days of age and 108 pigs (average body weight 5.94 kg) were allocated randomly into three groups, based on body weight and sex, with three replicates (12 pigs/replicate). The trial period was 26 days, including five days of pre-trial adaptation. Feed and water was available *ad libitum*. Penned groups of piglets were allocated randomly to one of three treatment diets: AGP control (1 g/t zinc bacitracin plus 0.5 g/t colistin sulphate and 0.2 ppm sodium selenite), Bio-Mos™ (1 kg/t) or Bio-Mos™ plus Sel-Plex™ (0.2 ppm replacing sodium selenite). Piglet growth, feed intake and the occurrence of diarrhoea were measured during the trial period. Incidence of diarrhoea was calculated using the following equation:

$$\text{Incidence of diarrhoea (\%)} = \frac{\text{total recorded cases of diarrhoea}}{\text{108 pigs x 21 days}} \times 100$$

Results were expressed as mean values with standard errors. Data were subjected to analysis of variance using the general linear models procedures of SAS software (SAS Institute, 1997) with pen as the experimental unit. Means were separated by the DUNCAN'S multi-comparison range test.

Table 1. Performance and incidence of diarrhoea of piglets receiving Bio-Mos™ instead of AGP or Sel-Plex™ instead of sodium selenite.

Treatment	AGP control	Bio-Mos 1 kg/t	Bio-Mos 1 kg/t + Sel-Plex 0.2 ppm
Initial body weight (kg)	5.97±0.30	5.98±0.26	5.88±0.18
Final body weight (kg)	11.36±0.88^{b}	11.74±0.74^{b}	11.97±0.64^{a}
Average daily feed intake (g)	406.35±9.29	406.68±24.37	380.12±16.71
Average daily gain (ADG) (g)	257.67±32.68^{b}	274.14±26.83^{a}	290.87±25.53^{a}
FCR	1.58±0.02^{a}	1.48±0.99^{ab}	1.31±0.09^{b}
Incidence of diarrhoea (%)	2.9	1.7	1.3

$^{a-c}$ Means with different superscripts in the same row are significantly different ($P<0.05$).

Bio-Mos™ alone significantly improved ADG and FCR ($P<0.05$) compared with piglets fed the AGP control. Piglets performed even better when Bio-Mos™ was combined with Sel-Plex™ ($P<0.05$), indicating that both products influenced the growth and feed efficiency of piglets. Diarrhoea incidence was reduced in the piglets receiving Bio-Mos, however this benefit was not enhanced greatly by addition of Sel-Plex™, indicating that the latter product had little direct effect on maintaining gut health.

In conclusion, Bio-Mos™ and Sel-Plex™ improve piglet performance and can be used to replace conventional AGPs and inorganic selenite. The incidence of diarrhoea can be reduced by supplementing piglet diets with Bio-Mos™ at 1 kg/t.

Reference

EDENS, F.W. (2001). Science and Technology in the Feed Industry, *Proceeding of the 17th Annual Symposium*. Nottingham University Press. 349-376.

DAVIS, M.E., BROWN, D.C., MAXWELL, C.Z., JOHNSON, Z.B., KEGLEY, E.B. and DVORAK, R.A. (2004). *Journal of Animal Science* **82**:581-587.

Bioavailability of different preparations of DL-tocopheryl acetate (Vitamin E) for weaning piglets

S. Jakob and P.A. Geraert

Adisseo France, S.A.S, Ets. de Commentry, rue Marcel Lingot, 03600 Commentry, France.

Vitamin E is a strong antioxidant that protects cell membranes and is an essential nutrient for swine. In addition, Vitamin E stimulates the immune system of animals and improves meat quality. Vitamin E in the form of DL-α-tocopheryl acetate is the most important vitamin used by the feed industry due to its outstanding stability, its physical and chemical properties and its efficacy. However, it is not fully bio-available, with about 60% of DL-α-tocopheryl acetate ingested by swine and poultry being excreted. The aim of this study was to determine whether a new preparation of DL-α-tocopheryl acetate would be more bio-available to piglets than standard DL-α-tocopheryl acetate.

Thirty crossbred weanling piglets (8.0 kg live weight) were divided into three groups (R1, R2, R3), kept individually and distributed according to a completely randomised block design. The piglets were fed *ad libitum* for 14 days on a corn and wheat based diet (corn 350 g/kg, wheat 150 g/kg, soybean meal 250 g/kg, skimmed milk powder 150 g/kg, sugar 50 g/kg, premix 50 g/kg, Lys 15 g/kg; ME 13.7 MJ/kg). The diet was supplemented with either 20 IU/kg (R1) or 150 IU/kg (R2) standard DL-α-tocopheryl acetate. R3 piglets were fed a diet supplemented with 150 IU/kg of a new, DL-α-tocopheryl acetate preparation (Microvit™ E Supra, Adisseo, France). On day 15 of the experiment the animals were slaughtered and their livers dissected, immediately frozen in liquid nitrogen and ground to powder. Muscle samples (*muscularis glutaeus maximus*) of about 9 cm^2 were obtained from the left hind leg and frozen immediately. The Vitamin E content of the samples was determined by HPLC and data were statistically analysed using one-way ANOVA.

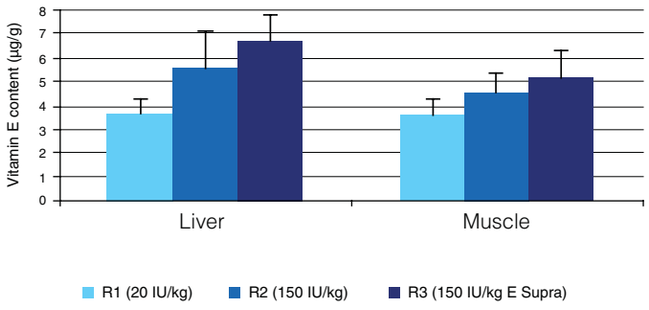


Figure 1. *Vitamin E content in liver and muscle of piglets fed diets supplemented with either standard DL-α-tocopheryl acetate or Microvit™ E Supra (mean ± SD) .*

Vitamin E levels in muscle and liver ranged from 3.6 µg/g to 6.7 µg/g (Figure 1) and were similar to Vitamin E levels reported earlier by Anderson *et al.* (1995). Vitamin E contents increased in liver (51%) and muscle (35%) as DL-α-tocopheryl acetate was increased from 20 IU/kg to 150 IU/kg in the diet ($P<0.05$). Adding Microvit™ E Supra to the diet increased Vitamin E content in the liver by 81% ($P<0.02$) and in muscle by 44% ($P<0.01$) compared to the contents in the livers and muscles of piglets fed 20 IU/kg DL-α-tocopheryl acetate. The relative comparison of the slopes of the regression curves of R1 and R2 (standard DL-α-tocopheryl acetate) and for R1 and R3 (E Supra) enabled an estimation of the biovailability of Vitamin E from the two sources. A comparison of the slopes of the curves for Vitamin E contents in livers (1.89 for standard DL-α-tocopheryl acetate and 3.00 for E supra) suggested an increase in relative bioavailability of E supra of 37%. For muscle samples, the slopes of the curves were 0.89 for standard DL-α-tocopheryl acetate and 1.61 for E Supra. As a result, an increase in relative bioavailability of E Supra of 44% was estimated. From this study we conclude that DL-α-tocopheryl acetate in the form of Microvit™ E supra is about 40% more bio-available than the current industry standard form of DL-α-tocopheryl acetate.

References

ANDERSON, L.E., MYER, R.O., BRENDEMUHL, J.H. and MCDOWELL, R.L. (1995). *Journal of Animal Science* **73:**490-494.

3 Nutrition, metabolism and genetics

Mechanisms for modification of porcine growth by beta-adrenergic agonists

H.J. Mersmann

Department of Pediatrics, Baylor College of Medicine, Houston, TX 77030.

Abstract

In pigs, oral beta-adrenergic receptor (βAR) agonists increase growth rate and deposition of skeletal muscle and decrease the percentage of carcass fat. In many cases, feed intake and the rate of fat deposition are decreased. The physiological agonists for βAR are epinephrine and norepinephrine. Activation of βAR increases cyclic adenosine monophosphate (cAMP), which then activates protein kinase A - an enzyme that phosphorylates proteins. Many potential mechanisms arise from the phosphorylation system of βAR-cAMP-protein kinase A. For example, hormone-sensitive lipase is activated to increase lipid degradation and acetylCoA carboxylase is inactivated to inhibit lipid synthesis. In porcine adipose tissue, lipid degradation is increased and lipid synthesis is decreased *in vitro* and *in vivo*. An enigma in pigs is that several βAR agonists that modulate growth (e.g. clenbuterol, cimaterol and ractopamine) have relatively weak activity *in vitro* and *in vivo*. There is a cAMP-response element (CRE) on the 5'-regulatory region of many genes and phosphorylation of the CRE-binding protein activates transcription. In pigs, synthesis of skeletal muscle protein and actin mRNA are increased. Muscle protein degradation is decreased in cattle and sheep, but there is no evidence for this in pigs. In pigs, blood flow to adipose tissue and skeletal muscle is increased. There is no consistent evidence for modification of hormones. Overall, mechanistic studies have produced both positive and negative results. This is not surprising given the many variables in experiments to study mechanisms. The specific βAR agonist used, duration of treatment, experimental design, animal age, breed, and species, husbandry practices and nutrition are highly variable. Furthermore, different skeletal muscles, adipose tissue depots and laboratory methodologies are used. Finally, there are several mechanisms for animals to adjust to chronic adrenergic stimulation by decreasing the effects of the βAR agonist. The adjustment in pigs is rather rapid so that the βAR effects are observed for only a few weeks.

Introduction

Beta-adrenergic receptor (βAR) agonists are compounds that bind to and activate βAR. Chronic oral administration of some βAR agonists increases growth rate and skeletal muscle deposition and decreases the percentage of fat in the carcasses of cattle, chickens, pigs, and sheep. In many cases decreased feed intake and rate of fat deposition are observed (see Watkins *et al*., 1990; Anderson *et al*., 1991; Moloney *et al*., 1991; Moody *et al*., 2000).

Effects of βAR agonists in pigs:

- Increased growth rate = consistent (except perhaps in entire boars).
- Reduced feed intake = variable.
- Increased muscle mass/rate of muscle deposition = consistent.
- Decreased fat mass/% fat = consistent.
- Rate of fat deposition = variable.
- Responses short-lived = desensitisation of βAR.

βAR structure and function.

Beta-adrenergic receptors belong to a family of membrane-bound receptors that traverse the plasma membrane seven times. There are more than 400 amino acids in the βAR. The receptors are glycosylated and the glycosylated molecular weight (MW) is about 65,000 daltons. There are three subtypes of βAR, the β_1AR, β_2AR and β_3AR. There are no introns in the gene for subtypes 1 and 2, but subtype 3 has an intron near the 3' end of the coding sequence. The continuous protein chain weaves in and out of the plasma membrane so that there are seven transmembrane domains (tm) with helical structure and a number of hydrophobic amino acids. The tm regions are essentially cylindrical in shape. The hydrophobic amino acids allow the tm to be compatible with the lipid membrane structure in which it is inserted. The βAR also has an initial amino-terminal extracellular tail and three extracellular loops, three intracellular loops and finally an intracellular tail containing the carboxyl-terminal amino acid. The extracellular and intracellular loops are formed as the continuous amino acid chain weaves in and out of the plasma membrane.

The ligand binding region is located in the membrane portion of the protein and is a pocket formed in the center of the seven tm. The important amino acids contributing to ligand-binding are an aspartate on tm 3, three serines on tm 4 and tm 5, and a phenylalanine on tm 6. There are many reviews of βAR structure and function (e.g. Strosberg, 1992, 1997; Schwinn *et al*., 1992; Kobilka and Hoffman, 1995), including a recent historical review by Lefkowitz (2004).

The βAR interact with stimulatory G proteins (G_s) proteins via the intracellular portions of the receptor. This interaction/binding has been localised to a number of amino acids in the third intracellular loop and fourth intracellular tail of the receptor. The activated G_s protein releases its alpha subunit that binds to and activates the enzyme, adenylyl cyclase that synthesises cyclic adenosine monophosphate (cAMP). The cAMP, an intracellular messenger, binds to the regulatory subunit of protein kinase A (PKA) which releases and activates the catalytic subunit. The catalytic subunit of cAMP-dependent protein kinase A (PKA) then can phosphorylate a number of proteins. Phosphorylation activates some proteins, e.g., the enzyme, hormone-sensitive lipase, the rate-limiting enzyme in lipolysis, i.e., the removal of fatty acids from triacylglycerol. Phosphorylation inactivates other proteins, e.g., the enzyme, acetylCoA carboxylase, probably the rate-limiting enzyme in fatty acid synthesis. Thus, in the adipocyte, an increase in activity of hormone-sensitive lipase along with a concomitant decrease in activity of acetylCoA carboxylase is expected to increase lipid catabolism and decrease lipid anabolism resulting in decreased fat deposition.

Some genes are responsive to cAMP because they have a cAMP response element (CRE) in the 5'-regulatory region of the gene. The cAMP response element binding protein (CREB) binds to the CRE. In cells wherein the βAR have been stimulated and the cAMP concentration increased with subsequent activation of PKA, CREB (bound to the CRE) is phosphorylated. The phosphorylated CREB changes conformation to bring about an increase in transcription of the gene (Latchman, 2004). Thus, another activity to consider for mechanisms of βAR agonists is to increase the transcription of genes with a CRE. Increased transcription of the gene (increased mRNA) usually results in increased translation or synthesis of the protein specified by the mRNA.

The βAR are present on almost all mammalian cell types. They regulate a multitude of physiological functions such as heart rate and force of contraction, blood pressure, bronchial musculature relaxation, uterine contraction, spleen contraction, release of several hormones including insulin, etc. Consequently, the effects of stimulation of βAR *in vivo* involve many cell types/tissues/organs and create complex potential mechanisms of action. Investigation of mechanisms in specific cell types/tissues/organs *in vitro* is suggestive of potential for such activities *in vivo*, but the true test is to provide evidence for the mechanism *in vivo*. The latter is sometimes difficult to achieve. Likewise, the potential involvement of multiple tissues and organs may produce a complex mixture of effects that might be highly variable depending on the environment, genetics, husbandry practices, and nutrition.

Recommended reviews on the physiology/pharmacology of βAR are Martin (1985) and Hoffman (2001). Reviews with emphasis on adrenergic modulation of adipocyte function include Fain and Garcia-Sainz (1983), Lafontan and Berlan (1993), and Robidoux *et al.* 2004. Reviews with emphasis on βAR modulation of growth in species associated with the agricultural sector are Beermann (1987, 2002), Hanrahan (1987), Smith (1987), Mersmann (1989a, 1995, 1998, 2002), Anderson *et al.* (1991), Moloney *et al.* (1991), Dunshea, (1993), Dunshea and D'Souza (2003), Mills and Mersmann (1995), Moody *et al.* (2000), and Mills (2002).

More recently, there is evidence for alternative pathways to phosphorylate hormone-sensitive lipase (Robidoux *et al*., 2004). These involve the mitogen- activated kinases (MAPK) including the extracellular signal-regulated kinases (ERK). Activation of the MAPK system opens the possibility for a multitude of intracellular signaling mechanisms. Initial studies suggest the classical PKA pathway for phosphorylation is totally operative at low concentrations of βAR agonist, whereas the MAPK pathway is operative only at high concentrations of agonist. Currently there is little or no research indicating the importance of these alternative pathways in the mechanisms for βAR agonists in modification of growth in animals.

Physiological βAR agonists

The physiological βAR agonists are the catecholamines, epinephrine (EPI), the hormone produced by the adrenal medulla and norepinephrine (NOR), the sympathetic nervous system neurotransmitter that also spills into the bloodstream. Thus, NOR can influence physiological processes as a neurotransmitter or as a plasma hormone. Adipose tissue is innervated by sympathetic nervous system fibers so that NOR can be released in the direct vicinity of the adipocytes. Furthermore, in most mammals, under resting conditions, the plasma concentration of NOR is usually two to three times greater than that of EPI (Table 1).

Table 1. Plasma Catecholamine Concentration (pg/mL).

Species	Epinephrine	Norepinephrine
Pig – 4 d[1]	142	456
Pig - 12 wk[2]	250	670
Cow[3]	56	152
Human[3]	64	203
Rat[3]	175	509

[1] Mayfield *et al.* (1989). [2] Mersmann, (1989). [3] Buhler *et al.* (1978).

It is probable that both NOR and EPI play a role in the physiological adrenergic responses. The greater plasma concentration of NOR than EPI suggests that NOR may be the more important ligand *in vivo*, at least in many situations. The plasma catecholamine concentrations are elevated when the animal is stimulated, e.g., frightened or exposed to cold and during fasting. Thus, fasting stimulates lipolysis via phosphorylation/activation of hormone-sensitive lipase to release nonesterified fatty acids (NEFA) to the bloodstream.

Over the latter two-thirds of the 1900s, a large number of compounds were synthesized with structures mimicking that of EPI and NOR. Many of these compounds were agonists, but many were antagonists. Antagonists are compounds with enough structural similarity to an agonist to bind to the βAR ligand-binding site, but with enough structural difference from an agonist that they do not activate the receptors. Antagonists compete with agonists for binding and in effect, act as inhibitors. The ultimate purpose of the extensive synthesis efforts was to produce a pharmaceutical that would be effective in alleviating some pressing clinical problem, e.g., dilation of the bronchial musculature in asthmatics. An ancillary outcome was the availability of multiple compounds that stimulated or inhibited βAR with a variety of potencies (the dose at which a compound stimulates or inhibits) and efficacies (the degree to which a compound stimulates or inhibits). The availability of multiple βAR agonists and antagonists greatly enhanced the investigation of βAR function.

βAR subtypes

Early studies of adrenergic-modulated physiology indicated many functions were controlled by EPI and NOR and that some functions were stimulated and some inhibited. Alquist (1948) was able to discern that there were two types of adrenergic receptors, alpha (αAR) and βAR (see Mersmann, 1989). Almost 20 years later, with the use of various synthetic analogs of EPI and NOR, Lands *et al.* (1967) classified the βAR into the β1AR and β2AR subtypes. Eventually, the αAR were classified into α_1AR and α_2AR and the β3AR was discovered. Each of the αAR subtypes has been further subdivided into three individual receptors with specific structure and function. The αAR and βAR tend to produce antithetical functions effecting a ying-yang type of control system. Cloning of each of the nine receptor subtypes allowed production of mice deficient in one or more of the αAR or βAR subtypes, i.e., knockout mice (Philipp and Hein, 2004). Knockout mice are useful to understand the physiology of a specific receptor because animal functions can be assessed in the absence of that receptor. Of course, the strong impetus to maintain homeostasis by compensatory responses cannot be eliminated. In some cases the knockout mouse presents physiology that suggests no receptor involvement when in fact, the receptor is normally involved, but compensatory responses have masked its effects.

Epinephrine has the capability to stimulate both αAR and βAR and usually is slightly more potent for β_2AR than β_1AR. Norepinephrine has much less affinity for αAR than EPI and is more potent for β_1AR than β_2AR. As interest in adrenergic pharmacology and physiology grew, agonists and antagonists that had specificity for a particular receptor subtype were synthesized. From a clinical perspective it was desirable to have compounds with specificity because treatment with EPI or NOR affected such a large spectrum of tissue/organ functions that patient management was difficult. For example, treatment of asthmatics with isoproterenol, a general βAR agonist not only dilated the bronchi, but adversely stimulated the heart. Today, there is a multitude of synthetic βAR agonists and antagonists available for research and quite a number available for use by clinicians. A few of these compounds are relatively specific for a βAR subtype.

The original classification of the adrenergic receptors mostly utilised tissue and organ preparations *in vitro*, e.g., contracting strips of heart muscle, as physiological and pharmacological models. Tissues or organs were bathed in solutions containing the agonist or antagonist of interest and in the case of heart muscle, the rate and force of contraction were measured. Variation of the concentration of the agonist or antagonist in the bathing media allowed the construction of dose x response curves for quantitative evaluation of the compound. The baseline or non-stimulated activity, the maximal response obtained with very high concentration of the compound, the ED_{50} or the concentration at which 50% of the maximal response is achieved (called the IC_{50} for antagonists), and the slope of response to the agonist or antagonist (when plotted using a logarithmic scale for the dose) are variables that may be quantified and

used to compare compounds. In the 1970s, techniques to measure binding of agonists and antagonists to βAR were developed (see Lefkowitz, 2004). Plasma membrane preparations were used as a source of the receptors and dose x response measurements were accrued. Thus, the ligand-binding results could also be interpreted quantitatively. Comparisons between physiological/pharmacological and ligand-binding results are readily made using the variables obtained from the dose x response curves. The earliest quantification of βAR in porcine tissues was reported in 1986 (see Mersmann and McNeel, 1992). Clearer definitions of ligand-binding to βAR in porcine tissues were reported a few years later (Coutinho *et al.*, 1992; Mersmann and McNeel, 1992; Spurlock *et al.*, 1993a).

Initially, using selected agonists and antagonists with physiological/pharmacological preparations followed later by ligand-binding methods, it became apparent that in the common laboratory species, rats and guinea pigs, the heart contained predominantly β1AR (>90%), whereas the bronchial musculature contained predominantly β2AR (>85%). Later, when the β3AR was discovered (see Arch and Kaumann, 1993), it was shown to predominate (>90%) in rodent brown and white adipose tissue. Thus, the rodent heart, bronchial musculature, and adipocyte became the tissues of preference to study specific βAR subtypes, i.e., prototypical tissues. The predominance of a single βAR subtype in a given tissue allowed the use of these prototypical preparations to classify various synthetic agonists and antagonists using both physiological/pharmacological and ligand-binding approaches. A few βAR agonists and antagonists were discovered that have considerable specificity for the individual prototypical βAR subtypes. Thus, metoprolol and CGP 20,712 are relatively specific for the β_1AR, fenoterol and ICI 118,551 for the β_2AR, and BRL 37,344 and CGP 12,177 for the β_3AR (CGP 12,177 is an agonist for the prototypical β_3AR, but an antagonist for the prototypical β_1AR and β_2AR).

Cloned βAR

Each of the three βAR subtypes has been cloned from several species. The cloned receptors have allowed elegant studies of receptor structure and function, including studies with selectively mutated subtypes. Each of the porcine βAR subtypes was cloned by Mills and associates (Liang *et al.*, 1997; Cao *et al.*, 1998; Smith *et al.*, 2001). Details of ligand-binding properties and function of the cloned porcine β_2AR expressed in Chinese hamster ovary cells (with no endogenous βAR) are available (Liang *et al.*, 2000). Given cloned βAR subtypes from a given species or a single subtype from several species, structural similarities for the nucleotide and derived amino acid sequences may be compared. Within a species the three βAR subtypes have >50% homologies, e.g., the three porcine subtypes have approximately 60% homology (Table 2).

Table 2. Porcine βAR - Subtype homology[1].

β_1AR	β_2AR	β_3AR
62	60	100

Smith *et al.* (2001.)

Table 3. Interspecies homology of βAR subtypes (%).

Subtype	Pig	Human	Rat	Cow
β_1AR[1]	100	92	91	
β_2AR[2]	100	87	84	
β_3AR[3]	100	85		90

[1] Cao *et al.* (1998.) [2] Liang *et al.* (1997.) [3] Smith *et al.* (2001.)

Within a subtype, the homology across species is usually >75% (see comparison of partial porcine sequences in McNeel and Mersmann (1999), and complete porcine sequences in Table 3).

The considerable differences in nucleotide sequence in many cases translate into differences in amino acid sequence that may change the functionality of the protein. This is especially true when the type of amino acid is changed, e.g., a neutral amino acid is substituted for an acidic amino acid or an acidic amino acid is substituted for a basic amino acid. If changes in amino acid sequence change functionality, a particular ligand may be potent for a βAR subtype in one species, but considerably less potent for the same subtype in another species. Furthermore, a ligand determined to be relatively specific for a prototypical receptor subtype may or may not be specific for that same subtype in another species. Specificity can only be determined by experimentation using receptor preparations from the species of interest. A striking example is that the prototypically determined β_2AR antagonist, ICI 118,551 is not specific for the cloned porcine β_2AR, but the prototypically determined β_3AR agonist, BRL 37,344 is specific for the cloned porcine β_2AR (Liang and Mills, 2001). Another example is the reversal of antagonistic and agonistic activity in the same receptor subtype from different species; the long standing prototypical βAR antagonist, propranolol is antagonistic toward the cloned mouse β_3AR, but is a weak agonist for the cloned human and bovine β_3AR (Pietri-Rouxel *et al.*, 1995).

Consequently, a ligand must be determined to be specific for a βAR subtype in the species of interest before it can be used to quantify the receptor subtypes present. Definitive answers only come from studies with cloned receptors expressed in cells that have no endogenous βAR (e.g. Liang *et al.,* 2000).

Single nucleotide differences, i.e., polymorphisms in human β_1AR, β_2AR, and β_3AR have been demonstrated and extensively studied over the past 10 years (Leineweber *et al.*, 2004). These polymorphisms are of interest as potential causative factors in various disease states. The βAR subtype polymorphisms discovered to date do not appear to be disease-causing, but rather provide risk factors for some diseases. Polymorphisms in the βAR subtypes of cattle, pigs, or sheep have not been explored as causative or contributive factors toward modulation of animal growth.

Tissue distribution of βAR subtypes

Given the predominance of specific βAR subtypes in given tissues in model laboratory species, the concept arose that the heart is controlled by β1AR, the bronchial musculature by β2AR and the adipose tissue by β3AR. Investigation of these same tissues in other species indicates that tissues or cell types usually do not contain an overwhelming percentage of β1AR or β2AR, but rather a mixture (Tables 4 and 5).

Table 4. Tissue Distribution of βAR Subtypes[1].

Tissue	Species	β_1AR	β_2AR
Heart	Human	50	50
	Rat	83	17
	Mouse	55	45
Lung	Human	27	73
	Rat	15	85
	Mouse	14	86

[1] All determined by ligand binding:
human = Sano *et al.*, (1993.)
rat = Minneman *et al.*, (1979.)
mouse = Ota *et al.*, (1993.)

Table 5. Tissue Distribution of Porcine βAR Subtypes.

Tissue	Method[1]	β_1AR	β_2AR	β_3AR
Heart	R	72	28	
	B	72	28	
Lung	R	67	33	
	B	58	42	
Liver	R	45	55	
	B	50	50	
Sk. Muscle	R	60	40	
	B	59	41	
Adipose	R	73	20	7
	B	81	19	

[1] R= mRNA with porcine probes (McNeel and Mersmann, 1999).
B= ligand-binding with porcine-specific ligands (Liang and Mills, 2002).

Thus, the human and pig heart contain <75% β1AR. Human bronchial musculature (i.e., lung) contains approximately 70% β2AR. On the other hand, porcine lung contains more β1AR (approximately 60%) than β2AR. In most mammals, the white adipocyte has only small amounts of β3AR in contrast to rodent cells. For example, in the pig (Table 5), the β_3AR population represents only 7% of the total βAR in contrast to rodent cells with approximately 90% β_3AR. As a result, the multitudinous literature regarding regulation of adipocyte metabolism by β3AR, gleaned from studies of rodent adipocytes, does not apply to many species. Porcine skeletal muscle contains approximately 60% β_1AR (Table 5). In several species, the literature suggests skeletal muscle contains predominantly β_2AR (see Mersmann, 1998). However, these reports usually utilise ligand-binding, but with ligands that have not been shown to be specific for the βAR subtypes in the species under study. In this light, the studies of tissue subtypes in pigs (Table 5) indicate remarkable agreement between ligand-binding methods (using a ligand specific for the cloned porcine subtypes) and mRNA measurements (using porcine derived probes).

The structural and functional homology of the βAR subtypes suggests they evolved, one from the other. However, a rational explanation for the driving forces to develop three different subtypes is not forthcoming.

As indicated previously, the subtypes have different affinities for the physiological agonists, EPI and NOR. However, it is difficult to discern how this might be physiologically advantageous because both agonists are usually increased under conditions of stress or fasting and most cell types/tissues/organs, other than the prototypical ones have a mixture of β_1AR and β_2AR. The β_3AR is not as sensitive to desensitisation as the other two subtypes so that in tissues where this receptor is expressed, it may allow βAR function during chronic exposure to βAR agonists. This differential regulation of the subtypes may not be very important in many species where the β_3AR are minimally or not expressed. Regardless, the multiple βAR subtypes present in many tissues of most mammals complicates the comprehension of which receptors might be targeted to specifically elicit a tissue-specific response.

Regulation of βAR

Activation of any receptor must be accompanied by a counter regulatory process; otherwise the receptor would continuously be activated. Catabolism of the physiological agonists EPI and NOR or of synthetic agonists is one mechanism to decrease the stimulatory response. The βAR activated by binding of an agonist are regulated by several acute mechanisms to decrease activity. The activated βAR may be phosphorylated by either the common, less specific kinase, protein kinase A or by a more specific kinase, the βAR kinase (Lohse *et al*., 1990; Lefkowitz, 2004). The βAR kinase, currently named G protein-coupled receptor kinase 2, is part of a family of G protein-coupled receptor kinases. Phosphorylation occurs at a number of serines and threonines predominantly present in the fourth intracellular portion or cytoplasmic tail of the βAR. Phosphorylation causes inactivation of the receptor or desensitization. Desensitization requires the binding of a specific protein, β-arrestin 1 or 2. The β-arrestin binding to the phosphorylated receptor competes with the G_s protein binding to inactivate the receptor (Lefkowitz and Shenoy, 2005). The β_3AR is not as susceptible to desensitization via phosphorylation because it does not contain many of the critical serine/threonine amino acids in the fourth intracellular tail of the receptor (Strosberg and Pietri-Rouxel, 1996).

Longer term regulation involves sequestration and removal of the receptors from the plasma membrane. A lesser number of βAR present on the cell surface yields a lesser response to the continued presence of βAR agonists. Thus, pigs fed a βAR agonist had a decreased adipocyte membrane receptor number within a few days (Spurlock *et al*., 1994). In skeletal muscle, the decrease was less and occurred over a more extended time. Similarly, pigs fed ractopamine had a markedly reduced lipolytic response to intravenous injection of the βAR fenoterol within 4 days of feeding (Dunshea and King, 1995). Porcine adipocytes incubated with a βAR agonist *in vitro* had decreased β_1AR and β_2AR concentration (about 50%) within six hours (Ding *et al*., 2000a). There was no change during this period in the mRNA for either receptor subtype, suggesting this desensitisation mechanism did not involve modulation of the transcripts. Mills (2002) has discussed these mechanisms in detail, including the implications for practical use of βAR agonists to modify animal growth.

As a tissue develops, the βAR total number and/or the subtype population may change. This is clearly demonstrated in the rodent-derived clonal adipocytes, wherein the preadipocytes have predominantly β_1AR with no detectable β_3AR. After differentiation, the adipocyte total βAR number increased dramatically and was composed of 90% β_3AR with only small percentages of the other two subtypes (Feve *et al*., 1991). In children <7 years old, β_2AR predominate in adipose tissue, whereas in adults, β_1AR predominate (Marcus *et al*., 1993); there are only minimal β_3AR present in human adipose tissue. In neonatal and growing pigs, there are no studies of adipocyte βAR subtypes. There was a decrease in total receptor number during post-weaning growth (Mersmann *et al*., 1997), but in another study the receptor number did not change (Akanbi and Mersmann, 1996).

In addition to desensitisation of agonist-activated βAR, there is potential for other hormones to regulate the βAR. Glucocorticoids decreased the total receptor number and the β_1AR and β_3AR, but markedly increased the β_2AR in rodent-derived clonal adipocytes *in vitro* (Feve *et al*., 1992). In porcine adipocytes *in vitro*, glucocorticoids did not change the βAR number after 21 hours, but transiently decreased the mRNA for the type 1 and 2 receptors (Ding *et al*., 2000b). In the porcine cells, insulin slightly increased the total βAR number and decreased the mRNA concentration for subtypes 1 and 2 at 21 hours incubation. In the rodent cells (Feve *et al*., 1994), insulin decreased the β_3AR. Some of the distinctions between the porcine and rodent cells may result from the distinct difference in receptor subtypes or from species-specific hormone responses, or from distinct hormone-sensitive DNA-response elements that regulate transcription of the βAR subtypes.

Alpha-Adrenergic Receptors

In adipose tissue, αAR have important effects to counteract the function of βAR. In rabbit adipose tissue the strong αAR response to EPI overwhelms the βAR response to EPI so that no lipolytic response was measurable (Lafontan and Agid, 1976). Treatment with an αAR antagonist to block the αAR response allowed lipolysis to proceed via the βAR response to EPI. Human adipocytes also have strong αAR responses (Galitzky *et al*., 1993). These antilipolytic responses are mediated by α_2AR. Extensive experiments using porcine adipose tissue *in vitro* were not able to demonstrate an αAR inhibition of lipolysis (Mersmann, 1984a; Hu *et al*., 1987). The lipolytic response of pigs infused with a βAR agonist *in vivo* was not modified by co-infusion of an αAR modulator (Mersmann, 1987). More recent studies using specific α_2AR antagonists in ligand-binding studies indicated the presence of these receptors in porcine adipocytes (Cleale *et al*., 1998a). In addition, βAR-stimulated lipolysis was enhanced by an α_2AR antagonist; these results and positive modification of nitrogen balance suggest selected α_2AR antagonists may be useful to increase muscle deposition with concomitant reduction in fat deposition in swine (Cleale *et al*., 1998a, 1998b).

Response of porcine adipose tissue to βAR agonists

The earliest report of a lipolytic response by porcine adipose tissue to EPI *in vitro* was by Trygstad *et al.* (1972). The lipolytic response *in vitro* was clearly delineated as mediated by βAR (Mersmann *et al.*, 1974). The lipolytic response to isoproterenol, a βAR-specific agonist was blocked with propranolol, a βAR-specific antagonist. Investigations using a wide variety of βAR agonists and antagonists that stimulated or inhibited lipolysis in rats indicated remarkable specificity of the porcine βAR (Mersmann, 1984b,c, 1992, 1996). Ligand-binding to the porcine adipocyte βAR also indicated the unique characteristics of the receptors, but did not lead to an indication of the subtypes present (Mersmann and McNeel, 1992; Mersmann *et al.*, 1993; Mersmann, 1996). The responses to a number of βAR agonists and antagonists were measured *in vivo* in pigs; in general, the results *in vivo* confirmed those observed *in vitro* (Mersmann, 1987). This research suggested the porcine adipocyte βAR had a totally different pharmacological spectrum compared to the rodent adipocyte βAR. The results also indicated that the porcine βAR subtypes could not be classified using βAR agonists or antagonists designated specific for receptor subtypes based on data derived using rodent tissues, i.e., the prototypical responses. Analysis of mRNA (McNeel and Mersmann, 1999) and ligand-binding using compounds specific for the cloned porcine βAR (Liang and Mills, 2002) indicated approximately 75% of the adipocyte βAR were subtype 1 (Table 5).

From the mid-1960s, there were strong indications, mostly by measuring increased plasma NEFA, that porcine lipolysis was stimulated by βAR *in vivo* (Cunningham and Friend, 1965; Hertelendy *et al.*, 1966; Persson *et al.*, 1971; Stanton and Mueller, 1973). In 1984, a number of publications from a USA company, American Cyanamide indicated favorable growth responses (increased growth and muscle mass concomitant with reduced fat mass and feed intake) in pigs treated with the βAR agonist, clenbuterol (Ricks *et al.*, 1984). Similar responses were indicated in cattle, chickens and sheep. In the following years several other βAR agonists were used to stimulate growth in pigs and other species. These include cimaterol, L-644,969, ractopamine, salbutamol, and zilpaterol. Ultimately, after well over a dozen years ractopamine received regulatory clearance in the USA for use in pigs.

The effects of βAR agonists on adipocytes are to increase lipolysis and decrease fatty acid and triacylglycerol synthesis *in vitro*. There are multiple reports from a number of laboratories indicating the stimulation of porcine adipocyte lipolysis *in vitro* by a variety of βAR agonists (summarized in Mersmann, 1989a, 1995, 1998, 2002; Mills and Mersmann, 1995; Bergen, 2001). Using βAR agonists that modify animal growth, demonstration of lipolytic activity in porcine adipocytes or adipose tissue slices *in vitro* is more problematic. Clenbuterol and ractopamine readily bind to porcine βAR (Mersmann *et al.*, 1993; Spurlock *et al.*, 1993b). However, the lipolytic response of each compound was marginal in adipocytes *in vitro* (summarised in Mills and Mersmann, 1995; Mersmann, 2002; Mills, 2002). Assay of lipolysis *in vitro* can be complicated because there are inhibitory responses that could mask the activity of agonists with lesser potency (require higher doses) or efficacy (produce less than a complete response). Stimulation of the adenosine receptor and catabolism of the intracellular messenger for the βAR, cAMP are two potential mechanisms for inhibition of the lipolytic response. Attenuation of these inhibitory responses allowed increases in the lipolytic response of porcine adipocytes to clenbuterol or ractopamine *in vitro*. Regardless, the stimulation was considerably less than that observed with a full agonist, isoproterenol. Addition of ractopamine to an isoproterenol-stimulated adipocyte incubation *in vitro* caused inhibition of the isoproterenol-stimulated lipolytic response (see Mills and Mersmann, 1995). This raises the possibility that ractopamine or clenbuterol might not stimulate lipolysis and inhibit lipid synthesis under some circumstances *in vivo*, but produce the opposite effects, i.e., decrease lipolysis and stimulate lipid synthesis. Lipid synthesis (both fatty acid and triacylglycerol synthesis) has been studied less extensively than lipolysis, but is inhibited by βAR agonists in porcine adipocytes *in vitro* (see Mills and Mersmann, 1995; Mersmann, 1998; Bergen, 2001). There are a few reports of increased lipolysis or decreased lipogenesis in adipose tissue removed from animals chronically treated with a βAR agonist. However, there are also reports indicating no difference in response in tissue obtained from control and treated animals. Also, Dunshea *et al.* (1998c) found that there was no effect of dietary ractopamine on *in vivo* lipogenesis in subcutaneous backfat of finisher boars and gilts. These negative data might result from desensitisation of the receptors that occurs quite rapidly in porcine adipose tissue, as indicated above or from the fact that very subtle changes in rates *in vivo* are not detectable *in vitro* because they are lost in the variability. A change of $\leq$ 5% *in vivo* extended over many days would produce a large change in fat deposition, but not be detectable *in vitro*.

Using cloned porcine β_1AR and β_2AR expressed in Chinese hamster ovary cells coupled with βAR antagonists determined to be specific for the porcine receptor subtypes, it was determined that ractopamine was bound by both porcine βAR subtypes; ractopamine had greater affinity for the β_2AR compared to the β_1AR (Mills *et al.*, 2003). This is contrary to the designation of ractopamine as being specific for the β_1AR, as determined using prototypical preparations and compounds (Moody *et al.*, 2000). The stimulation of porcine adipocyte lipolysis by ractopamine *in vitro* occurred via either receptor, but with greater efficacy produced via the β_2AR. The contributing role of the two receptors in vivo is not clear, but the β_1AR may be more important because it is present at three to four times the concentration of the β_2AR.

Either a decrease in lipid synthesis or an increase in lipid catabolism (lipolysis) or both functioning *in vivo* would reduce fat deposition. As indicated above, some of the early demonstrations of porcine adipocyte βAR function involved demonstration of increased NEFA concentration in the plasma when various adrenergic stimuli were applied. The agonistic or antagonistic activity of a number of compounds with porcine adipocytes *in vitro* was generally verified by infusion of these compounds *in vivo* to yield a change in plasma NEFA and glycerol, the products of lipolysis (Mersmann, 1987). These responses were acute and occurred over a period of hours. In pigs administered ractopamine there was no short- or long-term increase in plasma NEFA concentration suggesting no effect on lipolysis *in vivo* (Dunshea and King 1994). Ractopamine fed during growth from 60 to 90 kg body weight did not affect the rate of deposition of fat in pigs (Dunshea *et al.*, 1993a,b 1998a). Feeding of ractopamine for four days blunted the increase in plasma NEFA after itravenous injection of a lipolytic βAR agonist, fenoterol; feeding ractopamine for 24 days further blunted the response to fenoterol (Dunshea, 1993). The blunted response suggests a desensitisation of the βAR response in the chronically agonist-treated pigs perhaps resulting in no detectable change in the rate of fat deposition. By measuring the plasma NEFA response to increasing doses of the βAR agonists fenoterol and isoproterenol Dunshea *et al.* (1998b) were able to demonstrate that dietary ractopamine decreased adipose sensitivity to βAR stimulation without changing the maximal response.

On the other hand, there are reports of a decreased rate of fat deposition in pigs fed ractopamine (Mitchell *et al.*, 1991) or salbutamol (Oksbjerg *et al.*, 1996). In almost all studies, feeding of a βAR agonist to pigs results in less carcass fat (Watkins *et al.*, 1990; Moloney *et al.*, 1991). In some cases, this may result from dilution of the same total fat by a larger animal and carcass produced from an increased rate of protein deposition concomitant with an unchanged rate of fat deposition. In other cases, the response involves actual changes in the rate of fat deposition. Many factors such as diet composition, the environment, genetics, the specific βAR agonist used, the dose and duration of agonist treatment, etc. may modify the response to chronic feeding of a βAR agonist in pigs (see Moody *et al.*, 2000). Recently, Dunshea *et al.* (2005b) found that dietary ractopamine decreased fat deposition in entire and immunocastrated boars but not gilts and therefore the sex of the animal may also modulate the response to βAR agonists.

Response of porcine muscle to βAR agonist

Chronic administration of specific βAR agonists to birds or mammals, including pigs almost always results in increased growth rate coupled with increased muscle mass. An increase in muscle mass may occur because the protein synthesis rate was increased or the degradation rate was decreased or both. There are a number of reviews that document changes in skeletal muscle mass after treatment with a βAR agonist (Beerman, 1987, 2002; Yang and McElligott, 1989; Mersmann, 1989a, 1995, 1998, 2002; Moloney *et al.*, 1991; Kim and Sainz, 1992; Moody *et al.*, 2000; Mills, 2002)

There are βAR present on skeletal muscle of pigs (Spurlock *et al.*, 1993a). Analysis of βAR subtypes by porcine specific mRNA analysis and by ligand-binding using antagonists specific for the porcine subtypes indicated approximately 60% β_1AR and 40% β_2AR with essentially no β_3AR (Table 5).

The βAR agonist-stimulated increase in muscle mass is dependent on the dose of βAR agonist (see Moloney *et al.*, 1991; Moody *et al.*, 2000; Wray-Cahen, 2001). The deposition of muscle protein requires sufficient dietary amino acids and energy to support the increased protein accretion (Anderson *et al.*, 1991; Dunshea *et al.*, 1993b,1998; Moody *et al.*, 2000).

The βAR agonist-stimulated increase in muscle mass is not the result of cell hyperplasia *in vivo*, as demonstrated in pigs (Bergen *et al.*, 1989). However, some studies of muscle satellite cells *in vitro* indicated increased proliferation (see Beermann, 2002). The muscle mass increase resulted primarily from cell hypertrophy with an increase in white and intermediate cell size coupled with an increase in percentage of the large white fibers (see Moody *et al.*, 2000; Beermann, 2002). Pigs treated with ractopamine had decreased plasma urea nitrogen, suggesting that amino acids were being preferentially utilised for protein synthesis rather than oxidation (Dunshea and King, 1994). This result might be expected, but was not observed in genetically selected lean and obese pigs treated with ractopamine or cimaterol (Yen *et al.*, 1990a, 1990b). Increased muscle protein synthesis has been demonstrated in pigs (e.g., Bergen *et al.*, 1989; Adeola *et al.*, 1992), as well as cattle and sheep *in vivo* (see Mersmann, 1998; Moody *et al.*, 2000; Beermann, 2002). A decrease in protein degradation also might be expected to contribute to increased muscle mass. Degradation is even more difficult to measure *in vivo* than synthesis. Thus, it is seldom directly measured in cattle, pigs or sheep. Studies of muscle cells in vitro are also problematic and have produced evidence for increased protein synthesis and/or decreased protein degradation, but in many cases the rates were not changed (see Mersmann, 1998; Moody *et al.*, 2000; Beermann, 2002).

An increase in porcine muscle total mRNA and mRNA specific for the muscle protein α-actin strongly suggests increased skeletal muscle protein synthesis and a mechanism for cell and muscle hypertrophy (Helferich *et al.*, 1990; Grant *et al.*, 1993). Similar increases in α-actin in sheep and in myosin in cattle after treatment with a βAR agonist add credence to the observations in pigs (see Mersmann, 1998; Moody *et al.*, 2000; Beermann, 2002). Decreased protein degradation is strongly suggested by decreased activity of the muscle proteases, the calpains or by an increase in the calpain inhibitor, calpastatin. These changes have been clearly demonstrated in cattle and sheep, but not in pigs. It should be noted that treatment with a βAR agonist might have adverse effects on postmortem tenderness because of the decreased activity of the calpain protease system that is important in the postmortem tenderization process (Dunshea *et al.,* 2005a). This has been observed in sheep more than other species and does not appear to be a problem in pigs pigs at least at the doses of βAR used commercially (Moody *et al.*, 2000; Dunshea *et al.,* 2005a).

Overall, the evidence indicates treatment of pigs with an appropriate βAR agonist produces an increased rate of protein deposition that results from increased muscle protein synthesis. Evidence for decreased protein degradation is not forthcoming in pigs, but has been accrued for other species.

Other responses to βAR agonists

An increase in blood flow would increase the provision of substrates and energy sources for synthetic processes, e.g., muscle protein synthesis. Increased blood flow also would increase the removal of metabolic products that might inhibit various aspects of metabolism, e.g., the product of lipolysis, NEFA inhibits lipolysis. Thus, an increase in blood flow would favor an increase in muscle mass and perhaps a decrease in fat mass. Heart rate is increased upon administration of many βAR agonists, as observed in pigs (Mersmann, 1987). Blood flow to several adipose tissue depots and skeletal muscles was increased in pigs acutely administered a βAR agonist (Mersmann, 1989b). Continuous administration of a βAR agonist is followed by adaptive desensitization responses to decrease heart rate and blood flow. Decreased blood flow after chronic administration of a βAR agonist has been demonstrated in cattle and sheep, but there are no chronic studies in pigs (see Beermann, 2002). Maintenance of even a 5% increase in blood flow after desensitisation could have considerable effects on provision of metabolic substrates and removal of metabolic products when extended over time.

Chronic administration of an exogenous βAR agonist may alter the plasma concentration of various hormones by modification of the rates of release. Acute infusion of a βAR agonist to pigs caused an increase in endogenous plasma NOR (Mersmann, 1989c). The increase in NOR was only at higher doses, i.e., >10 ng/kg/min for isoproterenol and > 333 ng/kg/min for clenbuterol. With isoproterenol, there was an increase in EPI at the highest dose infused (100 ng/kg/min). Such changes were not observed in cattle in a similar experiment (Blum and Flueckiger, 1988). Infusion of clenbuterol to reserpinized pigs (to reduce endogenous catecholamines) increased plasma NEFA suggesting clenbuterol did not increase lipolysis in vivo by increasing the release of endogenous catecholamines (Hu *et* al., 1988).

Administration of ractopamine to pigs for short or long periods had no effect on plasma concentrations of NEFA or glucose (Dunshea, 1993; Dunshea and King, 1994). The plasma concentration of insulin was slightly lowered. Lower insulin might be expected to decrease adipocyte lipid synthesis and increase lipolysis; however glucose removal rates after insulin injection were not altered by ractopamine treatment. Also, there was no increase in insulin like growth factor-I (IGF-I). In another study using ractopamine-treated genetically lean and obese pigs, there was no ractopamine effect on plasma glucose, triacylglycerol, or NEFA (Yen *et al.*, 1990a). There was no difference in cimaterol-treated and control pigs in energy expenditure (Yen *et al.*, 1990b).

Studies in pigs or in cattle and sheep chronically administered a βAR agonist do not suggest a predominant mechanism for increased muscle and decreased fat mass involving alteration of hormones (see Moody, 2000; Beerman, 2002; Mersmann, 2002). Because of the variable nature of such studies, it is possible that such a mechanism may produce a portion of the effects in a particular study to the exclusion of other studies.

Summary of possible mechanisms for βAR agonist growth modulation in pigs

The effects of exogenous βAR agonists on animal growth, including assessment of potential mechanisms for the observed responses have been studied for many years by a multitude of laboratories. Given the diversity of approaches including animal genetics, experimental design, husbandry practices, laboratory methodologies, nutrition, and species/breeds it is not surprising that divergent results have been produced. It is also not surprising that some studies gave negative results, whereas many others produced a positive result. Regardless, the following is my summarization of mechanisms that have been proposed to explain the changes in animal and tissue growth in pigs chronically fed specific βAR agonists.

- Adipose tissue/adipocytes.
  - Increase lipid degradation/decrease synthesis.
    - Acute *in vitro* = much evidence, but clenbuterol, cimaterol and ractopamine are weak agonists.
    - Chronic animal treatment + incubation *in vitro* = variable.
    - Acute *in vivo* = increased lipid degradation similar to *in vitro*.
    - Decreased rate of fat deposition = variable
- Skeletal muscle.
  - Increase protein synthesis/decrease degradation.
    - Data *in vitro* = variable whether acute treatment or tissue from treated animals.
    - Synthesis increased *in vivo*, but degradation *in vivo* not demonstrated.
- Blood flow.
  - Increased heart rate.
  - Increased flow to various muscles and adipose depots.
- Endocrine modification.
  - Data highly variable; probably not usually a major factor.
- Energy metabolism.
  - No demonstrated effect.

References

ADEOLA, O., BALL, R.O. and YOUNG, L.G. (1992). Porcine skeletal muscle myofibrillar protein synthesis is stimulated by ractopamine. *Journal of Nutrition* **122**:488-495.

ALQUIST, R. P. (1948). A study of adrenotropic receptors. *American Journal of Physiology* **153:**586-600.

AKANBI, K.A. and MERSMANN, H.J. (1996). β -adrenergic receptors in porcine adipocyte membranes: modification by animal age, depot site, and dietary protein deficiency. *Journal of Animal Science* **74**:551-561.

ANDERSON, D.B., VEENHUIZEN, E.L., JONES, D.J., SCHROEDER, A.L. and HANCOCK, D.L. (1991). The use of phenethanolamines to reduce fat and increase carcass leanness in meat animals. In "Advances in Applied Biotechnology Series, Volume 12. Fat and Cholesterol Reduced Foods: Technologies and Strategies". pp. 43-73. eds C.Haberstroh and C.E. Morris (The Portfolio Publishing Co.: The Woodlands, TX)

ARCH, J.R. and KAUMANN, A.J. (1993). β-3 and atypical β -adrenoceptors. *Medicinal Research Reviews* **13**:663-729.

BEERMANN, D.H. (1987). Effects of β-adrenergic agonists on endocrine influence and cellular aspects of skeletal muscle growth. *Reciprocal Meats Conference Proceedings* **40**:57-63.

BEERMANN, D.H. (2002). ß-adrenergic agonist receptor modulation of skeletal muscle growth. *Journal of Animal Science* **80**(Suppl. 1):E18-E23.

BERGEN, W.G. (2001). The role of cylic AMP elevating agents and somatotropin in transcriptional and post-transcriptional regulation of lipogenesis and lipolysis in *Bos taurus* and *Sus scrofa*. *Recent Research Developments in Lipids* **5**:47-59.

BERGEN, W.G., JOHNSON, S.E., SKJAERLUND, D.M., BABIKER, A.S., AMES, N.K., MERKEL, R.A. and ANDERSON, D.B. (1989). Muscle protein metabolism in finishing pigs fed ractopamine. *Journal of Animal Science* **67**:2255-2262.

BUHLER, H.U., PRADA, M., HAEFELY, W. and PICOTTI, G.B. (1978). Plasma adrenaline, noradrenaline and dopamine in man and different animal species. *Journal of Physiology* **276**:311-320.

CAO, H., BIDWELL, C.A., WILLIAMS, S.K., LIANG, W. and MILLS, S.E. (1998). Rapid communication: Nucleotide sequence of the coding region for the porcine β1-adrenergic receptor gene. *Journal of Animal Science* **76**:1720-1721.

COUTINHO, L.L., BERGEN, W.G., MERKEL, R.A. and SMITH, C.K. (1992). Quantitative characterization of β -adrenergic receptor subtypes in porcine adipocytes. *Comparative Biochemistry and Physiology* **101C**:481-485.

CLEALE, R.M., INGLING, J.M., SEARCH, D.J., HADCOCK, J.R. and PAUCH, M.H. (1998a). Effects of α2-adrenoceptor antagonists on metabolic processes of swine: I. Effects on nonesterified fatty acid and plasma urea nitrogen concentrations in jugularly catheterized pigs. *Journal of Animal Science* **76**:1838-1848.

CLEALE, R.M., INGLING, J.M., SEARCH, D.J., HADCOCK, J.R. and PAUCH, M.H. (1998b). Effects of α2-adrenoceptor antagonists on metabolic processes of swine: II. Nitrogen balance responses. *Journal of Animal Science* **76**:1849-1858.

CUNNINGHAM, H.M. and FRIEND, D.W. (1965). Influence of epinephrine, norepinephrine and nicotine on blood levels of glucose, free fatty acids and amino nitrogen in pigs. *Journal of Animal Science* **24**:41-46.

DING, S-T., McNEEL, R.L., SMITH, E.O. and MERSMANN, H.J. (2000a). Modulation of porcine adipocyte β-adrenergic receptors by a β-adrenergic agonist. *Journal of Animal Science* **78**:919-926.

DING, S-T., SMITH, E.O, McNEEL, R.L. and MERSMANN, H.J. (2000b). Modulation of porcine adipocyte β-adrenergic receptors by hormones and butyrate. *Journal of Animal Science* **78**:927-933.

DUNSHEA, F.R. (1993). Effect of metabolism modifiers on lipid metabolism in the pig. *Journal of Animal Science* **71**:1966-1977.

DUNSHEA, F.R. and KING, R.H. (1994). Temporal response of plasma metabolites to ractopamine treatment in the growing pig. *Australian Journal of Agricultural Research* **45**:1683-1692.

DUNSHEA, F.R. and KING, R.H. (1995). Responses to homeostatic signals in ractopamine-treated pigs. *British Journal of Nutrition* **73**:809-818.

DUNSHEA, F.R. and D'SOUZA, D.N. (2003). Fat deposition and metabolism in the pig. In "Manipulating Pig Production IX", p. 127-150, ed. J.E. Paterson. (Australasian Pig Science Association: Werribee).

DUNSHEA, F.R., KING, R.H., and CAMPBELL, R.G. (1993a). Interrelationships between dietary protein and ractopamine on protein and lipid deposition in finishing gilts. *Journal of Animal Science* **71**:2931-2941.

DUNSHEA, F.R., KING, R.H., CAMPBELL, R.G., SAINS, R.D., and KIM, Y.S. (1993b). Interrelationships between sex and ractopamine on protein and lipid deposition in rapidly growing pigs. *Journal of Animal Science* **71**:2919-2930.

DUNSHEA, F.R., KING, R.H., EASON, P.J. and CAMPBELL, R.G. (1998a). Interrelationships between dietary ractopamine, dietary energy and sex on protein and fat deposition in growing pigs. *Australian Journal of Agricultural Research*. **49**:565-574.

DUNSHEA, F.R., LEURY, B.J. and KING, R.H. (1998b). Lipolytic responses to catecholamines in ractopamine treated pigs. *Australian Journal of Agricultural Research*. **49**: 875-881.

DUNSHEA, F.R., LEURY, B.J. TILBROOK, A.J. and KING, R.H. (1998c). Ractopamine increases glucose turnover without affecting lipogenesis in the pig. *Australian Journal of Agricultural Research* **49**:1147-1152.

DUNSHEA, F.R., RIKARD-BELL, C., CURTIS, M.A., EDWARDS, A.C., GANNON, N.J., HENMAN, D.J., MULLAN, B.P. and VAN BARNEVELD, R.J. (2005). A step-up ractopamine (Paylean) program increases lean tissue in all sexes and decreases fat tissue in boars and immunocastrates In 'Manipulating Pig Production X', p. 150, ed. J.E. Paterson. (Australasian Pig Science Association: Werribee).

FAIN, J.N. and GARCIA-SAINZ, J.A. (1983). Adrenergic regulation of adipocyte metabolism. *Journal of Lipid Research* **24**:945-966.

FEVE, B., BAUDE, B., KRIEF, S., STROSBERG, A.D., PAIRAULT, J. and EMORINE, L.J. (1992). Inhibition by dexamethasone of β 3-adrenergic receptor responsiveness in 3T3-F442A adipocytes. Evidence for a transcriptional mechanism. *Journal of Biological Chemistry* **267**:15909-15915.

FEVE, B., ELHADRI, K., QUIGNARD-BOULANGE, A. and PAIRAULT, J. (1994). Transcriptional down-regulation by insulin of the β 3-adrenergic receptor expression in 3T3-F442A adipocytes: a mechanism for repressing the cAMP signaling pathway. *Proceedings of the National Academy of Sciences USA* **91**:5677-5681.

FEVE, B., EMORINE, L.J., LASNIER, F., BLIN, N., BAUDE, B., NAHMIAS, C. and STROSBERG, A.D. (1991). Atypical β - adrenergic receptor in 3T3-F442A adipocytes. Pharmacological and molecular relationship with the human β 3-adrenergic receptor. *Journal of Biological Chemistry* **266**:20329-20336.

GALITZKY, J., LAFONTAN, M., NORDENSTROM, J. and ARNER, P. (1993). Role of vascular α-2 adrenoceptors in regulating lipid mobilization from human adipose tissue. *Journal of Clinical Investigation* **91**:1997-2003.

GRANT, A.L., SKJAERLUND, D.M., HELFERICH, W.G., BERGEN, W.G. and MERKEL, R.A. (1993). Skeletal muscle growth and expression of skeletal muscle α-actin mRNA and insulin-like growth factor I mRNA in pigs during feeding and withdrawal of ractopamine. *Journal of Animal Science* **71**:3319-3326.

HANRAHAN, J. (1987). Beta-agonists and Their Effects on Animal Growth and Carcass Quality (Elsevier: London).

HELFERICH, W.G, JUMP, D.B, ANDERSON, D.B, SKJAERLUND, D.M, MERKEL, R.A. and BERGEN, W. (1990). Skeletal muscle α-actin synthesis is increased pretranslationally in pigs fed the phenethanolamine ractopamine. *Endocrinology* **126**:3096-3100.

HERTELENDY, F., MACKLIN, L., GORDEN, R., HORINO, M. and KIPNIS, D. (1966). *Proceedings of the Society for Experimental Biology and Medicine* **121**:675-677.

HOFFMAN, B. (2001). Catecholamines, sympathomimetic drugs, and adrenergic receptor antagonist. In The Pharmacological Basis of Therapeutics, 10th edition. pp.235-238. eds J.G. Hardman, L.E. Limbird and A.G. Gilman. (McGraw-Hill: New York).

HU, C-Y., NOVAKOFSKI, J.E. and MERSMANN, H.J. (1987). Hormonal control of porcine adipose tissue fatty acid release and cyclic AMP concentration. *Journal of Animal Science* **64**:1031-1037.

HU, C., NOVAKOFSKI, J.E. and MERSMANN, H.J. (1988). Clenbuterol stimulated increase of plasma free fatty acids in reserpinized pigs. *Comparative Biochemistry and Physiology* **91C**:619-622.

KIM, Y.S. and SAINZ, R.D. (1992). β-adrenergic agonists and hypertrophy of skeletal muscles. *Life Sciences* **50**:397-407.

KOBILKA, B. and HOFFMAN, B. (1995). In Hypertension: Pathophysiology, Diagnosis and Management. pp. 841 -851. eds J.H. Laragh and B.M. Brenner. (Raven Press: New York).

LAFONTAN, M. and AGID, R. (1976). α and β adrenergic receptors in the regulation of rabbit white adipose tissue lipolysis. *Comparative Biochemistry and Physiology* **55C**:85-90.

LAFONTAN, M. and BERLAN, M. (1993). Fat cell adrenergic receptors and the control of white and brown fat cell function. *Journal of Lipid Research* **34**:1057-1091.

LANDS, A. M., ARNOLD, H., MCAULIFF, J.P., LUDUENA, F. P. and BROWN, T. G. JR. (1967). Differentiation of receptor systems activated by sympathomimetieamines. *Nature* 214:597-98

LATCHMAN, D.S. (2004). Eukaryotic Transcription Factors, 4th edn (Elsevier: Amsterdam). pp. 157-162.

LEFKOWITZ, R.J. (2004). Historical review: A brief history and personal retrospective of seven-transmembrane receptors. *Trends in Pharmacological Sciences* **25**:413-422.

LEFKOWITZ, R.J. and SHENOY, S.K. (2005). Transduction of receptor signals by β-arrestins. *Science* **308**:512-517.

LEINEWEBER, K., BUSCHER, R., BRUCK, H. and BRODDE, O.-E. (2004). ß-adrenoceptor polymorphisms. *Naunyn-Schmiedeberg's Archive of Pharmacology* **369**:1-22.

LIANG, W., BIDWELL, C.A., COLLODI, P.R. and MILLS, S.E. (2000). Expression of the porcine β2-adrenergic receptor in Chinese hamster ovary cells. *Journal of Animal Science* **78**:2329-2335.

LIANG, W., BIDWELL, C.A., WILLIAMS, S.K. and MILLS, S.E. (1997). Rapid communication: molecular cloning of the porcine β 2-adrenergic receptor gene. *Journal of Animal Science* **75**:2824.

LIANG, W. and MILLS, S.E. (2001). Profile of ligand binding to the porcine β2-adrenergic receptor. *Journal of Animal Science* **79**:877-883.

LIANG, W. and MILLS, S.E. (2002). Quantitative analysis of β-adrenergic receptor subtypes in pig tissues. *Journal of Animal Science* **80**:963-970.

LOHSE, M.J., BENOVIC, J.L, CARON, M.G. and LEFKOWITZ, R.J. (1990). Multiple pathways of rapid β 2-adrenergic receptor desensitization. Delineation with specific inhibitors. *Journal of Biological Chemistry* **265**:3202-3211.

MARTIN, C. (1985). Catecholamines, serotonin, and related regulators. In Endocrine Physiology. pp.270-318. (Oxford: New York)

MARCUS, C., BOLME, P., KARPE, B., BRONNEGARD, M., SELLDEN, H. and ARNER, P. (1993). Expression of β 1- and β 2-receptor genes and correlation to lipolysis in human adipose tissue during childhood. *Journal of Clinical Endocrinology and Metabolism* **76**:879-884.

MAYFIELD, S.R., STONESTREET, B.S., SHAUL, P.W., BRUBAKK, A.M., SUSA, J. and OH, W. (1989). Plasma catecholamine concentrations of newborn piglets in thermoneutral and cold environments. *Journal of Developmental Physiology* **11**:331-334

McNEEL, R.L. and MERSMANN, H.J. (1999). Distribution and quantification of β1-, β2-, and β3-adrenergic receptor subtype transcripts in porcine tissues. *Journal of Animal Science* **77**:611-621.

MERSMANN, H.J. (1984a). Specificity of β-adrenergic control of lipolysis in swine adipose tissue. *Comparative Biochemistry and Physiology* **77C**:39-42.

MERSMANN, H.J. (1984b). Adrenergic control of lipolysis in swine adipose tissue. *Comparative Biochemistry and Physiology* **77C**:43-53.

MERSMANN, H.J. (1984c). Absence of α-adrenergic inhibition of lipolysis in swine adipose tissue. *Comparative Biochemistry and Physiology* **79C**:165-170.

MERSMANN, H.J. (1987). Acute metabolic effects of adrenergic agents in swine. *American Journal of Physiology* **252**:E85-E95.

MERSMANN, H.J. (1989a). In Animal Growth Regulation, pp.337-357. eds D.R. Campion, G.J. Hausman and R.J. Martin. (Plenum: New York).

MERSMANN, H.J. (1989b). Acute changes in blood flow in pigs infused with β-adrenergic agonists. *Journal of Animal Science* **67**:2913-2920.

MERSMANN, H.J. (1989c). Influence of infused β-adrenergic agonists on porcine blood metabolites and catecholamines. *Journal of Animal Science* **67**:2633-2645.

MERSMANN, H. (1992). Regulation of porcine adipose tissue lipolytic activity by adrenergic agonists and antagonists. *Comparative Biochemistry and Physiology* **102C**:407-411.

MERSMANN, H.J. (1995). Species variation in mechanisms for modulation of growth by β-adrenergic receptors. *Journal of Nutrition* **125**:1777S-1782S.

MERSMANN, H.J. (1996). Evidence of classic β 3-adrenergic receptors in porcine adipocytes. *Journal of Animal Science* **74**:984-992.

MERSMANN, H. (1998). Overview of the effects of β-adrenergic receptor agonists on animal growth including mechanisms of action. *Journal of Animal Science* **76**:160-172.

MERSMANN, H.J. (2002). β-Adrenergic receptor modulation of adipocyte metabolism and growth. *Journal of Animal Science* **80(E.** Suppl. 1):E24-E29.

MERSMANN, H.J., AKANBI, K., SHPARBER, A. and McNEEL, R.L. (1993). Binding of agonists and antagonists to the porcine adipose tissue β-adrenergic receptor(s). *Comparative Biochemistry and Physiology* **106C**:725-732.

MERSMANN, H.J., BROWN, L.J., UNDERWOOD, M.C. and STANTON, H.C. (1974). Catecholamine-induced lipolysis in swine. *Comparative Biochemistry and Physiology* **47B**:263-270.

MERSMANN, H.J, CAREY, G.B. and SMITH, E.O. (1997). Influence of nutritional weaning on porcine adipocyte β-adrenergic and adenosine A1 receptors. *Journal of Animal Science* **75**:2368-2377.

MERSMANN, H. and McNEEL, R.L. (1992). Ligand binding to the porcine adipose tissue β-adrenergic receptor. *Journal of Animal Science* **70**:787-797.

MILLS, S.E. (2002). Implications of feedback regulation of β-adrenergic signaling. *Journal of Animal Science.* **80(**Suppl. 2**)**:E28-E32.

MILLS, S.E. and MERSMANN, H.J. (1995). In The Biology of Fat in Meat Animals. pp.1-34. eds S.B. Smith and D.R. Smith. (American Society of Animal Science: Champaign).

MILLS, S.E., SPURLOCK, M.E. and SMITH, D.J. (2003). β-adrenergic receptor subtypes that mediate ractopamine stimulation of lipolysis. *Journal of Animal Science* **81**:662-668.

MINNEMAN, K.P., HEGSTRAND, L.R. and MOLINOFF, P.B. (1979). Simultaneous determination of β-1 and β-2-adrenergic receptors in tissues containing both receptor subtypes. *Molecular Pharmacology* **16**:34-46.

MITCHELL, A.D., SOLOMON, M.B. and STEELE, N.C. (1991). Influence of level of dietary protein or energy on effects of ractopamine in finishing swine. *Journal of Animal Science.* **69**:4487-4495.

MOLONEY, A.P., ALLEN, P., JOSEPH, R.L. and TARRANT, P.V. (1991). Influence of β-adrenergic agonists and similar compounds on growth. In Growth Regulation in Farm Animals: Advances in Meat Research volume 7, pp.455-513. eds A.M. Pearson and T.R. Dutson. (Elsevier: New York).

MOODY, D.E., HANCOCK, D.L. and ANDERSON, D.B. (2000). Phenethanolamine repartitioning agents. In Farm Animal Metabolism and Nutrition pp.65-96. ed J.P. F. Mello. (CABI: New York).

OKSBJERG, N., FERNANDEZ, J.A., JORGENSEN, H., OLSEN, O.H., ROLPH, T. and AGERGAARD, N. (1996). Effects of salbutamol on protein and fat deposition in pigs fed two levels of protein. *Journal of Animal Physiology and Animal Nutrition* **75**:1-12.

OTA, A., MATSUI, H., ASAKURA, M. and NAGATSU, T. (1993). Distribution of β 1- and β 2-adrenoceptor subtypes in various mouse tissues. *Neuroscience Letters* **160**:96-100.

PERSSON, B., GENTZ, J., HAKKARAINEN, J. and KELLUM, M. (1971). *Pediatric Research.* **5**:435-445.

PHILIPP, M. and HEIN, L. (2004). Adrenergic receptor knockout mice: distinct functions of 9 receptor subtypes. *Pharmacology and Therapeutics* **101**:65-74.

PIETRI-ROUXEL, F. and STROSBERG, A.D. (1995). Pharmacological characteristics and species-related variations of β 3-adrenergic receptors. *Fundamentals of Clinical Pharmacology.* **9**:211-218.

RICKS, C.A., BAKER, P.K. and DALRYMPLE, R.H. (1984). Use of repartitioning agents to improve performance and body composition of meat animals. *Reciprocal Meats Conference Proceedings* **37**:5-11.

ROBIDOUX, J., MARTIN, T.L. and COLLINS, S. (2004). β-adrenergic receptors and regulation of energy expenditure: a family affair. *Annual Reviews of Pharmacology and Toxicology* **44**:297-323.

SANO, M., YOSHIMASA, T., YAGURA, T. and YAMAMOTO, I. (1993). Non-homogeneous distribution of β 1- and β 2-adrenoceptors in various human tissues. *Life Sciences* **52**:1063-1070.

SCHWINN, D.A., CARON, M.G. and LEFKOWITZ, R.J. (1992). The β -adrenergic receptor as a model for molecular structure-function relationships in G-protein-coupled receptors. In The Heart and Cardiovascular System, 2nd Edition, pp.1657-1684, eds H.A. Fozzard, E. Haber, R.B. Jennings, A.M. Katz and H.E. Morgan. (Raven Press: New York).

SMITH, T.R., BIDWELL, C.A. and MILLS, S.E. (2001). Rapid communication: nucleotide sequence of the porcine β3-adrenergic receptor gene. *Journal of Animal Science* **79**:781-782.

SMITH, S.B. (1987). Effects of β-adrenergic agonists on cellular metabolism *Reciprocal Meats Conference Proceedings* **40**:65-74.

SPURLOCK, M.E., CUSUMANO, J.C., JI, S.Q., ANDERSON, D.B., SMITH, C.K., HANCOCK, D.L. and MILLS, S.E. (1994). The effect of ractopamine on β-adrenoceptor density and affinity in porcine adipose and skeletal muscle tissue. *Journal of Animal Science* **72**:75-80.

SPURLOCK, M.E., CUSUMANO, J.C. and MILLS, S.E. (1993a). (-)-[3H]-dihydroalprenolol binding to β-adrenergic receptors in porcine adipose tissue and skeletal muscle membrane preparations. *Journal of Animal Science* **71**:1778-1785.

SPURLOCK, M.E., CUSUMANO, J.C. and MILLS, S.E. (1993b). The affinity of ractopamine, clenbuterol, and L-644,969 for the β-adrenergic receptor population in porcine adipose tissue and skeletal muscle membrane. *Journal of Animal Science* **71**:2061-2065.

STANTON, H. and MUELLER, R. (1973). Metabolic responses induced in neonatal swine by norepinephrine, epinephrine, and isoproterenol. *Proceedings of the Society for Experimental Biology and Medicine* **143**:492-494.

STROSBERG, A.D. (1992). Biotechnology of β-adrenergic receptors. *Molecular Neurobiology* **4**:211-250.

STROSBERG, A.D. (1997). Structure, function and regulation of the β-adrenergic receptors. In Recombinant Cell Surface Receptors: Focal Point for Therapeutic Intervention, pp.55-73. ed M.J. Browne. (Landes Bioscience: Georgetown, TX).

STROSBERG, A.D. and PIETRI-ROUXEL, F. (1996). Function and regulation of the β 3-adrenoceptor. *Trends in Pharmacological Science* **17**:373-381.

TRYGSTAD, O., FOSS, I., VOLD, E. and STANDEL, N. (1972). Suppressed lipolysis in genetically fat pigs. *FEBS Letters* **26**:311-314.

WATKINS, L.E., JONES, D.J., MOWREY, D.H., ANDERSON, D.B. and VEENHUIZEN, E.L. (1990). The effect of various levels of ractopamine hydrochloride on the performance and carcass characteristics of finishing swine. *Journal of Animal Science*. **68**:3588-3595.

WRAY-CAHEN, D. (2001). Performance-enhancing substances. In Swine Nutrition, Second Edition. pp.427-446. eds A.J. Lewis and L.L. Southern (CRC Press: Boca Raton)

YANG, Y.T. and McELLIGOTT, M.A. (1989). Multiple actions of β-adrenergic agonists on skeletal muscle and adipose tissue. *Biochemical Journal* **26**:1-10.

YEN, J.T, MERSMANN, H.J., HILL, D.A. and POND, W.G. (1990a). Effects of ractopamine on genetically obese and lean pigs. *Journal of Animal Science* **68**:3705-3712.

YEN, J.T., MERSMANN, H.J., NIENABER, J.A., HILL, D.A. and POND, W.G. (1990b). Responses to cimaterol in genetically obese and lean pigs. *Journal of Animal Science* **68**:2698-2706.

Effect of Bioplex® copper and zinc on pig carcass and meat quality

A. Hernández*, B.P. Mullan**, K. Turnley***, D.N. D'Souza*** and J.R. Pluske*

*Murdoch University, Murdoch, WA 6150. **Department of Agriculture, South Perth, WA 6151. ***Alltech Biotechnology PL, Dandenong, Vic. 3175.

The effect of copper (Cu) supplementation on back fat deposition in pigs is not conclusive. High dietary levels of copper sulphate decreased back fat thickness (Dove, 1992; Kawas *et al*., 1996), while including organic forms of Cu tended to increase back fat (Fremaut, 2003) or had no effect (Henman, 2001). In addition, the effect of Cu on meat quality has not been well studied. The aim of this experiment was to compare carcass and meat quality from pigs fed Cu and Zn in the form of three levels of Bioplex® to a standard diet supplemented with sulphates. The experiment was designed as a completely randomised arrangement of treatments, with four experimental diets. The study used 160 female pigs (Large White x Landrace) through the growing and finishing phases (25–107 kg live weight). The diets fed were: 'low' Bioplex (LB) aimed at providing 25 ppm of Cu and 40 ppm of Zn per kg diet, 'medium' Bioplex (MB) aimed at providing 80 ppm of Cu and Zn per kg diet and 'high' Bioplex (HB) and 'high' Sulphate or standard (HS) aimed at providing 160 ppm of Cu and Zn per kg diet. Copper and Zn levels refer to total levels in the diets. The mineral supplement incorporated into the diets contained Cu and Zn sulphate or Bioplex® Cu and Bioplex® Zn (Alltech Biotechnology P/L, Victoria, Australia) according to their required levels in each diet. Pigs were fed *ad libitum*. A random sub-sample of four pigs per pen (five pens/treatment) was chosen for carcass and meat quality analyses. Subcutaneous fat deposition was assessed on the hot carcasses at the P2 site and belly. The pork quality indices measured were drip loss, colour (L*, a*, b*) and pH, on the *M. longissimus thoracis* between the 12th and 13th rib, with samples removed at 24 h post slaughter and standardised to 20 mm thickness. Data were analysed by one-way analysis of variance.

Table 1. Effect of feeding three levels of Bioplex® Cu and Zn compared to a standard diet supplemented with sulphates on subcutaneous fat depth and pork quality of growing/finishing pigs.

		Experimental diets					
Targeted levels		LB	MB	HB	HS		
- Copper (ppm)		25	80	160	160		
- Zinc (ppm)		40	80	160	160		Statistics
Mineral form		Bioplex	Bioplex	Bioplex	Sulphate	s.e.d.[2]	P=
n=[1]		20	20	20	20		
Hot carcass weight (kg)		72.2	73.7	73.7	74.0	2.39	0.357
Dressing percentage (%)		67.1	68.0	68.1	68.8	1.31	0.060
Subcutaneous fat depth (mm)	- P2	12.9	15.9	14.3	15.2	2.42	0.082
	- Belly	18.9	22.9	21.7	21.6	3.61	0.158
Meat quality: - pH		5.46	5.49	5.49	5.50	0.045	0.415
- Drip loss (mg)		43.9	43.8	41.2	48.5	14.32	0.308
- Colour:	- L*	53.1	52.0	52.2	52.4	2.32	0.669
	- a*	5.62	5.77	5.27	5.59	0.412	0.840
	- b*	4.35	4.13	3.95	3.97	0.741	0.674

[1] Number of pigs sampled/ treatment; [2] SED: Standard error of difference.

Although not significant, carcasses from LB-fed pigs tended to have a lower dressing percentage (P=0.060) and tended to be leaner (P=0.082) compared to pigs fed the other diets. The results also indicate that supplementation with Bioplex® Cu and Zn did not have a detrimental effect on pork quality. These data suggest that including Bioplex® Cu and Zn at lower rates may have a beneficial impact on subcutaneous fat depth.

The financial support of Alltech Biotechnology P/L and Murdoch University are acknowledged.

References

DOVE, C.R. (1992). *Journal of Animal Science* **70** (Suppl 1) Abs. pp. 15.

KAWAS, J.R., MORENO, J.M., SEPULVEDA, J.I., ALEJO, T.E. and GARZA, J.F. (1996). *Journal of Animal Science* **74** (Suppl. 1) Abs. pp. 183.

HENMAN, D. (2001). *Proceedings of Alltech's 17th Annual Symposium*, Lexington KY, USA, pp. 297-304.

FREMAUT, D. (2003). *Proceedings of Alltech's 19th Annual Symposium,* Lexington KY, USA, pp.171-178.

Feeding pigs low levels of organic copper and zinc during the grower and finisher phases benefits performance and reduces faecal excretion

A. Hernández*, B.P. Mullan**, K. Turnley***, D.N. D'Souza*** and J.R. Pluske*

*Murdoch University, Murdoch, WA 6150. **Department of Agriculture, South Perth, WA 6983. ***Alltech Biotechnology PL, Dandenong South, Vic. 3175.

Using organic forms of copper (Cu) and zinc (Zn) in the diets of nursery pigs reduces the content of these minerals in the diet without adversely affecting pig performance and while also reducing faeces excretion (Wu *et al.*, 2001). However, few studies have examined the effect of feeding Cu and Zn at low levels in both grower and finisher pigs. The aim of this experiment was to compare the performance and faecal mineral excretion of pigs fed Cu and Zn in an organic form as Bioplex® or in an inorganic form as sulphate, at two levels of dietary inclusion. The experiment was designed as a 2x2 factorial, with two mineral forms (organic and inorganic) and two inclusion levels (low and high). One hundred and sixty female pigs (Large White x Landrace) were used through the growing and finishing phases (25–107 kg live weight). The low level aimed to provide 25 ppm of Cu and 40 ppm of Zn per kg while the high level aimed to provide 160 ppm of Cu and 160 ppm of Zn per kg. These levels were fed in diets formulated for the growing and finishing phases of pigs. The mineral supplement incorporated in the diets contained Cu and Zn sulphate or Bioplex® Cu and Bioplex® Zn (Alltech Biotechnology P/L, Victoria, Australia) according to their required levels in each diet. Pigs were fed *ad libitum*. Measurements of individual live weight were made weekly throughout the experiment and faecal samples were collected from a random sub-sample of 4 pigs/pen (5 pens/treatment) at 36 and 97 kg live weight. Analysis of variance, using the pen as the unit, was used to examine the main effects of mineral form and inclusion level, and all interactions between these factors on average daily gain (ADG), voluntary feed intake (VFI), feed conversion ratio (FCR) as well as faecal excretion of Cu and Zn. Growth performance was analysed over the experimental period and faecal excretions were averaged from individual samples collected during the grower and finisher periods.

Table 1. Performance and faecal excretion of grower pigs fed Bioplex® or sulphate at low or high levels.

Targeted levels	Experimental diets					Statistics		
- Copper (ppm)	25	25	160	160		P=		
- Zinc (ppm)	40	40	160	160				
Mineral form	Sulphate	Bioplex	Sulphate	Bioplex	s.e.d.[2]	IL[3]	MF[4]	IL x MF
n=[1]	5	5	5	5				
Live weight (kg)								
- Start	24.4	24.6	24.3	24.8	1.31	0.995	0.710	0.823
- Final	106.8	107.7	107.5	107.3	1.15	0.828	0.641	0.470
ADG (g)	913	920	924	922	23.3	0.635	0.864	0.799
VFI (kg/pig/d)	2.48	2.42	2.46	2.39	0.071	0.613	0.154	0.956
FCR	2.76^{b}	2.64^{a}	2.68^{ab}	2.61^{a}	0.055	0.088	0.012	0.522
Faecal levels								
- Cu (mg/kg)	122^{b}	148^{b}	701^{a}	698^{a}	28.0	<0.0001	0.509	0.421
- Zn (mg/kg)	370^{c}	290^{b}	732^{a}	764^{a}	28.0	<0.0001	0.188	0.006

[1]Number of pens/treatment, with eight pigs per pen; [2] SED: Standard error of difference; [3] IL: inclusion level, [4] MF: mineral form; ^{a,b,c} means in the same row with different superscripts differ significantly (P<0.05)

There were no statistical differences in final live weight, ADG or VFI between treatments, however FCR was improved (P=0.012) by including Bioplex® in the diet. There was a significant main effect of inclusion level on the excretion of Cu in faeces (P<0.0001) and a significant interaction IL x MF on the excretion of Zn (P=0.006), with pigs fed LB having a lower concentration of Zn in faeces than pigs fed LS. These data indicate that total dietary levels of 25 and 40 ppm of Bioplex® Cu and Zn during the grower and finisher phase had a beneficial impact on performance and the amount of Cu and Zn excreted into the environment.

The financial support of Alltech Biotechnology P/L and Murdoch University are acknowledged.

References

WU, C., TSUNODA, A., BOLLINGER, D.W., CARLSON, M.S., VEUM, T.L. and TIBBETTS, G.W. (2001). *Proceedings of Alltech's 17th Annual Symposium* Lexington, KY, USA, pp. 285-295.

Acute fasting of the neonatal piglet stimulates ghrelin secretion

M.J. Gresham*, R.E. Newman*, P.A. Sheehy*, P.C Thomson*, L.R. Giles**, K. Scrimgeour* and P.C. Wynn*

*University of Sydney, Camden, NSW 2570. **Department of Primary Industries, Camden, NSW 2570.

Ghrelin, an octanylated peptide of 28 amino acids is produced in X/A oxyntic cells of the stomach and is the endogenous ligand of the growth hormone secretagogue receptor (GHS-R) located in hypothalamic nuclei. Although ghrelin induces feeding in humans and rats through the GHS-R (Wren *et al.*, 2001), little is known of its role in the neonatal pig. In this pilot study, we tested the hypothesis that feed deprivation in the neonatal pig stimulates the stomach to synthesise and secrete ghrelin into the circulation.

Eleven piglets of mixed sex (hybrid, mainly Large White x Landrace) were selected from a single litter 12 h after birth. Six piglets selected randomly were separated for 12 h, as a group under an infrared light. Three treated animals were sacrificed by CO_2 asphyxiation 24 h after birth (24 h), while the three remaining piglets were returned to the sow and sacrificed at 108 h after birth (108 h). Five piglets remained with the sow as controls and three piglets were sacrificed at 24 h, while the remaining two were sacrificed at 108 h. Blood samples (5 ml) and stomach tissue were collected. Plasma glucose and triglyceride concentration were measured by auto analysis. Total stomach tissue and total plasma ghrelin content were analysed by radioimmunoassay.

Table 1. Mean total plasma ghrelin, glucose and triglyceride concentrations and stomach tissue ghrelin content in 11 neonatal piglets. Piglets either remained on the sow (N=5) or were separated (N=6) between 12 h and 24 h after birth and sampled following asphyxiation with CO_2 at 24 h and 108 h after birth.

Piglet treatment	Remained on sow		Separated 12-24 h after birth	
Sampling time post-partum	24 h	108 h	24 h	108 h
Plasma ghrelin (pg/ml)	367±28[a]	741±174[a]	380±14[a]	1164±51[b]
Tissue ghrelin (pg/ml/g)	116±17[a]	303±23[b]	287±57[b]	181±54[b]
Plasma glucose (mMol/L)	7.0±0.96[a]	9.0±0.05[a]	4.3±0.96[b]	8.3±1.26[a]
Plasma triglyceride (mMol/l)	0.5±0.13[a]	1.9±0.64[b]	0.3±0.15[a]	2.3±0.14[b]

[a,b]Mean±SEM values within rows with different superscripts differ significantly (P≤ 0.05)

Feed deprivation for 12 hours on day one in the neonate stimulated ghrelin synthesis in stomach tissue ($P<0.05$) but not in plasma (Table 1). However, plasma ghrelin concentrations increased ($P<0.05$) in response to this treatment at 108 h. Conversely, the production of ghrelin in the stomach was increased ($P<0.05$) in acutely-fasted pigs at 24 h but not at 108 h (Table 1). Circulating glucose concentrations were suppressed significantly ($P<0.05$) by the 12 h fast, while triglyceride levels showed a similar trend. Thus, both substrates appear to be involved in the energetics of the neonate. The four-fold increase in plasma triglyceride concentrations at 108 h suggested that dietary lipid is more available as an energy substrate at this time point (Mellor *et al.*, 1986), although its relative importance in providing energy will depend on the extent of hydrolysis and fatty acid oxidation. There was little correlation between circulating ghrelin levels and energy availability in the first 24 h, although there may be a negative relationship with stomach ghrelin synthesis. The responses in energy substrate to fasting were not sustained through to 108 h. The persistent elevation in plasma ghrelin in the treated group at 108 h suggested that the acute fast on day one induced a long-term influence on the ghrelin regulatory axis for at least four days. We conclude that there is a temporal delay in secretion of ghrelin from the stomach into the peripheral circulation immediately after birth and hypothesise that a challenge to energy homeostasis at this time may impact on the endocrine regulation of energy metabolism and feeding behaviour for at least several days. Similar investigations involving larger numbers of animals are currently being carried out.

Supported in part by the Australian Research Council and Pfizer Animal Health.

References

WREN, A.M., SEAL, L.J., COHEN, M.A., BRYNES, A.E., FROST, G.S., MURPHY, K.G., DHILLO, W.S., GHATEI, M.A. and BLOOM, S.R. (2001) *Journal of Clinical Endocrinology & Metabolism* **86:** 5992-5995.

MELLOR, D.J. and COCKBURN, F. (1986). *Quarterly Journal of Experimental Physiology* **71**: 361-379.

Oral administration of the growth hormone secretagogue GHRP-6 stimulates growth hormone secretion

R.E. Newman*, M.J. Gresham*, K. Scrimgeour*, P.C. Thomson*, A. Norris*, L.R. Giles**, J. McEwan***, G. Russell-Jones*** and P.C. Wynn*

University of Sydney, Camden, NSW 2570. **Department of Primary Industries, Camden, NSW 2570. ***Access Pharmaceuticals Australia Pty Ltd, Chatswood, NSW 2067.

Enhancing endogenous growth hormone (GH) production in the growing pig could offer an alternative strategy to daily injections with porcine somatotrophin. Previous studies have demonstrated that the metenkephalin analogue GHRP-6 stimulates GH synthesis in the pig as well as its release through the specific GH secretogogue receptor GHSR (Sanchez-Hormigo *et al*., 1998). In addition, previous studies (Russell-Jones, 1995) have shown that Vitamin B12 (VB12) can act as a carrier to transport therapeutic proteins and peptides (such as GHRP-6) through the gastrointestinal tract and into the circulation. We tested the hypothesis that oral administration of a GHRP-6:VB12 conjugate could be used to boost circulating GH and provide a diabetogenic effect on insulin status in the pig. Eighteen female pigs (mainly Large White X Landrace) were allocated randomly at 67.1 ± 1.5 kg (mean ± SEM) live weight to individual pens in the same room maintained at 22.0 ± 0.7ºC with a 12 h (0600 to 1800 h) light regime. Each animal was fed a pelleted VB12-free diet containing 13.7 MJ digestible energy and 6.2 g available lysine per kg. A GHRP-6 analogue was conjugated to dithiopyridyl-VB12, and sprayed onto a VB12 free finisher ration at a concentration of 10.5 mg/kg. Cannulae were introduced into the jugular of each pig via the ear vein 24 h before feeding the experimental diets. Animals were fasted for 18 h before being fed *ad libitum* from 10.00 h onwards a diet containing either the GHRP-6:VB12 conjugate or a control diet containing the VB12 carrier alone. Any residue was removed to determine daily feed intakes. Blood samples (3 ml) were taken at intervals of one hour for 24 h and the plasma stored at -20ºC until assayed. Circulating GH and insulin concentrations were determined by radioimmunoassay.

This study supported the hypothesis that oral administration of a GHRP-6:VB12 conjugate can significantly ($P<0.05$) boost peripheral circulation of GH (Figure 1) and insulin (Figure 2) in the growing pig. These increases were detected two hours after feeding and persisted for three hours for GH and four hours for insulin. The responses, however, were not sustained beyond this time despite the continued presence of conjugate in the feed. In addition, there were no significant differences in feed intakes for the two dietary groups. The study demonstrates that GHRP-6 delivered orally as a VB12 conjugate has the potential to stimulate peripheral GH concentrations. The failure for these responses to be sustained beyond six hours may be due to the desensitisation of pituitary somatotropes in response to continuous activation. The commercial potential of this technology will depend on its optimisation as a feed additive to sustain elevated circulating GH concentrations.

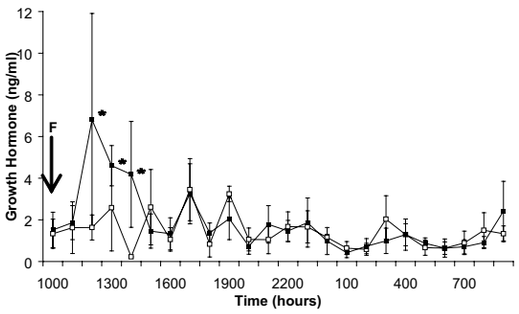


Figure 1 *Plasma GH concentrations in gilts fed (□) VB12 carrier or (■) GHRP-6:VB12 conjugate.*

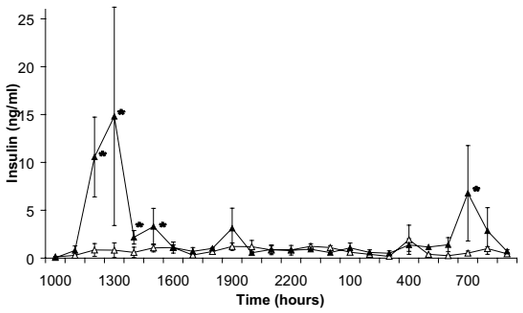


Figure 2 *Plasma insulin concentrations in gilts fed (▲)GHRP-6VB12 conjugate or (Δ) or VB12 carrier .*

(F)*Feeding commenced, *LSD P<0.05*

This work was supported in part by Pfizer Animal Health and the Australian Research Council.

References

RUSSELL-JONES, G.J. (1995). In 'Peptide-based drug design: controlling transport and metabolism' pp. 181-198, eds. M.D. Taylor and G.L. Amidon. (American Chemical Society, Washington, D.C).

SANCHEZ-HORMIGO, A., CASTANO, J.P., TORRONTERAS, R., MALAGON, M.M and GRACIA-NAVARRO, F. (1998). *Life Sciences* **63:**2079-2088.

Acid insoluble ash as a marker to estimate feed wastage

C.D. Hofmeyr, D.J. Kerton and F.R. Dunshea

Department of Primary Industries, Werribee, Vic. 3030.

Wasted feed ends up in effluent and represents a significant cost to pig producers as well as causing potential problems if nutrients contained within the feed flow into the environment. Any measures that reduce feed wastage will improve profitability and sustainability of pig enterprises. However, before feed wastage can be reduced, a reliable way of quantifying feed wastage on a shed or piggery basis is required. Dunshea *et al.* (2003) concluded that n-hexatriacontane or acid insoluble ash (AIA) could be used as markers to estimate feed wastage, although the former is probably too expensive for routine on-farm use.

This study validated a method of measuring feed wastage in commercial piggeries using the method described by Dunshea *et al.* (2003). Two piggeries were involved in the study, each with a different feeding system. One piggery uses an automated feed delivery system with feed available on demand while the other has a more traditional self-feeder system. No exogenous markers were added to the existing diets and instead the acid insoluble ash component in the diets was used as the marker (Dunshea *et al.*, 2003).

The effluent, faeces and feed from finisher pigs were sampled in a single shed on each farm. Using a submersible pump lowered into the effluent channel, effluent samples were pumped sequentially into buckets from which effluent sub-samples were taken. In addition, faecal grab samples were obtained from pigs and feed samples were taken from the feeders above the channel sampled. Sample jars were sealed and returned to the laboratory for further analysis. The samples were dried in a fan forced oven at 85ºC for 108 hrs to determine dry matter content. Acid insoluble ash was analysed in duplicate using the method outlined by Prawirodigo (1999). Samples were taken twice at each farm from the same shed and effluent channel. The intervening period was three weeks at Farm A and one week at Farm B. These visits were designated as Farm A1 and A2 and Farm B1 and B2.

Dry matter measurements for the faecal grab samples and the feed samples were consistent for each farm, although there were differences between the farms. Table 1 depicts the results of the acid insoluble ash determinations. As described earlier, the effluent was pumped into a sequence of buckets from which sub-samples were taken. These buckets are shown in the table as A to F. It was expected that the effluent AIA values would fall between the feed and faecal grab values. With the exception of Farm B2, all the effluent AIA values fell outside the expected range. Reasons for this could include contamination from cement or concrete that sloughed off into the bucket or dirt or clay that ran into the effluent channel. During sampling at Farm A2, it was noted that clay and rainwater were running into the effluent channel.

Table 1. Acid insoluble ash (%) in effluent, faecal and feed dry matter at two piggeries over time*.

				Effluent			Faeces	Feed
Bucket	A	B	C	D	E	F		
Farm A1	16.5	16.8	18.2	18.3	17.4	18.3	12.8	1.7
Farm A2	11.7	17.3	12.2	12.6	10.1	9.7	6.7	1.0
Farm B1	8.6	7.9	6.7	7.3			5.7	1.3
Farm B2	8.2	7.3	6.6	5.9			10.9	1.3

* See text for explanation of Farm A1-A2 and Farm B1-B2 and A-F bucket samples.

In conclusion, the fact that the AIA values for the effluent fell outside the expected range made it clear that either AIA concentration due to fermentation or AIA contamination from other sources occurred. Therefore, AIA is not a reliable marker and it is clear that a unique marker needs to be found. Such a marker should not occur naturally in or around a piggery and would either be added to the feed to enable an accurate determination of AIA or be present in the feed already.

Supported in part by Australian Pork Limited.

References

DUNSHEA, F.R., WATT, M., KERTON, D.J., HOFMEYR, C.D. and EASON, P.J. (2003). A marker method to estimate feed waste. In 'Manipulating Pig Production IX', p. 155, ed J.E. Paterson. (Australasian Pig Science Association: Werribee)

PRAWIRADIGDO, S. (1999). Sources of variability in amino acid availability in cottonseed meal, PhD Thesis, University of Melbourne.

Australian Pork

The rheological properties of liquid feed can be influenced by a single multi-enzyme preparation

S. Jakob*, M. Türk** and T. Zenke**

*Adisseo France S.A.S, rue Marcel Lingot, France. **Leibniz-Institut für Agrartechnik Potsdam-Bornim e.V., Germany.

Swine diets are commonly supplemented with enzymes that hydrolyse non-starch polysaccharides (NSP). Supplementing the diets of piglets and pigs with Rovabio™ Excel (Adisseo, France) can increase the digestibility of nutrients and increase animal performance (Jakob *et al.,* 2003). In addition, NSP-cleaving carbohydrolases have been reported to decrease gut viscosity. However, swine producers are now using liquid feeds and multi-enzyme preparations could act on liquid feed before it is ingested. The objective of the present study was to determine the impact of a multi-enzyme preparation on the rheological parameters of liquid feed.

A wheat-based diet was tested. The basal diet consisted of 720 g/kg wheat, 47 g/kg wheat bran, 175 g/kg soybean meal and 40 g/kg premix. Two treatments were tested: 1) the basal diet and 2) the basal diet supplemented with 50 g/t Rovabio™ Excel AP. Diets were mixed with water to obtain dry matter (DM) contents of between 25% and 35% and pH was adjusted to 5.2 using lactic acid. Mixing and flow curves of the liquid feeds were determined using a rotary viscometer fitted with a horseshoe agitator (mixing curve) or an internal rotating cylinder (flow curve). A mixing curve, expressing the torque (mN m), was established. On the same samples, a flow curve was determined by increasing the shear rate (1/s), enabling precise calculation of the apparent viscosity of the liquid feed (Pa/s). Three repetitions per treatment were carried out and the data obtained were statistically analysed using repeated measures ANOVA and the post Scheffe's test in the Statview software package.

Feed viscosity was reduced by 30% ($p<0.01$) when the enzyme formulation was mixed with the feed for 10 min (Figure 1). The greatest impact of the enzyme on feed viscosity occurred during the first 5 min of mixing. Following 90 min of stirring, the relative torque of the liquid feed was reduced by 40% ($p<0.01$), indicating a continued action and stability of the enzyme complex throughout the trial. Dependent on the measured shear rate, a total reduction of more than 40% ($p<0.01$) of the apparent viscosity of the liquid feed was measured (Figure 2). Such a reduction could help producers save on costs for pumping and maintenance of pumps while providing low viscosity feed to pigs. When the flow curves in relation to the DM content are compared, it becomes clear that the DM content of liquid feed could be increased by 2-2.5% without reducing the initial feed viscosity. This would result in reduced water intake and less manure production without losing the advantages of a liquid feeding system. In conclusion, supplementing liquid feed with Rovabio™ Excel can enhance the rheological properties of liquid feed.

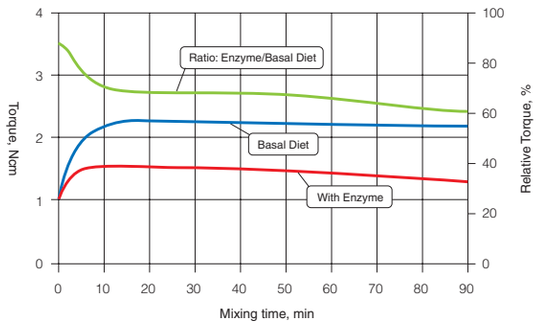


Figure 1*: Mixing curves indicating the changes in the torque (N cm) or the relative torque (%) over time.*

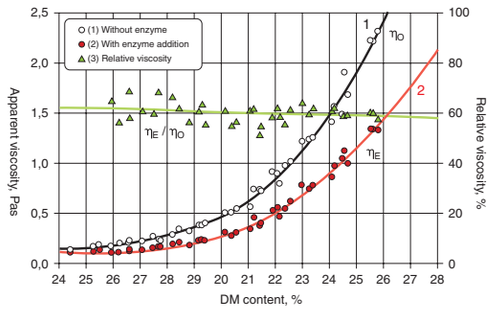


Figure *2:* *Flow curves of the basal diet (o), the basal diet supplemented with a multi-enzyme preparation (●) in relation to the DM content of the liquid feed, indicating the relative viscosity (▲) reduction.*

References

JAKOB, S., GERAERT, P.A., SCHWARTING, G., FERLE, S. and ROTH, F.X. (2003). In 'Manipulating Pig Production IX', p. 50, ed. J.E. Paterson (Australasian Pig Science Association: Werribee).

Plasma glycerol is not a lipostatic signal to reduce feed intake in pigs

D.K. Revell** and I.H. Williams*

*The University of Western Australia, Nedlands, WA 6009. ** Current address: CSIRO Livestock Industries, Wembley, WA 6913.

Blood-borne signals are known to influence feed intake and, in a number of animal models used to investigate human-related metabolic disorders, it has been hypothesised that metabolites of fat metabolism may suppress feed intake. In pig production, it has been well established that feed intake of sows during lactation is inversely related to body fatness (Dourmad, 1991; Revell *et al*., 1998). Fat sows have elevated concentrations of non-esterified fatty acids (NEFA) and glycerol (Revell *et al*., 1998) and both of these metabolites may act to regulate feed intake. Glycerol may serve as a signal of body fatness, as its concentration depends on the hydrolysis of triglycerides from adipose tissues or, in the case of lactating animals, from the mammary gland, rather than reflecting fat absorption from the digestive tract. In this study, we used growing gilts to determine if elevated plasma glycerol concentration may act as a signal to reduce feed intake.

Four Large White x Landrace gilts (78.6 ± 2.83 kg live weight, LW; mean ± SE) were used. Two gilts received a five day continuous intravenous infusion of glycerol at the rate of 76 mg/kg live weight/day via catheters inserted into the jugular vein, whilst the other two gilts received a control infusion of saline. Plasma samples were collected daily for determination of glycerol concentration and voluntary feed intake was determined on a daily basis. Comparisons were made by a t-test.

The intravenous infusion of glycerol significantly increased ($P<0.01$) plasma concentration of this metabolite by two orders of magnitude (21± 1.5 *vs.* 2724 ± 505 μM,), which was higher than we expected based on limited available data on glycerol turnover rate. Voluntary feed intake was not affected by the increased plasma glycerol concentration. The data in Table 1 show the average values across the five days of infusion.

Table 1. Plasma glycerol concentration and voluntary feed intake of gilts that received an intravenous infusion of glycerol or saline.

Pig:	1	2	3	4
Treatment (i.e., infusate):	Saline	Saline	Glycerol	Glycerol
Body weight (kg)	82.5	70.5	79.0	82.5
Plasma glycerol concentration (μM)	22	19	3230	2219
Voluntary feed intake (g/kg LW/day)	39	36	38	38

This study demonstrated that glycerol has no direct role in the control of voluntary feed intake of gilts, even at higher-than-physiological concentrations. Although only two animals received the intravenous glycerol infusion, the marked response in plasma glycerol concentration (two orders of magnitude above control values) without any effect on feed intake indicated to us that further investigation was not warranted. The conclusion that plasma glycerol concentration does not play a central role in regulating feed intake has also been reached by others working with rat models (e.g. Ramirez and Friedman, 1982; Carpenter and Grossman, 1983) or humans (Bjorvell *et al*., 1984). The phenomenon of reduced feed intake shown by fat sows during lactation is unlikely to be associated with a single metabolite, and more likely reflects a complex interrelationship between the central control of feed intake, hormone concentrations, and the supply of substrates for milk production from either body reserves or the diet.

References

BJORVELL, H., HYLANDER, B. and ROSSNER, S. (1984). *International Journal of Obesity* **8**:129-133.

CARPENTER, R.G. and GROSSMAN, S.P. (1983). *Physiology and Behavior* **30**:57-63.

DOURMAD, J.Y. (1991). *Livestock Production Science* **27**:309-319.

RAMIREZ, I. and FRIEDMAN, M.I. (1982). *Physiology and Behavior* **29**:921-925.

REVELL, D.K., WILLIAMS, I.H., MULLAN, B.P., RANFORD, J.L. and SMITS R.J. (1998). *Journal of Animal Science* **76**:1729-1737.

Identification of positive clones of porcine endogenous retrovirus from the Korean native pig bacterial artificial chromosome library

W.Y. Jung*, J.E. Kim*, Y.S. Kwon*, K.C. Jung*, S.L. Yu*, C. Moran**, C.S. Park*, D.I. Jin*, E.W. Park*** and J.H. Lee*

*Chungnam National University, Daejeon 305-764, Republic of Korea. **National Livestock Research Institute, Suwon 441-350, Republic of Korea. ***University of Sydney, Sydney, NSW 2006.

The pig (*Sus scrofa*) is regarded as the best xenotransplant organ donor for humans and pre-clinical trials have already been carried out with pig xenografts. For example, hepatic failure has been overcome using perfusion of human blood through pig livers and porcine hepatocytes as a bridging strategy and pancreatic islet cells have been evaluated as a treatment for Type 1 Diabetes. In addition, foetal neuronal tissue has been implanted as a therapy for Parkinson's disease. However, since Patience *et al.* (1997) reported that a porcine kidney cell line released porcine endogenous retrovirus (PERV) particles capable of infecting human cells there has been considerable concern about xenotransplantation. In addition, the transplantation of porcine pancreatic islets into mice with severe combined immunodeficiency led to *in vivo* expression of PERVs, indicating an increased risk of PERV infection in immunosuppressed human patients (van der Laan *et al.*, 2000). Porcine gamma retroviruses are classified mainly as PERV-A, PERV-B and PERV-C, based on their envelope proteins. They are present at about 30 to 50 copies in the porcine genome (Le Tissier *et al.*, 1997) and the PERV integration sites vary between breeds (Lee *et al.*, 2002; Gorbovitskaia *et al.*, 2003).

An intensive and closed recovery breeding-program for Korean native pigs was commenced a decade ago largely by the National Livestock Research Institute in Korea as part of a national animal conservation program. Using a native pig boar, a Korean native pig bacterial artificial chromosome (BAC) library was constructed with about five genome equivalents of coverage (Jeon *et al.*, 2003). To examine PERV copy number and integration sites in the Korean native pig genome, we screened the BA library with PERV specific protease primers to identify PERV positive clones and three envelope specific primer pairs for the identification of PERV types (Bösch *et al.*, 2000). Currently 45 PERV positive clones comprising 8 PERV-A and 37 PERV-B have been identified. The most potentially pathogenic PERV-C was not identified in this breed, indicating a lower xenotransplantation hazard if these pigs were to be used as donors. The end-sequences of the PERV positive BAC clones generated so far contain 131 new STSs (Sequence Tagged Sites). The BAC end sequences were used to search the NCBI (National Center for Biotechnology Information) human genomic sequences (http://www.ncbi.nlm.nih.gov/), with 16% (21/131) of the usable BAC-end STSs giving significant BLAST (Basic Local Alignment Search Tool) hits (E value < e-5). These matches with the human sequence will help predict the porcine map locations for the PERV inserts. The precise PERV integration sites in Korean native pigs will be determined using the IMpRH (INRA-University of Minnesota porcine Radiation Hybrid) panel (Yerle *et al.*, 1998) and primers designed from the BAC-end sequences. Together with information on the functionality of the inserts, the maps will give valuable information for breeding a PERV-free line of pigs for xenotransplantation.

This work was supported by the Korea Science & Engineering Foundation.

References

BÖSCH, S., ARNAULD, C. and JENSTIN, A. (2000). *Journal of Virology* **74**:8575-8581.

GORBOVITSKAIA, M., LIU, Z., and BOURGEAUX, N. (2003). *Immunogenetics* **55**:262-270.

JEON, J.T., PARK, E.W., and JEON, H.J., (2003). *Molecules and Cells* **16**:113-116.

LE TISSIER, P., STOYE, J. P., TAKEUCHI, Y., PATIENCE, C. and WEISS, R.A. (1997). *Nature* **389**:681-682.

LEE, J.H., WEBB, G.C., ALLEN, R.D.M. and MORAN, C. (2002). *Journal of Virology* **76**:5548-5556.

PATIENCE, C., TAKEUCHI, Y. and WEISS, R.A. (1997). *Nature Medicine* **3**:282-286.

VAN DER LAAN, L.J., LOCKEY, C., GRIFFITH, B.C. (2000). *Nature* **407**:90-94.

YERLE, M., PINTON, P., and ROBIC, A. (1998). *Cytogenet Cell Genet* **82**:182-188

Accuracy of sample weights of varying sample sizes to predict true average batch weight

B. Schauer, N. Cogger, D.E. Lawton and G. Skilton*

Massey University, Palmerston North, New Zealand. *Aorere Farms, Wanganui, New Zealand.

Producers often weigh a sample of pigs to assess the performance of each grower batch. However, little is known about the accuracy with which weighing different portions of a batch ('sample size') predicts the true weight of a batch. This abstract presents the analysis of a historical dataset to determine how accurately different sample sizes represent the true weight of the entire batch.

Weight records were collected routinely on a commercial farrow-to-finish farm in the North Island, New Zealand. The dataset included 130 batches of Large White x Landrace pigs weaned between December 2001 and June 2004. Records of sample weights were available for each mixed-gender pen at transfer from the weaner to the grower shed (in total 1039 pens) and at transfer from the grower to the finisher shed (in total 1219 pens). Pigs in each batch were divided amongst six pens in the weaner shed, eight in the grower shed and up to ten in finisher shed. It was routine farm management to allocate pigs to pens as follows: At weaning, pigs were sized to match pen mates, while at subsequent transfers the smallest pigs of each pen were mixed to create new pens. The median number of pigs in each grower and finisher pen was 13 (range: 6-17) and 10 (range: 8-14), respectively.

One batch with only seven grower pens was excluded from the analysis. Eight grower pens and eight randomly selected finisher pens were chosen to represent each batch (in total 1032 pens per production stage). Per batch random samples with one to seven pens were selected for each production stage. The 'true' weight for all eight pens as well as the mean weight and proportion of pigs for each random sample were calculated. The association between sample weights and true weight was assessed using the R-squared value, which was created in SAS 9.1. Random sampling steps were repeated five times to increase statistical power. The mean of the resulting five R-squared values are presented.

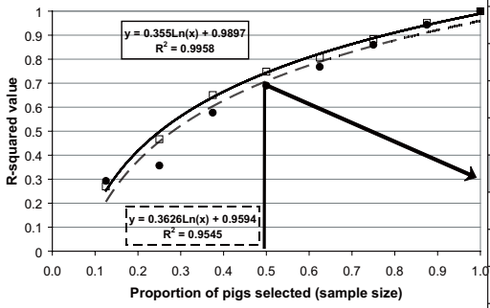


Figure 1. *Change of R-squared value with sample size (grower: ●, dashed line; finisher: □, solid line). The R-squared value measures the strength of association between weight of random samples and the true average weight of the batch. Logarithmic regression lines fitted.*

Sampled proportion	* R^2 grower	* R^2 finisher
0.10	0.12	0.17
0.20	0.38	0.42
0.30	0.52	0.56
0.40	0.63	0.66
0.50	**0.71**	**0.74**
0.60	0.77	0.81
0.70	0.83	0.86
0.80	0.88	0.91
0.90	0.92	0.95

Table 1. *R-squared values (R2) for the relationship between varying sample sizes and the true average weight of the batch as predicted by logarithmic regression equations in Figure 1 (shaded area indicates the proportion of pigs that resulted in 70% accuracy). Predicted R^2 for grower and finisher stages*

The mean weight of a batch was 33.1 kg (95% CI[a]: 32.8-33.4 kg) and 64.8 kg (95%CI: 64.4-65.2 kg) at transfer to the grower and finisher shed, respectively. The median coefficient of variation (CV) was 9.3% (range: 4.6-20.2%) for the grower and 6.7% (range: 2.7-16.0%) for the finisher pens. Increasing the number of randomly selected pens enhanced the accuracy of sample weights, but in a diminishing manner. Fitted logarithmic regression lines were of similar shape for the grower and finisher dataset (Figure 1). Table 1 shows predicted R-squared values for each 0.1 stepwise increase in proportion of pigs contributing to the random sample. The values indicate that, for instance, increasing the sample size from 0.2 to 0.3 enhances the accuracy of sample weight by 14%. Predicted R-squared values are 3-5% lower for grower pens than for finisher pens. The larger between-pen weight variation of grower pens (CV of 9.3% *vs.* 6.7%) may explain the reduced accuracy. However, the impact of between-pen variation on accuracy of random samples needs to be further investigated. These results can be used as a way to balance time and cost factors against accuracy of sample weights. A reasonable accuracy of sample weights of more than 70% will be achieved if at least half the batch is weighed following random selection of pens.

Mean and variation in back fat influence profit of pig production

S. Hermesch

Animal Genetics and Breeding Unit*, Armidale, NSW 2351.

Profit of pig production is influenced by several traits. Selection decisions should therefore be based on an economic index ($Index), which is the sum of estimated breeding values (EBVs) of individual traits multiplied by their economic value. The economic value of a trait represents the change in profit when the trait is increased by one unit, keeping all other traits constant. Cameron and Crump (2001) presented economic values for several performance traits, and noted that the economic value for back fat (BF) depends on the mean BF. Payment systems in Australia often set a base price that is reduced for pigs exceeding a certain limit in BF. Therefore, the proportion of pigs that do not achieve the base price affects the average return per pig and depends not only on the mean value but also on the variation in BF. The aim of this study was to derive economic values for BF assuming different means and standard deviations (sd) in BF.

The calculations were based on a carcass weight of 80 kg and a base price of A$2.30 per kg carcass weight. This base price was reduced by A$0.10 per kg carcass weight for every one mm increase in BF once pigs reached the threshold of 14 mm BF. The average price per kg carcass weight was the proportion of pigs in each BF level multiplied by the corresponding price per kg carcass weight, which was then summed over all BF levels. Five different BF means (11, 12, through to 15 mm) were considered as well as two levels of variation (sd of 2.5 mm and 1.5 mm). Economic values were derived from the change in returns when BF was increased by one mm and were shown on a per slaughter pig basis. Economic values for BF varied considerably from A$ –0.73 to A$ -7.27 per mm per pig (Table 1). Economic values for BF were larger for higher means, since a larger proportion of pigs had a reduced price. Therefore, an increase in mean BF by one mm had a larger effect on returns. Given a mean of 11 mm and 12 mm in BF, economic values for BF were higher in combination with a larger sd since a larger proportion of pigs had 14 or more mm BF. Similarly, the economic values of BF were larger for a higher mean in BF together with a lower SD, since a price penalty applied to more pigs (75% versus 66% for a mean of 15 mm).

Table 1. Average price per kg carcase weight (Price), proportion of pigs with price penalty (Penalty) and economic values (Ec. value) of back fat for different trait means and standard deviations (sd).

SD (mm)	Mean (mm)	11	12	13	14	15
2.5	Price ($/kg)	2.28	2.26	2.22	2.17	2.11
	Penalty (%)	12	21	34	50	66
	Ec. value (A$/mm/pig)	-1.69	-2.76	-4.00	-5.24	-6.31
1.5	Price ($/kg)	2.30	2.29	2.26	2.21	2.14
	Penalty (%)	2	9	25	50	75
	Ec. value (A$/mm/pig)	-0.73	-2.02	-4.00	-5.98	-7.27

Profit functions include both returns and costs of production. Only differences in returns were considered, similar to Hovenier *et al.* (1993) who derived economic values for meat quality. Fatter pigs may have a higher feed intake. However, these differences in costs associated with other traits are taken into account in the economic values of those traits. Many payment systems also consider carcass weight. The concept presented here can be extended easily to incorporate variation in slaughter weight. The example shows that economic values for BF may range considerably between different levels of performance. Individual breeders are able to incorporate their own payment system, their performance in BF and carcass weight in the $Index of PIGBLUP in order to take non-linear economic weights for BF into account. Further, returns were higher for a lower sd especially when the mean BF was closer to the threshold of a price penalty. Causes of variation should be explored to develop avenues to reduce variation. This may include optimisation of matings using information on EBVs to minimise variation in progeny performance.

This work was funded by the APL. *AGBU is a joint venture of NSW Department of Primary Industries and The University of New England.

References

CAMERON, N.D. and CRUMP, R.E. (2001). In 'Manipulating Pig Production VIII', p. 146, ed P.D. Cranwell. (Australian Pig Science Association: Werribee).

HOVENIER, R., BRASCAMP, E.W., KANIS, E., VAN DER WERF, J.H.J. and WASSENBERG, A.P.A.M. (1993). *Journal of Animal Science* **71**:1429-1433.

Australian Pork

Estimated breeding values of sires predict average progeny performance

A.C. Hansson and S. Hermesch

Animal Genetics and Breeding Unit*, Armidale, NSW 2351.

Estimated breeding values (EBVs) reflect the genetic merit of animals. Therefore, ranking of animals based on EBVs assists with selection for particular traits. Sires pass half their genes onto their progeny. Thus, half of the differences in sire EBVs are expected to be reflected in their mean progeny performance. The aim of this study was to analyse whether across-herd EBVs of sires in the National Pig Improvement Program (NPIP) (Crump and Hermesch, 2003) could predict half of the difference in progeny performance for average daily gain (ADG) and back fat (BF).

Across-herd EBVs were available for boars from seven Large White herds participating in the NPIP that were subsequently used as sires in the Gatton Large White herd. These sire EBVs for ADG and BF were generated without using information from Gatton. Progeny (n: 1743) of 29 sires were recorded between September 1999 and December 2004 at Gatton. Each sire had a minimum of 20 progeny. Progeny records were limited to within three standard deviations from the mean for weight at recording (60-114 kg), ADG (465-860 g) and BF (7-17 mm). Using PROC GLM in SAS v. 6.03, regression coefficients (β ± SE) of sire EBVs were calculated adjusting ADG and BF for month within year of recording and sex. Live weight was fitted for BF as a linear co-variable. Sire EBVs are plotted against corrected progeny performance (Figure 1).

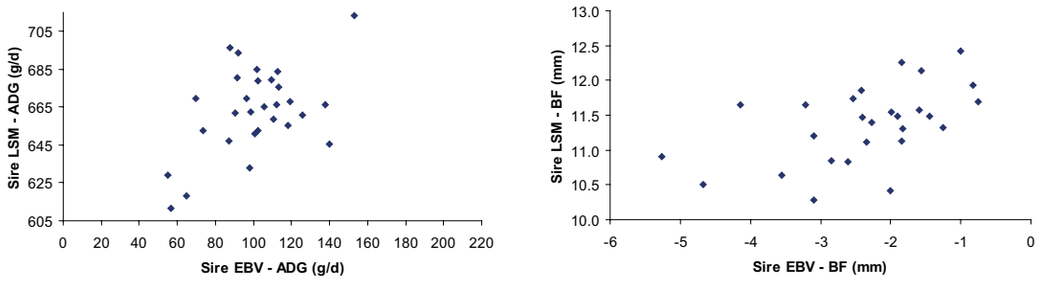


Figure 1. *Scatterplot of the relationship between individual sire estimated breeding values (EBVs) and sire least square means (LSM) of progeny for average daily gain (ADG) and back fat (BF).*

The regression coefficients were 0.42 ± 0.08 for ADG, which was not significantly different from the expected 0.5, and 0.31 ± 0.06 for BF. The result for ADG was similar to the study by MacBeth and McPhee (1999) who found a regression coefficient of sire EBVs of 0.44 ± 0.03 in Large White. Regression coefficients for BF were not reported. The positive relationship between sire EBVs and progeny performance is apparent in Figure 1 for both traits.

The regression coefficient was significantly lower than the expectation of 0.5 for BF, which may indicate that the heritability estimate currently used in the NPIP for BF may be too high. However, the result may also be due to sampling effects. Therefore, a simulation program (Crump, 2005) was used to evaluate the BF data. Results showed that there was a 70% chance of obtaining a regression coefficient between 0.4 and 0.6. In summary, the results demonstrate that across-herd EBVs of sires do predict differences in average progeny performance.

This work was supported by the APL and made possible with the contribution from Mark Bauer and Gatton College. *AGBU is a joint venture of NSW Department of Primary Industries and the University of New England.

References

CRUMP, R. E. and HERMESCH, S. (2003). In 'Pig Genetics Workshop Notes' (Animal Genetics and Breeding Unit: Armidale).

CRUMP, R.E. (2005). In 'Manipulating Pig Production X', p. 101, ed J.E. Paterson (Australasian Pig Science Association: Werribee).

MACBETH, G.M. and MCPHEE, C.P. (1999). In 'Manipulating Pig Production VII', p. 95, ed P.D. Cranwell. (Australasian Pig Science Association: Werribee).

Australian Pork

Simulation of estimated breeding value demonstration trials

R.E. Crump

Animal Genetics and Breeding Unit*, Armidale, NSW 2350.

On-farm demonstrations could promote the value of using genetic information, such as estimated breeding values (EBVs), for making decisions about which pigs to select or buy as breeding stock. However, to be useful, such demonstrations require valid statistical comparisons to be made between the offspring of different boars. Unfortunately, even when the basic design of such trials is adequate, the outcomes do not always demonstrate the value of EBVs as desired, because of uncertainty associated with the boar EBVs and the mean performance of their offspring. To overcome such problems, simulation can be used at the design stage or as an explanatory tool alongside the trial analysis. The aim of this study was to simulate an EBV demonstration trial using a real-life example.

True breeding values (TBVs) were simulated for boars and selection traits based on the EBV for each trait and its accuracy. Sire-family means for boars were simulated based on half the current TBV and knowledge of the genetic, common litter and environmental variances. The regression of sire-family mean on boar EBV was performed. The expected value of the regression coefficient is one half, and summarising the simulated values across many replicates allows inferences to be made about the outcome of a real EBV trial. Input parameters are given in Table 1.

Table 1. Boar EBVs and accuracies for back fat and average daily gain plus the number of dams they were mated to and number of recorded offspring generated in the trial.

Boar	Back fat		Average daily gain		No. of Dams	No. of Offspring
	EBV (mm)	Accuracy	EBV (g/day)	Accuracy		
A	-5.8	0.91	44	0.80	4	36
B	-4.2	0.97	56	0.92	3	22
C	-1.5	0.95	101	0.87	5	39

For back fat (BF) there was a 40% chance of getting a regression coefficient in the range 0.4-0.6 (Figure 1). For average daily gain (ADG) this was only 22% and there was an 8% chance of the trial showing that progeny ADG performance decreased with increasing ADG EBV.

Figure 2 shows the theoretical distribution of the TBVs of the boars for BF and ADG. As a result of the lower heritability of ADG (0.18 *vs.* 0.52 for BF) and proximity of the ADG EBVs to one another, the chance that the ranking of the boars on TBV would differ from their EBV ranking was quite high for this trait. Simulation can therefore be used as an effective tool in both the design and interpretation of EBV demonstration trials.

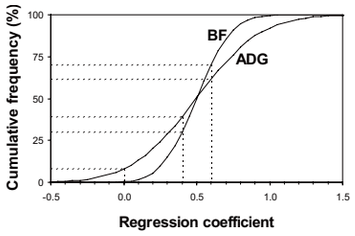


Figure 1. *Simulated cumulative frequencies of boar EBV regression coefficients for back fat (BF) and average daily gain (ADG).*

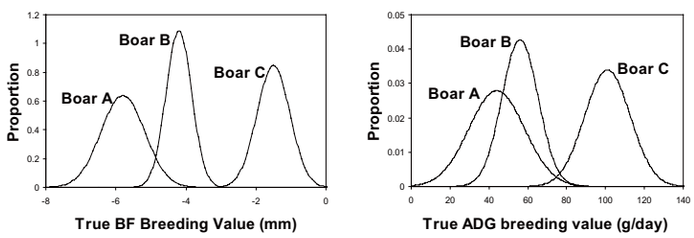


Figure 2. *Distributions of true breeding values of example boars for back fat (BF) and average daily gain (ADG).*

This work was funded by Australian Pork Limited. NSW Department of Primary Industries provided the example. *AGBU is a joint venture of NSW Department of Primary Industries and the University of New England.

Estimated breeding values for number born alive in pigs - do they really work?

M. Suárez*, J.A. Braun**, S. Nakavisut* and S. Hermesch*

*†Animal Genetics and Breeding Unit, Armidale, NSW 2351. **Myora Farm, Mount Gambier, SA 5290.

Estimated breeding values (EBVs) have been used in pig genetic improvement programs for several years. Reproductive traits, such as number born alive (NBA), have only low to moderate heritability (*et al.,* 2005). Nevertheless, using EBVs, selection for NBA in maternal lines at the seedstock level has resulted in genetic improvement at the purebred level, which should flow to the commercial level in the next generation. Progeny receive half their genes from their dam so EBVs for NBA of purebred dams should predict half the average differences in performance for litter size of their daughters. Rather than using EBVs, some producers instead rely on the phenotypic performance of the dam as a predictor of genetic merit for litter size. The aim of this paper was to investigate whether EBVs for NBA of dams at the purebred level predict performance of crossbred daughters at the commercial level. In addition, the ability of EBVs and phenotypic performance of dams to predict NBA was compared.

Performance records (n = 10316) from 2552 crossbred (F1) sows at two commercial piggeries were collected between June 1995 and December 2004 for the trait NBA. Estimated breeding values for NBA of their purebred Landrace (LR) and Large White (LW) dams (n = 1689) from MYORA Farm's maternal lines were estimated using PIGBLUP (Crump, 2003). Due to multiple-sire mating, the EBVs of the sires were not available for these F1 sows. An average performance for NBA was estimated for the purebred dams at MYORA Farm, including all the litters available between parities one and 10 (5052 for LW and 3089 for LR) and all the records within a certain range (2-22 NBA). The statistical analysis was performed using a GLM procedure in SAS (SAS Institute Inc, 1989). Each dam breed was analysed separately. The performance records of the F1 sows were corrected for farrowing season, parity and farrowing day of the week; all of these classes being nested within piggery. Corrected records for F1 sows were then regressed on the dams' EBV for NBA as well as dams' average phenotypic performance, in separate models.

The regression coefficient for NBA of F1 sows on the EBV for NBA of their dams was 0.44 (0.06) in LW dams and 0.45 (0.12) in LR dams (Table 1). These values are not significantly different from 0.5 ($P<0.05$) and clearly demonstrate that about half of what the EBV for NBA predicted at the purebred dam level was expressed by their daughters' performance, as expected. In contrast, the regression coefficient of the F1 sows NBA on their dams' average performance was 0.09 (0.02) for LW dams and 0.15 (0.04) for LR dams, indicating a poor prediction of daughters' performance for NBA based on dam phenotype for this trait.

Table 1. Dam breed, number and litters; F1 daughters' numbers and litters; regression coefficients (b) for daughters' performance on the EBV for number born alive, and on the phenotypic performance of the dam (with standard errors; SE).

Dam breed	N of dams/litters	N of daughters/litters	b EBV (SE)	b Phen. (SE)
Large White	1,065 / 5,052	1,644 / 7,039	0.44 (0.06)	0.09 (0.02)
Landrace	624 / 3,089	908 / 3,277	0.45 (0.12)	0.15 (0.04)

EBVs of purebred dams for number born alive estimated using PIGBLUP (Crump, 2003) at the seedstock level predicted half of the average performance of their daughters at the commercial level, as expected. In contrast, the dams' average litter size recorded at the seedstock level was a poor predictor of the performance of their daughters, which is a direct consequence of the low to moderate heritability of this trait. Estimated breeding values for NBA follow the expectations and are the best option to enhance selection decisions at seedstock level to predict litter size of crossbred sows at commercial piggeries.

†AGBU is a joint venture between NSW Department of Primary Industries and the University of New England. This work was funded by MYORA Farm, Mount Gambier, SA.

References

CRUMP, R. E. (2003). 'PIGBLUP Version 5.1 User Manual', (Animal Genetics and Breeding Unit: Armidale).

SAS INSTITUTE INC. (1989) SAS/STAT User's Guide, Version 6, Fourth Edition, Volume 2, Cary, NC: SAS Institute Inc.

SUAREZ, M., BRAUN, J.A., HERMESCH, S. and GRASER, H.-U. (2005). In 'P*roceedings of the 16th Conference of the Association for Advancement of Animal Breeding and Genetics*', Noosa, Australia. (In Press).

4 Gene markers and feed evaluation

Gene markers and how they can be used in selection

C. Moran

Centre for Advanced Technologies in Animal Genetics and Reproduction (Reprogen), University of Sydney NSW 2006.

Introduction

In this review I will discuss the current status of porcine genomic studies, beginning with a review of progress in porcine quantitative trait locus (QTL) mapping. If genetic markers are to assist selection programs, we must first identify the genetic regions affecting expression of economically important traits, before we can hope to use markers to identify animals carrying favourable genetic variants within these regions. Next I will describe the frustratingly slow progress towards a complete, publicly available genome sequence for the pig. A complete genome sequence is vital for several reasons. First it will greatly assist in identifying the genes and their variants underlying, and responsible for, the QTL discovered in mapping studies. Such genes and their mutations provide the best possible tools for marker assisted selection. The genome sequence will also provide a rich resource of novel genetic markers, ready for the emerging generation of genotyping systems. Single nucleotide polymorphisms (SNP) are likely to predominate in the future replacing, or at least supplementing, micro-satellite markers. To fully exploit such markers, we require hundreds of thousands of SNP for implementation in large scale automated genotyping systems. Next I will describe an example of a successful European gene identification project which has proceeded all the way from broadly mapped QTL in the pig, through positional candidate gene identification to identification of the mutation - the so-called quantitative trait nucleotide (QTN) - responsible for a substantial effect on growth and muscle deposition. This case illustrates that in some cases, not only is there light at the end of the tunnel, but that we have emerged at the other end with the ideal type of genetic marker in hand ready for implementation in selection. Perhaps just as importantly, we have gained an expanded understanding of the fundamental biological processes involved in growth. Finally I will briefly describe some recent work in my laboratory that has eliminated a candidate gene for boar taint related to androstenone.

Progress in QTL mapping

Bidanel and Rothschild (2002) reviewed the first eight years of progress in porcine QTL mapping, which began with the first ever reported domestic animal QTL - a fatness QTL on pig chromosome 4 (Andersson *et al.,* 1994). Rothschild's laboratory has subsequently developed a very useful electronic tool, PigQTLdb (Lu *et al.,* 2005), which is a database of published porcine QTLs (http://animalgenome.org/QTLdb/). As of 2004, the database reported 791 QTLs for 291 different traits from 73 published studies. The database reports results from 1995 (when only five QTL were listed) onwards. Publication of QTL peaked in 2001 with 250 QTL but has subsequently declined dramatically to only 42 in 2004.

Interpreting trends in the rate of QTL discovery (Figure 1) requires some caution since there are potential biases in reporting findings of unknown relative importance and given the large scale and complexity of QTL studies, considerable delays occur in their publication. Furthermore, many QTL studies are not reported due to conditions of confidentiality imposed by funding agencies or companies protecting potential IP positions. Thus, the figures displayed in Figure 1 substantially under report the overall QTL research effort. Conversely, the QTL are often reported for highly correlated traits that, not unexpectedly, map to the same position. For example, most studies report several and sometimes many alternative measures of back fat at different anatomical positions, ages, body weights and/or using different measuring devices with the results presented as separate QTL discoveries. It is therefore not surprising that QTLs for back fat are the most commonly reported (Table 1), especially when the different versions of this trait are taken into account. Most of the variation in these many versions of the back fat trait is almost certainly attributable to the same underlying genes and thus there is considerable over reporting of these QTL. False discovery is also a potential problem, especially as most of the QTL reported do not meet the most stringent statistical standards, but failure to detect QTL due to underpowered experiments is also a serious problem.

Regardless, the severe decline in the rate of publication of QTL discoveries is cause for concern. Has there been a worldwide decline in funding for porcine QTL studies? Are more laboratories going down the route of commercial confidentiality and keeping their results in-house? Has the scientific progress in QTL discovery outstripped the technological potential for implementation in practical animal breeding programs? These are important questions and the answers are not obvious.

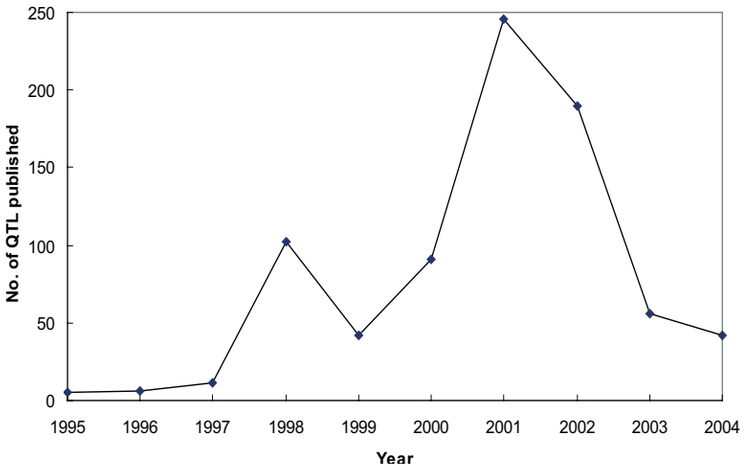


Figure 1. *Time trends in reporting porcine QTL discoveries.*

Choosing appropriate traits for QTL mapping

Another important concern regarding the application of the outcomes of porcine QTL studies in genetic improvement programs relates to the traits for which QTL mapping are being performed. As shown in Table 1, easily measured traits like back fatness predominate the QTL database with back fatness alone accounting for at least 68 or 8.6% of the reported QTLs. It has long been known that marker–assisted selection (MAS) has its greatest potential benefit for traits that are difficult to measure and which usually are of low heritability. For traits that are easy to measure and highly heritable, such as back fat, conventional performance-based selection is cheap and efficient to apply and very little additional benefit would flow from using genetic markers. A cost benefit analysis of the implementation of MAS would clearly be unfavourable, as Hayes and Goddard (2003; 2004) clearly demonstrated in realistic simulations of the application of markers. Of course, there are still considerable research benefits to be obtained from QTL studies in these cases, as they will contribute to the long-term improvement of our understanding and potentiate manipulation of important biological process like growth and fat deposition in other ways.

Table 1. Categories of traits for which QTL have most frequently been reported in the pig (from PigQTLdb)

Number of QTL reported	Trait
40	Back fat (average) thickness
24	Loin eye area
23	Color longissimus dorsi
17	Body weight at birth
17	Back fat thickness at last rib
16	pH 24 hours post mortem (loin)
16	Intramuscular fat percentage
15	Carcass length
14	Number of nipples
11	Back fat at last lumbar vertebra
11	Ovulation rate

What type of traits should be incorporated into QTL studies? Traits that are difficult, expensive or impossible to measure on candidates for selection or their relatives have the most potential to benefit from MAS. Post-slaughter traits such as loin eye area, meat colour, muscle pH 24 hours post mortem and intramuscular fat percentage fit these criteria. Reproductive traits like ovulation rate are also potential candidates for MAS, as are efficiency traits like food conversion ratio. Selection for disease resistance using genetic markers is an ideal application of the technology, since once resistance encoding genomic regions are recognised there would be no further need for exposure of animals to pathogens. Ideally the traits for which QTL should be sought are those traits that are not normally measured or selected upon in commercial breeding programs due to expense, difficulty or risk. If QTL can be recognised for these traits, then substantial benefits could accrue from using markers to assist in the multi-trait genetic improvement program.

Genomic distribution of QTLs

There are currently insufficient reported QTLs to address the question of the uniformity or otherwise of their chromosomal distribution from a meaningful biological perspective. However it is clear from Figure 2 that some chromosomes, including SSC4 and 7 are over represented at this stage, whereas some other chromosomes including SSC3, 5 and 13 are under represented. The first ever QTL for any domestic animal were reported on chromosome 4 by Andersson *et al.* (1994). The majority of the large number of reported QTL subsequently reported on this chromosome also relate to growth and fatness, perhaps reflecting an emphasis on analysing this chromosome to confirm the original observation and /or the relative ease of measurement of growth and fatness traits.

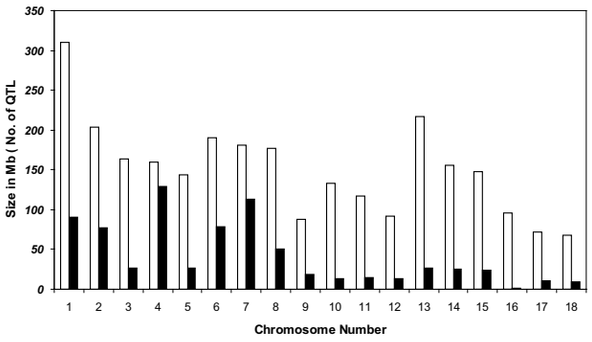


Figure 2. *Distribution of QTL across autosomes in relation to the size of the autosomes in megabase pairs. White bars represent the size of chromosome in megabase pairs; black bars represent the number of QTL mapped to that chromosome.*

Towards a porcine genome sequence

Progress towards completing a full pig genome sequence that is publicly available has been frustratingly slow. A privately funded consortium has already made some progress towards a partial and rudimentary coverage of the porcine genome and has recently made the raw data publicly available, as described below. Unfortunately, the publicly funded effort driven by biomedical interest in the pig as a model organism has not eventuated. Fortunately, the United States Department of Agriculture has provided funding for a fall back position, which together with contributions from international laboratories should provide a modest genome coverage within about two years.

The Danish-Chinese collaboration

The Royal Veterinary and Agricultural University, the Danish Institute of Agricultural Sciences and the National Committee of Pig Breeding, Health and Production from Denmark and the Chinese Academy of Science and especially the Beijing Genomics Institute in China have been collaborating since 2001 on a major genome and expressed sequence tag (EST) sequencing project. This project explicitly aims to apply for, hold and exploit patents on sequences or partial sequences of genes and to transfer useful research results and technology into the Chinese and Danish pig industries. Although all sequences were intended for public release after six months delay, it was not until early 2005 that the results were published and the raw, unannotated data released (Wernesson *et al.,* 2005). The 2.083 x 10^9 bp sequence produced from Hampshire, Yorkshire, Landrace, Duroc and Erhualian pigs is equivalent to 0.66X genome coverage and represents hits on about 48% of the sequences in the porcine genome. In summary, the porcine genome sequence is incomplete and in its current unannotated form is of limited use. Nevertheless, it represents an important and substantial advance.

Public consortium sequencing project

Most publicly funded genome sequencing efforts have been co-ordinated via the United States National Institutes of Health (NIH). At the 2004 International Society of Animal Genetics (ISAG) meeting held in Tokyo, progress and problems with the public pig genome sequencing effort were discussed. Unfortunately, for reasons that are unclear, the pig appears to have fallen off the queue for the program of genome sequencing supported by the NIH/ National Human Genome Research Institute, despite previous assurances that it would join the next round after the bovine sequence. This is particularly disappointing given that the unannotated but complete bovine genome sequence was released publicly on October 7 2004. It is also very puzzling when one considers that the pig is one of the main sources of animal protein consumed worldwide, is an excellent and widely used biomedical model species, is likely to become of even greater biomedical significance if xenotransplantation becomes established as a medical therapy and has all the necessary resources in place for completion of a genome sequence including very detailed genetic maps and

BAC contigs. Instead, in August 2004 the NIH announced that it had committed substantial resources to sequencing a diverse array of other mammalian species, including the African savannah elephant (*Loxodonta africana*), the European common shrew (*Sorex araneus*), the European hedgehog (*Erinaceus europeaus*), the guinea pig (*Cavia porcellus*), the lesser hedgehog tenrec (*Echinops telfairi*), the nine-banded armadillo (*Dasypus novemcinctus*), the rabbit (*Oryctolagus cuniculus*), the domestic cat (*Felis catus*), since it is an important medical model for studying disease, and the orangutan (*Pongo pygmaeus*) as another primate closely related to humans. At this stage, the human, mouse, rat, chicken, dog and bovine genomes have been sequenced and the tammar wallaby (*Macropus eugenii*), the grey short-tailed opossum (*Monodelphis domestica*) and even the platypus (*Ornithorhynchus anatinus)* are in the pipeline. Even obscure vertebrate species like the pufferfish (*Tetraodon nigroviridis*) have a completed sequence with the lamprey (*Petromyzon marinus*), skate (*Raja erinacei*) and three-spined stickleback (*Gasterosteus aculeatus*) being readied for sequencing. While there are excellent biological reasons for sequencing all of these genomes based on insights to be derived from comparative analysis of the sequences, it still seems to verge on the bizarre that the pig has been removed from the list, given its economic importance and its indisputable biomedical relevance.

In March 2005, NIH released its next list of 12 new species prioritised for genome sequencing. Only one mammal, the marmoset (*Callithrix jacchus*), an even more distantly related primate to humans than those already sequenced or in the queue, is included with the remaining eleven species being chosen on the standard criteria of their 'potential to fill crucial gaps in biomedical knowledge'. Puzzlingly the list includes the pea aphid (*Acyrthosiphon pisum*), since it is 'an insect which causes hundreds of millions of dollars of crop damage each year' (NIH, 2005). A medical angle was contrived in this case by stating that 'Understanding this resistance [to insecticides by the pea aphid] at a molecular level can lead to safer and more effective pesticides and *improve human nutrition*.'

It is believed that the NIH may have formed the opinion that alternative sources of funding would become available for generating a porcine genome sequence and for this reason dropped it from its priority list, despite supporting the bovine genome sequence in the previous round.

Imminent prospects for porcine genome sequencing

On March 29 2005, the Co-operative State Research, Education and Extension Service of the USDA (CREES) announced a National Research Initiative on Porcine Genome Sequencing (http://www.csrees.usda.gov/fo/fundview.cfm?fonum=1380), with the aim of producing a draft sequence (3x coverage) of 90 percent of the porcine genome as a contribution to the international Swine Genome Sequencing Consortium. Funding of US$5million is provided for 2005 and an additional $5 million will be provided in the second year. Grants from this fund are available only to US institutions and individuals. However given the stated intent of draft assembly of the sequence and deposition of all information into a publicly accessible, pre-existing database, this program will clearly benefit porcine geneticists worldwide.

The international Swine Genome Sequencing Consortium has other sources of support than CREES. The Agricultural Research Service (ARS, another arm of the USDA has also committed several million and the Institute for Pig Genetics (Netherlands), INRA (France), Iowa Pork Board, Iowa State University, the National Livestock Research Institute (South Korea), the National Pork Board (USA), North Carolina Pork Board, North Carolina State University, the Roslin Institute (UK), the Sino-Danish Consortium, University of Illinois, and the Wellcome Trust Sanger Institute have already produced sequences or have pledged significant support to the pig genome sequencing project. It may be realistic to expect that, by late 2006, at least the early draft sequences will be publicly available.

From quantitative trait locus to quantitative trait nucleotide

Ideally all QTL mappers would like to identify the mutation(s) in a specific gene underlying the QTL effects they have identified. This would not only provide the best possible genetic test for exploiting the favourable allele in marker assisted selection but would also illuminate the biology underlying the biological effect, since the gene could then be related to a metabolic pathway or signalling system that might be amenable to alternative forms of manipulation to improve productivity. Many examples of successful molecular discoveries in domestic animals are now available, initially for genes of large effect such as the double muscling phenotype in cattle (Grobert *et al.,* 1997), the Booroola fecundity gene in sheep (Galloway *et al.,* 2000) and the RN gene for excess glycogen content causing acidity in processed pork (Milan *et al.,* 2000) but extending more recently to genes originally discovered in QTL scans such as the DGAT1 locus affecting fat content of milk in dairy cattle (Grisart *et al.,* 2002).

Discovery of a QTL for muscle growth on pig chromosome 2

Back-to-back papers in *Nature Genetics* by Jeon *et al.* (1999) and Nezer *et al.* (1999) reported the independent identification in Sweden and Belgium of a QTL mapping to chromosome 2 with a substantial effect on muscle and fat deposition in the pig. It explained 15–30% of the phenotypic variation in muscle mass and 10–20% of the variation in back fat thickness in the resource pedigrees in which it was mapped. The QTL was discovered in routine genome scans for QTLs for numerous traits being performed in many laboratories throughout the world at that time.

Imprinting a clue to QTL identity

However, the QTL mapping to chromosome 2 was unusual from the start since the effect of the QTL alleles depended on whether they were inherited from the mother or the father. In this case, only the paternally inherited allele is expressed, with the maternally inherited allele imprinted to repress its expression. Suspicion immediately fell on the insulin-like growth factor 2 (IGF2) locus, since comparative mapping indicated it lay in the relevant region of porcine chromosome 2. IGF2, which is widely expressed prenatally, was known to be similarly imprinted in humans, mice and pigs. On *a priori* biochemical grounds, IGF2 might be expected to have an effect on muscle growth since it is known to mediate the effect of growth hormone and stimulate the action of insulin and is known to regulate growth. Amarger *et al.* (2002) carried out a detailed comparative analysis of the structure and properties of the IGF2 and adjacent loci in the pig in the lead up to the identification of the mutation.

Subsequent mapping refined the position of the QTL to an interval of 250 kb at the tip of chromosome 2, containing the IGF2 locus (Nezer *et al.,* 2003). An intensive analysis of mutations in IGF2 and surrounding loci commenced. Animals were available with or without the growth-enhancing QTL allele, but there were many mutations within IGF2 and the surrounding genes to sift through. The objective was to find in these animals a DNA sequence variant that matched exactly the inheritance of the growth enhancing QTL allele – this was termed 'mapping by haplotype sharing'. Van Laere *et al.* (2003) finally reported the full characterisation of the mutation in IGF2 responsible for the QTL effect in late 2003. This was extremely important and useful from a practical sense as it means that an accurate and direct test is now available for the favourable allele at this locus.

Intronic mutation responsible for growth enhancing QTL effect

However at the scientific level, it revealed something profoundly important as well. The mutation responsible for the effect did not lie in a protein encoding region of the gene as one might expect, nor did it lie in the 5" upstream regulatory region of the gene, where one might also expect an effect of transcription of the gene to influence the growth phenotype. Instead it was actually found to lie within intron 3 of IGF2. An intronic location had been deemed so unlikely that in fact the causative mutation was ignored for some time after it was first discovered and was not given serious consideration until the genetic evidence from analysis of haplotype sharing compelled it, having eliminated all other DNA sequence variants. It was also shown that the mutation prevents the binding of a protein factor believed to be a repressor of transcription. Pigs inheriting the mutation from their sire have a threefold increase in IGF2 messenger RNA expression in postnatal muscle, but not in other tissues, and this is believed responsible for the enhanced growth effect.

From the perspective of pig QTL mappers, the outcome of these studies provides confirmation that we will eventually discover the genes affecting economically important traits in pigs. In the case of this QTL, the imprinted nature of the effect greatly assisted in homing in on a very good candidate locus. However it also illustrates that the biology of growth is complex and still poorly understood and that studies such as these will also discover new biological principles and new opportunities for interventions in manipulating growth and other characteristics.

Eliminating a candidate gene for a boar taint QTL

Boar taint is the undesirable off-odour and off-flavour emanating from cooked meat of the male pig (Brooks and Pearson, 1986). Androstenone (5α-androst-16-en-3-one), a steroid pheromone related to and synthesised in parallel with steroid hormones (Claus, 1979) is a major cause. Since castration of male pigs is not routinely practiced in Australia, boar taint due to accumulation in fat tissues of this sex pheromone is a potentially serious problem.

Bidanel et al. (1996) and Milan *et al.* (1998), using a Meishan x Large White F2 mapping resource, reported that a QTL for androstenone levels in entire males mapped to pig chromosome 7 in the approximate vicinity of the major histo-compatibility complex (MHC). The biosynthesis of steroid hormones and pheromones is extremely complex with numerous enzymes involved and potential products. However one enzyme involved in this process, steroid 21 hydroxylase P450c21 (CYP21), was noted as a positional candidate, since it also mapped to the MHC in the pig (Geffrotin *et al.*, 1990; Chardon and Renard, 1999) as it also does in other species. The *CYP21* gene is about 3.36 kb in length and contains 9 introns and 10 exons (Burghelle-Mayeur *et al.,* 1992)

Consequently several laboratories were keen to test CYP21 as a candidate and Mr Payam Arasta took this up as a PhD project in my laboratory. We used the US43 pedigree bred at Bunge (QAF) Meat Industries at Corowa as our mapping and candidate evaluation resource. This resource consists of 596 progeny in eight sire families with a total of 130 dams. Three hundred and twenty six of the progeny had androstenone measurements. Two sire families had already provided significant evidence of heterozygosity for a QTL for androstenone levels on SSC7 (Kerr *et al.,* 2001) in the vicinity of the MHC and in a similar position to the QTL reported by the French INRA laboratory. PCR primers were designed to amplify the CYP21 gene in four overlapping fragments of 1051 bp from position 458 to 1509, 914 bp from position 1344 to 2285, 940 bp from position 2164 to 3104 and 875 bp from position 2834 to 3709. These were then amalgamated. PCR products were amplified from four of the eight sires and analysed for the presence of single nucleotide polymorphisms (SNPs). Thirty six SNPs were identified in the four sires but all were either synonymous (ie did not change the amino acid sequence of the CYP21 protein) or were located in introns. There was some evidence from an analysis of potential transcripts using Genscan (Burge and Karlin, 1997) that the G allele at an intronic A/G SNP at position 2329 might favour an alternative transcript causing an in-frame insertion of 105 bp in the messenger RNA and an additional 35 amino acids in the protein. A TauI PCR-RFLP (restriction fragment length polymorphism) for this 2329A/G SNP was devised and all progeny and parents of the US43 resource were genotyped. Variance analysis of the 2329 G/A genotypes showed that segregation of the SNP in these sire families had no significant impact ($p=0.75$) on androstenone levels. Finally an RT-PCR assay designed to detect the alternative transcript was devised. This was applied to RNA purified from testes of boars from QAF Meat Industries of A/G genotype, but provided no evidence of the use of the alternative transcript.

We eventually concluded from this exhaustive sequencing analysis of four sires, including examination of an alternative transcript as another potential source of variation in CYP21 structure and activity, that we could confidently exclude CYP21 as the gene underlying the SSC7 androstenone QTL. Quintanolla el al. (2003) have similarly excluded CYP21 after sequencing the locus in two founder animals in their resource pedigree, one Meishan and one Large White, after finding no SNPs that caused amino acid substitutions. Therefore the search for the gene and the mutations underlying this QTL must continue in other genes in this region of SSC7. A complete porcine genome sequence will be invaluable in this and all other QTL identification tasks.

Conclusions

Numerous QTLs, many with potential for application in marker assisted selection, have been reported for the pig and undoubtedly many more have been discovered but not reported. However the apparent decline in the rate of porcine QTL discovery is cause for concern. International efforts to complete the porcine genome sequence and make it publicly available are continuing and the United States Department of Agriculture has recently made a substantial financial commitment to the completion of this task. Hopefully the availability of this sequence will revitalise porcine genomic studies and assist in drilling down to the genes underlying the already discovered QTL. Despite the current delays, I anticipate that the genome sequence will be more or less complete by the end of 2006, although full annotation of the sequence may take longer. The genome sequence will have a major impact on the way that porcine genomics and genetic studies are performed and will lead to accelerated discovery of genes and mutations that have economically important effects. The path from QTL to QTN has already been travelled for the IGF2 effect on porcine muscle growth. This illustrates the immediate and obvious short-term benefits, namely an accurate genetic test, and the potential longer terms benefits from fundamental biological discoveries. In the case of the boar taint QTL on SSC7, we have been able to exclude an obvious candidate locus and the task of going from QTL to QTN still lies ahead of us.

References

AMARGER, V., NGUYEN, M., VAN LAERE, A.S., BRAUNSCHWEIG, M., NEZER, C,. GEORGES, M. and ANDERSSON, L. (2002). Comparative sequence analysis of the INS-IGF2-H19 gene cluster in pigs. *Mammalian Genome* **13**:388–398.

ANDERSSON, L., HALEY, C.S., ELLEGREN, H., KNOTT, S.A., JOHANSSON, M., ANDERSSON, K., ANDERSSON-EKLUND, L., EDFORS-LILJA, I., FREDHOLM, M., HANSSON, I., HAKANSSON, J. and LUNDSTRÖM, K. (1994). Genetic mapping of quantitative trait loci for growth and fatness in pigs. Science **263**:1771-1774.

BIDANEL, J.P. and ROTHSCHILD, M. (2002). Current status of quantitative trait locus mapping in pigs. Pig News & Information. **23**:39N-54N.

BIDANEL, J.P., MILAN, D., CHEVALET, C., WOLOSZYN, N. CARITEZ, J.C., GRUAND, J., LE ROY, P., BONNEAU, M. RENARD, C., VAIMAN, M., GELLIN, J. and OLLIVIER, L. (1997). Chromosome 7 mapping of a quantitative trait locus for fat androstenone level in Meishan X Large White F2 entire male pigs. In 'Boar taint in entire male pigs, EAAP publication No. 92', pp. 115- 118.

BROOKS, R. I. and PEARSON, A. M. (1986). Steroid hormone pathways in the pig, with special emphasis on boar odor: a review. *Journal of Animal Science* **62**:632-645.

BURGE, C. and KARLIN, S. (1997). Prediction of complete gene structures in human genomic DNA. *Journal of Molecular Biology* **268**:78 - 94.

BURGHELLE-MAYEUR, C., GEFFROTIN, C. and VAIMAN, M. (1992). Sequences of the swine 21-hydroxylase gene (CYP21) and a portion of the opposite-strand overlapping gene of unknown function previously described in human. Biochimica et Biophysica Acta-Bioenergetics **1171**:153–161.

CHARDON P., RENARD C. and VAIMAN, M. (1999). The major histocompatibility complex in swine. Immunological Review **167**: 179-192.

CLAUS, R. (1979). Boar taint as model of a steroidal pheremone. Acta Endocrinologica **91**:432-433.

GALLOWAY, S.M., MCNATTY, K.P., CAMBRIDGE, L.M., LAITINEN, M.P., JUENGEL, J.L., JOKIRANTA, T.S., MCLAREN, R.J, LUIRO, K., DODDS, K.G., MONTGOMERY, G.W., BEATTIE, A.E., DAVIS, G.H. and RITVOS, O. (2000). Mutations in an oocyte-derived growth factor gene (BMP15) cause increased ovulation rate and infertility in a dosage-sensitive manner. *Nature Genetics* **25**:279-283.

GEFFROTIN, C., CHARDON, P., DE ANDRES-CARA, D.F., FEIL, R., RENARD, C. and VAIMAN, M. (1990). The swine steroid 21-hydroxylase gene (CYP21): cloning and mapping within the swine leucocyte antigen complex. *Animal Genetics* **21**:1-13.

GRISART, B., COPPIETERS, W., FARNIR, F., KARIM, L., FORD, C., BERZI, P., CAMBISANO, N., MNI, M., REID, S., SIMON, P., SPELMAN, R., GEORGES, M. and SNELL, R. (2002). Positional candidate cloning of a QTL in dairy cattle: identification of a missense mutation in the bovine DGAT1 gene with major effect on milk yield and composition. *Genome Research* **12**: 222-231.

GROBET, L., MARTIN, L.J., PONCELET, D., PIROTTIN, D,. BROUWERS, B., RIQUET, J,. SCHOEBERLEIN, A., DUNNER, S., MENISSIER, F., MASSABANDA, J., FRIES, R., HANSET, R. and GEORGES, M. (1997). A deletion in the bovine myostatin gene causes the double-muscled phenotype in cattle. *Nature Genetics* **17**: 71-74.

HAYES, B. J. and GODDARD, M. E. (2003). Evaluation of marker assisted selection in pig enterprises. *Livestock Production Science* **78**:198-211.

HAYES, B. J. and GODDARD, M. E. (2004). Break-even cost of genotyping genetic mutations affecting economic traits in Australian pig enterprises. Livestock Production Science **89**:235-242

HU, Z-L, DRACHEVA, S, JANG,W, MAGLOTT, D, BASTIAANSEN, J, REECY, J and ROTHSCHILD M. (2005). A Quantitative Trait Loci Resource and Comparison Tool for Pigs: PigQTLDB. In 'Plant and Animal Genome XIII', p.839, San Diego.

JEON, J.-T, CARLBORG, O., TORNSTEN, A., GIUFFRA, E., AMARGER, V., CHARDON, P., ANDERSSON-EKLUND, L., ANDERSSON, K., HANSSON, I., LUNDSTROM, K. and ANDERSSON, L. (1999). A paternally expressed QTL affecting skeletal and cardiac muscle mass in pigs maps to the IGF2 locus. *Nature Genetics* **21**:157–158.

KERR, R., CHEN, Y., MORAN, C., GRASER, H. and LUXFORD, B. (2001). Marker assisted selection for profitable pigs. Australian Pork Limited Confidential Research Report. Project Number: US43. 224 pp.

MILAN, D., BIDANEL, J.P., LE ROY, C., CHEVALET, P. WOLOSZYN, N., CARITEZ, J.C., GRUAND, J., LAGANT, H., BONNEAU, M., LEFAUCHEUR, L., RENARD, C., VAIMAN, M., MORMEDE, P., DESAUTES, C., AMIGUES, Y., BOURGEOIS, F., GELLIN, J and OLLIVIER L. (1998). Current status of QTL detection in Large White x Meishan crosses in France. Proceedings of the 6th World Congress on Genetics Applied to Livestock Production. **26**:414-417.

MILAN, D., JEON J.T., LOOFT, C., AMARGER, V., ROBIC, A., THELANDER, M., ROGEL-GAILLARD, C., PAUL, S., IANNUCCELLI, N., RASK, L., RONNE, H., LUNDSTROM, K., REINSCH, N., GELLIN, J., KALM, E., ROY, P.L., CHARDON, P. and ANDERSSON. L. (2000). A mutation in PRKAG3 associated with excess glycogen content in pig skeletal muscle. *Science* **288**:1248-1251.

NEZER, C., MOREAU, L., BROUWERS, B., COPPIETERS, W., DETILLEUX, J., HANSET, R., KARIM, L., KVASZ, A, LEROY, P. and GEORGES M. (1999). *Nature Genetics* **21**:155–156.

NEZER, C., COLLETTE, C., MOREAU, L., BROUWERS, B., KIM, J.J., GIUFFRA, E., BUYS, N., ANDERSSON, L. and GEORGES, M. (2003). *Genetics* **165**:277–285.

NIH/NATIONAL HUMAN GENOME RESEARCH INSTITUTE (2004). NHGRI adds 18 organisms to sequencing pipeline. NIH News Release August 5 2004. http://www.genome.gov/12511858.

NIH/NATIONAL HUMAN GENOME RESEARCH INSTITUTE (2005). NHGRI Targets 12 More Organisms for Genome Sequencing. NIH News Release March 1 2005 http://www.genome.gov/13014443.

QUINTANILLA, R., DEMEURE, O., BIDANEL, J.P., MILAN, D., IANNUCCELLI, N., AMIGUES, Y., GRUAND, J., RENARD, C., CHEVALET, C. and BONNEAU, M. (2003). Detection of quantitative trait loci for fat androstenone levels in pigs. *Journal of Animal Science* **81**:385-394.

VAN LAERE, A-S., NGUYEN, M., BRAUNSCHWEIG, M., NEZER, C., COLLETTE, C., MOREAU, L., ARCHIBALD, A.L., HALEY, C.S., BUYS, N., TALLY, M., ANDERSSON, G., GEORGES, M. and ANDERSSON, L. (2003). A regulatory mutation in IGF2 causes a major QTL effect on muscle growth in the pig. *Nature* **425**:832-836.

WERNERSSON, R., SCHIERUP, M.H., JØRGENSEN, F.G., GORODKIN, J., PANITZ, F., STÆRFELDT, H-H., CHRISTENSEN, O.F., MAILUND, T., HORNSHØJ, H., KLEIN, A., WANG, J., LIU, B., HU, S., DONG, W., LI, W., WONG, G.K.S., YU, J., WANG, J., BENDIXEN, C., FREDHOLM, M., BRUNAK, S., YANG, H. and BOLUND, L. (2005). Pigs in sequence space: A 0.66X coverage pig genome survey based on shotgun sequencing. BMC Genomics 6: 70. (http://www.biomedcentral.com/1471-2164/6/70).

Searching for single nucleotide polymorphisms in candidate genes for fertility

J. Aldenhoven*, A. Spötter** and O. Distl**

*University of Sydney, NSW 2006. **University of Veterinary Medicine, Hannover, Germany.

In this project we developed a list of candidate genes for fertility-related traits, such as embryonic survival and number of piglets, based on their known function and relationship to fertility established previously in human and mouse studies. The aim was to discover single nucleotide polymorphisms (SNPs) within the candidate genes to determine whether they could have an effect on fertility and potential application in marker assisted selection (MAS).

Porcine specific primers were developed to amplify sections of the candidate genes using either Expeditor (Public Release, v1.0, http://www.genome.iastate.edu/cgi-bin/expeditor/expeditor2), developed by Hu *et al.* (2004) or the first method used by Aldenhoven *et al.* (2003). A total of 29 primer pairs were designed in exons to amplify the more variable introns. The amplification reaction (15 µl final volume) was performed using 20 ng of porcine DNA, 10xPCR buffer, 100 µM each dNTP, 10 pmol primer, 5% DMSO and 1U Taq DNA polymerase (Qbiogene, Heidelberg, Germany). Thermo-cycling was carried out under the following conditions: Initial denaturation at 94˚C for 4 min followed by 35 cycles (94˚C for 30 s, 58 or 60˚C for 1 min, 72˚C for 30 s). Template DNA for sequencing was obtained from eight pig breeds (Meishan, Mangalitza, Duroc, Pietrain, German Landrace, Hampshire, Husum Red Pied and German Large White). Amplification products were purified using Montage PCR_{96} Cleanup kit (Millipore). Sequencing reactions were performed according to the manufacturer's instructions with DYEnamicTM ET Terminator Cycle Sequencing Kit, and resolved on a MegaBACE sequencer (Amersham Biosciences). Sequences were then analysed using Sequencher v 4.2 (Gene Codes Corporation).

SNPs were detected in 18 of the 29 amplicons, with details for nine of the genes/amplicons analysed presented in Table 1. The identified SNPs will be used in associated research in a German resource pedigree to test for significant additive and dominant gene effects on embryonic survival and number of piglets born alive. The discovery of a gene with a significant effect on fertility traits could enable MAS to be used within pig breeding programs in a similar way that the marker for the oestrogen receptor locus (Rothschild *et al.,* 1996), has been used to increase litter size in pigs in the US and Europe.

Table 1. SNP presence, absence and type and corresponding human exons for nine sequenced porcine genes.

Gene name	Size of amplicon	SNP (Y/N)	SNP type/ location in amplicon	Genbank accession No.	Corresponding human exons
AHCY	749	Y	A/G, 515	BP461530	4-5
ERBB2	591	N	-	AY117054	24-25
FN1	411	Y	C/T, 228 G/T, 241 C/T, 291	AY274117	44-45
HSPG2	573	N	-	BI467707	15-17
MAP3K3	694	Y	C/T, 67 C/T, 86	BE2331761	11-12
NCAM1	526	N	-	AW414520	8-9
PPARD	345	Y	C/T, 202 C insertion, 219	AY188501	7-8
RBP4	268	Y	A/G, 230	NM_214057	4
STC1	815	N	-	BI182587	2-3

J. Aldenhoven was supported by the Australian Federation of University Women-NSW Inc.

References

ALDENHOVEN, J., CHEN, Y. and MORAN, C., (2003). *Cytogenetic and Genome Research* **102**:128-132.

HU, Z-L., GLENN, K., RAMOS, A.M., OTIENO, C.J., REECY, J.M. and ROTHSCHILD, M.F. (2005). *Journal of Heredity* **96**:80-82

ROTHSCHILD, M., JACOBSON, C., VASKE, D., TUGGLE, C., WANG, L., SHORT, T., ECKARDT, G., SASKI, S., VINCENT, A., MCLAREN, D., SOUTHWWOD, O., VAN DER STEEN, H., MILEHAM, A., and PLASTOW, G. (1996). *Proceedings of the National Academy of Sciences of the United States of America* **93**:201-205.

Reliability of trial designs for a proof of estimated breeding values analysis

A.C. Hansson, R.E. Crump and S. Hermesch

Animal Genetics and Breeding Unit*, Armidale, NSW 2351.

Selection of genetically superior pigs as parents should lead to improved progeny performance. Trials can be used to demonstrate that selection of superior sires based on estimated breeding values (EBVs) will result in an increase in the average performance of progeny. The expected regression coefficient from the regression of mean progeny performance on sire EBV is 0.5, since sires pass half their genes to their progeny. The aim of this study was to assess various trial designs using the simulation program of Crump (2005) for back fat (BF) to illustrate key factors that influence the probability of obtaining a regression coefficient between 0.4 and 0.6 (0.4-0.6 probability).

Data from the National Pig Improvement Program (http://npip.une.edu.au) were used to provide realistic guidelines for the simulation inputs. For each boar, the simulation program (Crump, 2005) required BF EBV and EBV accuracy (ACC), as well as number of recorded progeny and litters. Each simulated design was replicated 10,000 times. The design that maximises the 0.4-0.6 probability should be selected for use in a trial.

Designs were considered with combinations of five and ten boars with high (95%) and low (75%) ACC. The change in number of sires was used to reflect designs when sire number was limiting. In addition, low ACC reflect young sires with possible genetic superiority to older sires with higher ACC. For simplicity, sire use was balanced and BF EBVs were evenly spread within a range of 4 mm or 2 mm. In addition, the number of total progeny and progeny per litter were varied. The 0.4-0.6 probability for each design is shown in Table 1.

Table 1. The probability of obtaining a regression coefficient of sire estimated breeding value (EBV) between 0.4 and 0.6 for back fat (BF) for varied progeny numbers, progeny per litter, ranges of BF EBVs (Range, mm), number of sires and EBV accuracies (ACC, %).

Design	A		B		C		D	
Total progeny (#)	480		960		960		960	
Progeny per litter (#)	8		8		6		8	
Range (mm)	4		4		4		2	
Sires (#):	5	10	5	10	5	10	5	10
ACC: 75%	41.9	46.5	43.6	50.9	44.8	53.0	22.9	26.8
ACC: 95%	58.1	58.3	67.2	70.2	68.7	72.0	37.4	38.4

For all designs, changing the number of sires from five to ten was more effective in increasing the 0.4-0.6 probability when ACC was low (Table 1). Increasing the total number of progeny recorded and the total number of litters in a trial was most effective when ACC was 95% (Design A versus Design B). When the number of progeny per litter was decreased from eight to six (and thus the number of litters was increased), the 0.4-0.6 probability was slightly increased. Therefore, this increase in probability indicates that it was better to record fewer progeny per litter from more litters, but the total number of progeny remains constant. This was due to a reduction in the impact of dam effect, both genetic and common environmental.

By decreasing the EBV range from 4 mm to 2 mm (Design D), the 0.4-0.6 probability was reduced dramatically. This decrease may be due to sire EBVs overlapping each other, which then increases the chance of the true breeding value ranking differently from the EBV. These simulations have illustrated the influence of key factors in the design of trials. First, the range of EBVs of sires should be maximised. This may require the use of frozen semen from older boars, in addition to the use of younger boars, to achieve wide ranges in EBVs. Second, younger boars have lower ACC in general. Consequently, proportionally more and younger boars need to be used. Finally, there are a number of factors and their interactions that determine the outcome of any design. Therefore, each trial design should be evaluated with the program by Crump (2005) before the trial is implemented.

This work was funded under APL. *AGBU is a joint venture of NSW Department of Primary Industries and the University of New England.

References

CRUMP, R.E. (2005). In 'Manipulating Pig Production X', p. 101, ed J.E Paterson (Australasian Pig Science Association: Werribee).

Australian Pork

Feeding for maximum lean growth does not always maximise profitability

D. Sirisatien*, P.C.H. Morel** and G.R. Wood*

*Macquarie University, NSW 2109. **Massey University, Palmerston North, New Zealand.

Feed intake, live weight and back fat thickness of pigs are measured routinely to generate lean growth curves (NRC, 1998). Lean growth curves are used to formulate diets that meet the pig's nutrient requirements for maximum lean growth. This approach is also assumed to maximise profitability. Because the number of diets fed and their energy and amino acid content and feeding level vary, there is a large number of possible feeding schedules. Only one, however, will result in maximum profit. To determine such a feeding schedule, a computer program linking linear programming, a simple stochastic pig growth model and non-linear optimisation mathematics has recently been developed (Alexander *et al.,* 2005). In this paper we will compare the economical merit of feeding pigs to maximise lean growth (Lean) or to maximise profitability (Optimise) using weekly diet changes for pigs between 20 kg and 85 kg live weight.

In the growth model, genotypes are characterised by their potential for maximum protein deposition (Pdmax), minimum body lipid to protein ratio (minLP) and digestible energy intake level expressed as a fraction (p) of the standard NRC curve. The 18 genotypes investigated were a combination of three Pdmax values (120, 160 and 200), three minLP values (0.6, 0.8 and 1.0) and two p values (0.8 and 1.0). The growth model was also used to calculate the nutrient requirements needed to reach maximum lean growth, i.e. maximum protein deposition. For each combination of genotype and feeding strategy (Lean or Optimise) growth was simulated for a population of 200 pigs and typical New Zealand economic condition and payment schedule. Each simulation was replicated 10 times.

Table 1. Mean gross margin per pig place and year ($NZ), with SD in brackets, for each of 18 pig genotypes and two diet formulation methods (Lean and Optimise).

	p	0.8	0.8	1.0	1.0
Pdmax	minLP	Lean	Optimise	Lean	Optimise
120	0.6	77.5 (1.0)	168.7 (7.1)	38.6 (0.7)	40.5 (1.0)
120	0.8	72.7 (1.3)	138.9 (6.8)	41.3 (1.0)	43.7 (0.8)
120	1.0	65.9 (1.8)	99.0 (6.7)	43.5 (0.7)	46.3 (1.0)
160	0.6	272.4 (5.2)	333.3 (5.2)	119.8 (2.6)	185.2 (10.0)
160	0.8	255.0 (2.3)	296.4 (2.8)	113.5 (2.1)	150.1 (12.4)
160	1.0	240.9 (4.2)	251.3 (3.3)	100.8 (3.8)	118.4 (3.5)
200	0.6	389.4 (1.7)	390.2 (1.4)	337.9 (2.6)	370.0 (7.5)
200	0.8	376.5 (1.8)	378.4 (3.1)	321.3 (3.1)	334.7 (3.1)
200	1.0	311.0 (5.5)	316.7 (6.8)	260.3 (4.9)	311.4 (8.7)

For all the genotypes investigated, the gross margin per pig place and year was higher for Optimise than Lean (Table 1). Parameters Pdmax and p were the main determinants of the difference in profitability between the Optimise and Lean. The greatest differences occurred for the following (Pdmax, p) combinations: (120, 0.8) and (160, 1.0). The least differences occurred for: (120, 1.0) and (200, 0.8). The reason for the differences is now described. Under lean growth, protein is deposited irregularly and at a rate under Pdmax; while under optimal growth, protein is deposited less irregularly and at a rate closer to Pdmax. Lean growth fixes the ratio of lysine to digestible energy and demands a higher energy density to balance amino acids, which in turn limits available protein. We conclude that feeding for maximal lean growth compromises profitability. We also note that loss in profitability is related to pig type.

References

ALEXANDER, D.L.J., MOREL, P.C.H., and WOOD, G.R. (2005) "Optimisation in Action", ed J. Pinter, (Kluwer Academic Publisher) (*in print).*

NRC (1998) In 'Nutrient Requirements of Swine, 10th edn' pp 31-46. (National Academic Press: Washington, DC, USA).

A new concept for feed evaluation

S. Boisen

Danish Institute of Agricultural Sciences, DK-8830 Tjele, Denmark.

Feed quality and feed costs are dominating factors in pig production. An accurate feed evaluation system is important for improving production and reducing feed costs as well as addressing the problems associated with surplus nutrients entering the environment. Traditionally, feed evaluation has focused on energy value with energy evaluation systems usually based on results from animal experiments.

Feed evaluation for pigs is currently done under many different national systems, which are based on digestible energy (DE), metabolisable energy (ME) or net energy (NE). However, performance results in commercial pig production are influenced by many factors depending on the specific production system. In addition, none of the current feed evaluation systems account for the additive energy value from contributing nutrient fractions in the final diet (Boisen and Verstegen, 2000). Therefore, small-scale animal experiments may not be an ideal basis for a feed evaluation system aimed at general recommendations.

Feed evaluation should therefore be based directly on the individual properties of the feed, as this would enable optimal diets to be developed for specific production purposes. Furthermore, feed evaluation needs to relate specifically to different steps during the feed production process:

- Individual feedstuffs and other ingredients should have relevant properties, such as digestible nutrients and other compounds, characterised precisely before the diets are produced.
- The impact of processing on the characteristics of the feedstuffs and the complete diet should be controlled.
- Nutrient composition and other compounds in the diet should be related to the specific feeding purpose.

The digestibility of nutrients is usually based on table values, which are not always accurate as the energy value of feedstuffs changes with the composition of organic matter present. However, this variation can be measured accurately by *in vitro* methods that simulate the degradation process of the digestive tract (Boisen, 2000). Such analyses are necessary for an accurate characterisation of feedstuffs.

To optimise animal diets, it is crucial to achieve the correct proportion of digestible amino acids and energy value of the digestible nutrients. Standardised digestible amino acids (SDAA) can be obtained from tabulated values or from analyses of *in vitro* digestibility, corresponding to the real digestibility, after correction for specific endogenous amino acid losses (Boisen, 2000). Potential physiological energy (PPE) from ingredients can be calculated from documented ATP potentials given in textbooks. The contributing properties of SDAA and PPE are additive in the diet and should be optimised according to a minimal surplus of amino acids.

Starch is usually the main energy source in pig diets and as a pure energy source has no specific properties, unlike other nutrient fractions, such as sugars, dietary fibre, lipids and proteins. Starch is therefore considered as the energy reference for all other dietary components. Thus, the energy value of other dietary components is determined by the impact on the energy value of the diet when starch is replaced by a specific component. For example, the energy value of dietary lipids, which in growing pigs are assumed to be deposited directly, would include the energy saved from the fatty acid synthesis from glucose (starch).

In practice, many feedstuffs are used to optimise pig diets. These feedstuffs can be very different in their composition of non-starch nutrients. Therefore, feed optimisation requires guidelines on the composition of these nutrients as well as their single components, e.g. the fatty acid compositions of lipids. Furthermore, all other specific components with nutritional influence, such as anti-nutritional factors, should be considered according to their possible negative impact on the energy and protein value of feeds. In the future agreement on an international feed evaluation system that is based directly on the properties of a feed would enable developments in 1) the science of feed evaluation, 2) general digestive physiology 3) animal performance under different production conditions and 4) advantages for the international trade of feeds.

References

BOISEN, S. (2000). In 'Feed evaluation. Principles and Practice', pp. 153-168, eds P.J. Moughan, M.W.A. Verstegen and M.I.Visser-Reyneveld. Wageningen Pers.

BOISEN, S. and VERSTEGEN, M.W.A. (2000). In 'Feed evaluation. Principles and Practice', pp. 57-76, eds P.J.Moughan, M.W.A. Verstegen and M.I.Visser-Reyneveld. Wageningen Pers.

Implementation of a new feed evaluation system for pigs

S. Boisen* and P. Tybirk**

*Danish Institute of Agricultural Sciences, DK-8830 Tjele, Denmark. **Danish Agricultural Advisory Centre, DK-8200 Aarhus, Denmark.

A new feed evaluation system for pigs became the official feed evaluation system in Denmark in 2004. The system is based on a new concept for feed evaluation as described by Boisen (2005). The purpose of this paper is to provide details about the performance of the new system in evaluating feed value. The new system calculates feed value based on the chemical and *in vitro* digestibility analyses of feedstuffs. In addition, specific variations in carbohydrate composition are considered (Table 1). The energy value is calculated by specific energy coefficients for the nutrient fractions (Table 2).

Table 1. Chemical composition (% of the feedstuff) and *in vitro* digestibility (%) of selected common feedstuffs for pig diets.

Feedstuff	DM[1]	Ash	CP[2]	CF[3]	EDOM[4]	EDOMi[5]	EDN[6]	ECcarb[7]
Barley	85	1.9	9.2	2.6	84	79	90	11.6
Wheat	85	1.6	9.9	2.1	91	87	93	11.6
Soybean meal	88	6.5	42.7	2.5	91	72	95	9.0

Dry matter. [2] Crude protein. [3]Crude fat. [4]Enzyme digestibility of organic matter.(at faecal level). [5]Enzyme digestibility of organic matter at *ileal* level. [6]Enzyme digestibility of crude protein at *ileal* level. [7]Energy coefficient for digestible carbohydrates

Table 2. Energy value of slaughter pig diets using the new Danish feed evaluation system.

Nutrient fraction	Calculation of fractions (% of feed)	Energy factor (MJ per kg)[1]
RDCP[2]	CP x EDN/100	9.9
RDCF[3]	CF x 90/100	31.7
EDC[4]	OM[5] x EDOMi/100 - (RDCP +RDCF)	11.3[6]
FERMC[7]	OM x (EDOM - EDOMi)/100	7.0[8]
EIDMi[9]	OM x (100 - EDOMi) /100 + 0.3 x ash	- 2.8[10]

[1]Potential physiological energy (see text). [2] Real digestible crude protein. [3] Real digestible crude fat. [4] Enzyme digestible carbohydrates. [5] Organic matter. [6] General coefficient for diets; Individual coefficients for feedstuffs - see Table 1. [7] Fermentable carbohydrates. [8]Energy value of absorbed short-chained fatty acids (SCFA) from fermented organic matter, mainly carbohydrates. [9]Enzyme indigestible dry matter at *ileal* level. [10]Estimated energy costs for specific extra losses of protein and lipids throughout the digestive tract.

The energy value (FUp) of feedstuffs and diets for pigs is corrected for specific energy costs caused by undigested dry matter (Table 2). Similarly, the specific extra costs for amino acids are corrected for by using a standardised digestibility for amino acids (SDAA). Table values for SDAA (Pedersen and Boisen, 2002) are used in feed optimisation. Table 3 shows the standard energy value of the feedstuffs in Table 1.

Table 3. Nutrient fractions (% of feedstuffs) contributing to the energy value in diets for growing pigs (FUp per kg)[1].

Feedstuff	RDCP	RDCF	EDC	FERMC	EIDMi	FUp per kg
Barley	8.3	2.3	55.0	4.2	18.0	1.05
Wheat	9.2	1.9	61.5	3.3	11.3	1.16
Soybean meal	40.6	2.3	15.9	15.5	24.8	0.89

[1]1FUp corresponds to 7.38 MJ per kg to adjust a typical diet to the former Danish system. Thus:
1 FUp per 100kg = (9.9 x RDCP + 31.7 x RDCF + ECcarb x EDC + 7.0 x FERMC - 2.8 x EIDMi)/7.38

Optimising the diets generally reduced the protein contents of diets for growing pigs. This resulted in reduced nitrogen surplus from pig production with beneficial environmental consequences. The new official Danish system is still premature and requires further development and improvement in some aspects. Therefore, the system should be considered as a first step toward a scientifically correct system of feed evaluation, which will increase production and reduce the environmental problems arising from pig production.

References

BOISEN, S. (2005) In 'Manipulating Pig Production X'. p.115 ed J.E. Paterson, pp xx (Australasian Pig Science Association, Werribee).

PEDERSEN, C. and BOISEN, S. (2002). Acta Agric. Scand., Sect. A., *Animal Sci.* **52**:121-140.

Early weaning affects pig growth performance and carcass characteristics

C.L. Collins*,** , B.J. Leury**, B.G. Tatham* and F.R. Dunshea*,**

*Department of Primary Industries, Werribee, Vic. 3030. **University of Melbourne, Vic. 3030.

Piglets typically undergo a growth check at weaning due to several environmental and social stresses. Early weaning of piglets can result in larger growth depressions immediately following weaning than when pigs are weaned at over three weeks of age (Dunshea, 2003). However, given optimum environmental and dietary conditions, early-weaned animals are able to compensate for any growth depression and achieve slaughter weights at the same age as those weaned conventionally. However, early weaning of piglets can result in negative effects on carcass composition. For example, Dunshea *et al.* (2003) observed that pigs weaned at 14 days of age had a higher depth of back fat at slaughter than those weaned at 28 days. The aim of this investigation was to determine the effect of weaning age and sex on the growth performance and body composition of pigs.

Two hundred and forty pigs (120 males and 120 females) were weaned at either 13 or 21 days of age. Pigs within each replicate were weaned on the same day, with the actual ages of the 13-day-old group ranging from 10-16 days and for the 21-day-old group ranging from 18 to 24 days of age. Pigs were offered *ad libitum* access to feed for the entire experimental period. Pen feed intake and individual live weights were recorded weekly from weaning through to slaughter. At weaning, eight 'focus' animals were selected randomly from each pen of 20 pigs. These animals were scanned six times from weaning through to slaughter using dual energy X-ray absorptiometry (DXA) to measure changes in body composition. Data were analysed using ANOVA.

Table 1. Effect of a 13 or 21 day weaning age (W) and sex (S) on body composition.

Sex	Boar		Gilt			Significance		
Weaning age	13 days	21 days	13 days	21 days	SED[a]	S	W	S x W
Lean content of whole body (%)								
90 days of age	73.5	72.8	72.7	70.7	0.39	<0.001	0.001	0.079
119 days of age	70.1	69.5	69.7	67.5	0.34	<0.001	<0.001	0.018
146 days of age	66.8	65.9	65.0	64.4	0.67	<0.001	0.10	0.74
Fat content of whole body (%)								
90 days of age	12.8	12.1	12.9	13.1	0.33	0.045	0.45	0.094
119 days of age	13.9	14.5	15.0	15.6	0.38	0.004	0.13	0.91
146 days of age	15.9	16.3	17.1	17.4	0.71	0.027	0.40	0.93

[a]Standard error of the difference for sex x treatment

The growth path from weaning to 90 days of age was similar for both treatment groups. Daily gain from weaning until 90 days of age was greater in gilts than in boars (540 *vs* 514 g/d, P=0.03), particularly for early-weaned gilts as indicated by an interaction (P=0.06). Thus, early-weaned gilts grew faster than their boar counterparts up to 90 days of age (537 *vs* 489 g/d) while there was no difference between the sexes in the pigs weaned at 21 days of age (544 *vs* 540 g/d). From 90 days of age gilts grew more slowly than boars (800 *vs* 924g/d, P<0.001) but there was no main or interactive effects of weaning age. At slaughter (146 days of age), gilts were lighter than boars (88.7 vs 94.6 kg, P<0.001) while there was no main effect of weaning age (P=0.70). However, there was an interaction (P=0.08) between weaning age and sex such that early-weaned gilts were heavier than gilts weaned at 23 days of age (89.5 *vs* 87.9 kg) while the converse was true for boars (92.9 *vs* 96.4 kg). There were no treatment effects on body fat content at 90, 104 or 119 days of age (Table 1). At 90 days of age the early weaned pigs had a higher lean tissue and this was particularly so for the gilts as indicated by the interaction (P=0.08) (Table 1). The effect of weaning age on percentage lean tissue declined over the finisher phase but still tended to be evident at slaughter (65.9 *vs* 65.1%, P=0.10). These data suggest that early weaning may increase growth rate and carcass lean in gilts but may reduce growth of boars.

References

LEIBBRANDT, V.D., EWAN, R.C., SPEER, V.C. and ZIMMERMAN, D.R. (1975). *Journal of Animal Science* **40**:1077-1080.

DUNSHEA, F.R., KERTON, D.K., CRANWELL, P.D., CAMPBELL, R.G., MULLAN, B.P., KING, R.H., POWER, G.N. and PLUSKE, J.R. (2003). *Australian Journal of Agricultural Research* **54:**363-370.

5 Air quality, dietary fibre and feed quality

The impact of air quality on animal health

P.K. Holyoake

University of Sydney, Camden, NSW 2570.

Abstract

In intensive livestock operations where animals are housed densely in confinement, airborne contaminants including gasses (ammonia and carbon dioxide) and bio-aerosols (dust - including micro-organisms and their cellular products) are generated. High levels of airborne contaminants can have a negative impact on the health and productivity of animals. To predict and quantify the impact that any independent atmospheric contaminant or combination of atmospheric contaminants will have on the health and performance of pigs is difficult because it depends on the concurrent presence of pathogenic micro-organisms, the ability of the immune system to resist the pathogen load, any pre-existing respiratory deficit and the influence of other factors such as temperature and social stress.

The respiratory tract has several inherent defence mechanisms that help prevent disease. The nasal passages help trap large air particles and prevent their access to the lung. The muco-ciliary apparatus and alveolar goblet cells in the bronchial tree remove inhaled particles and/or destroy those that lodge in the lungs. Many lymphocyte 'pools' in the lung and surrounding tissues play an essential role in initiating immune reactions or eliminating antigens of microbial, toxic or environmental origin by immunological effector mechanisms.

Airborne gasses reduce the local defence mechanisms of the respiratory tract and allow infectious pathogens to colonise and cause disease. Ammonia acts as an irritant to the eyes and upper respiratory tract (nose and trachea), resulting in ocular and nasal discharge and coughing in exposed animals. Ammonia also reduces the clearance mechanisms of the muco-ciliary escalator in the trachea and bronchioles by increasing mucous viscosity and decreasing the number of mucous-producing goblet cells. Carbon dioxide is usually not harmful but its presence at high concentrations in livestock buildings is suggestive of poor ventilation and high concentrations of other contaminants.

Dust in piggery buildings may contain a mixture of aerosolised feed particles, bedding, exfoliated skin, viable and non-viable micro-organisms and dried products from the animals' manure. Dust particles may also adsorb noxious gasses (Takai *et al*., 2002). Large dust particles act as irritants to the upper respiratory tract, while respirable dust particles (less than 5 μm in diameter) are small enough to move down into the lungs. The impact that respirable dust has on the lung and the health of the animal will depend on the substances carried with it, and range from acute allergic reactions to low-grade chronic inflammation.

The majority (99%), of micro-organisms in the atmosphere of piggery buildings are dead and are likely to cause little direct damage. Research suggests that endotoxin (a component of the cell wall of gram-negative bacteria) can be extremely damaging to the respiratory tract and the health of the animal. It appears that endotoxin has little effect on the upper respiratory tract, but is cytotoxic to pulmonary cells and is instrumental in stimulating alveolar macrophages to recruit neutrophils, triggering immune and inflammatory changes in the lung.

The impact that air quality has on respiratory disease in pigs depends on the virulence of the pathogen and the nature and concentration of the contaminants in the environment. Controlled challenge experiments suggest that pigs are not affected significantly by *Mycoplasma hyopneumoniae* infection in the presence of high concentrations of atmospheric ammonia. In contrast, ammonia increases the severity of respiratory disease in pigs challenged with *Bordetella bronchiseptica* and *Pasteurella multocida*. Many on-farm epidemiological studies have demonstrated the effect that air quality has on the health and productivity of pigs, to the extent that improving the environment (improving hygiene, reducing stocking density, optimizing ventilation, implementing best practice manure management strategies, controlling temperature and humidity) may be a more beneficial management strategy to improve growth performance than attempts to eliminate specific infectious pathogens (Banhazi *et al*., 2004c; Banhazi *et al*., 2005a; Banhazi *et al*., 2005b).

Introduction

Animals housed in confinement facilities in Australia include pigs, chickens and stabled horses. As a result, the environments in which these animals are housed become contaminated with the by-products generated from the animals, their faeces, bedding materials (if used) and feed. These by-products include gasses (ammonia and carbon dioxide) and bioaerosols (dust, bacteria and their components and fungal spores).

Air quality studies have sought to identify and quantify the contaminants present in the animals' environment and to characterize their impact on animal health. This is a difficult task at the farm level, given that there are likely to be a multitude of individual contaminants present at any one time at varying concentrations depending on such factors as stocking density, level of hygiene, ventilation rates, temperature, humidity and pig activity. In addition, the concentrations of airborne pollutants change over time (both diurnally and seasonally) and spatially. Therefore, the impact of these pollutants on the health of animals would not be static and most probably would also change over time. The response of individual animals housed in such an environment is likely to depend on the underlying health and immune responsiveness of the individual's respiratory system.

The first reports indicating health hazards for humans working in intensive livestock production systems were published more than 20 years ago. A number of specific clinical entities have been recognized among humans working in the intensive animal industries. Recent studies in Australia have sought to determine the degree of risk placed on the respiratory health of humans who care and manage intensively-housed animals, particularly pigs (Holyoake, 2002; Driesen, 2003). Studying the effects of airborne pollutants on human subjects is relatively easy, when compared to animal studies. Using current measurement methods (such as personal dust samplers) it is possible to study individual pollutant exposure of farm workers and therefore develop an understanding of possible health effects. However, similar data on exposure doses of individual pigs cannot be obtained under farm conditions. Yet, it would be important to obtain such data as overseas and Australian studies have demonstrated that exposure dose rates can be very different from concentrations measured by static instrumentation (area measurements) (Bartz and Hartung, 1993; Holyoake *et al.*, 2002; Moore-Colyer, 1996; Talai *et al.*, 1996). Therefore, studying the relationship between the concentrations of airborne pollutants measured by stationary instruments and individual exposure doses of animals would be essential in order to develop a full understanding of the effects of airborne pollutants on animals under farm conditions.

This review paper focuses on the published literature, as well as recent unpublished data stemming from locally-funded research in Australia, pertaining to air quality and how it affects the health and growth performance of intensively-housed pigs. The first section of this paper will describe the structural and functional defense mechanisms of the respiratory tract that allow animals to cope with atmospheric contaminants. The nature of non-infectious air contaminants (ammonia, carbon dioxide, dust, micro-organisms and endotoxin) and their impact on the respiratory system are discussed. Information from the published literature is presented on how these agents interact with each other and with infectious agents to cause respiratory disease and poor performance in pigs. Epidemiological studies on the impact that air quality has on the health and growth performance of pigs on-farm are presented together with studies that highlight the relationship between hygiene and air quality.

The impact of atmospheric byproducts on the lung

A number of infectious agents are widely considered to be primary aetiological agents of swine respiratory disease. However, non-infectious airborne contaminants that are carried on dust such as endotoxin (part of the cell wall of gram negative bacteria) and peptidoglycan (part of the cell wall of all bacteria, particularly gram positive), may also contribute to respiratory disease. The potentially hazardous gasses and bioaerosols present in livestock buildings have been reviewed by Donham (1995) and are presented in Table 1. Despite the multitude of contaminants identified, the main substances routinely measured during air quality monitoring are ammonia, carbon dioxide, dust (total and respirable), viable airborne bacteria and endotoxin.

Table 1. Dust sources and microorganisms in swine buildings. Taken from Gonyou *et al.,* (1999).

Dust sources	Bacteria and Fungi
Feed particles: grain dust, antibiotics Swine protein: faeces, urine, dander, serum Other agents: bedding, endotoxin, dust mites, mold, pollen, insect parts, mineral ash, field dust, building materials, microbial proteases, ammonia adsorbed to particles, infectious agents	Gram-positive cocci: *Staphyloccocus* spp, *Micrococcus* spp, *Aerococcus* spp, *Streptococcus* spp, *Enterococcus* spp. Gram positive bacilli: *Corynebacterium* spp. *Bacillus* spp. Gram-negative bacilli: *Acinetobacter* spp., non-fermentative gram-negative bacillus (*Enterobacter* spp, *Pasteurella* spp, *Vibrio* spp) Fungi: *Alternaria* spp, *Cladosporium* spp. *Penicillium* spp.

The nasal passages serve to filter, humidify and warm inspired air. The design of the nasal turbinates creates a swirling motion to the inspired air so that most particles larger than 5 μm in diameter impinge upon the mucus blanket in the rostral portion of the nasal cavity. These entrapped particles are transported by ciliary action to the pharynx and swallowed (Gonyou *et al.,* 1999).

When these functions cannot be adequately performed by the nasal passages due to damaged turbinates, antigenic and non-antigenic particles may reach the lower respiratory tract where they may cause or aggravate disease conditions (Jericho, 1968). The predominant immunoglobulin in the upper respiratory tract (nose, pharynx, larynx and trachea) is IgA (Pabst and Binns, 1994).

The lung is the internal body organ with the most extensive environmental exposure and the most intimate contact with tissue, blood and the atmosphere (Jericho, 1968). Yet, despite continuous exposure, the normal broncho-pulmonary system is able to maintain its sterility. The lung of the pig clears more bacteria from the blood than the liver or spleen due to a huge number of pulmonary intravascular macrophages which cover 16% of the lung capillary surface (Pabst and Binns, 1994). The basic defense mechanism of the lung relies on clearance of particles within the bronchial tree by the muco-ciliary apparatus, and phagocytosis of those particles that deposit in the alveoli by the alveolar macrophages. The lung contains large numbers of lymphocytes found in different compartments: (1) the pulmonary intravascular pool, which is organ-specific and shows a unique migration pattern; (2) the interstitial lymphocyte pool, which is equivalent to the whole blood pool; (3) the bronchus-associated lymphoid tissue (BALT) which develops as a result of microbial stimulation; (4) the intraepithelial and lamina propria lymphocytes of the bronchi, with their typical subset composition; and (5) the lymphocytes in the bronchoalveolar space which can be sampled by bronchoalveolar lavage (Pabst and Binns, 1994). The major immunoglobulin class in the lung is IgG. In conditions of optimal air quality, the respiratory system of pigs is able to eliminate 99% of a given exposure of *Staphyloccus aureus* within 6 hours, and 99.9% of a given exposure of *Pasteurella multocida* within 24 hours. This clearance is partly related to the rate of decay of the bacteria after aerosolization (Baekbo, 1998). Bio-aerosols (particularly dust) and gasses (ammonia) have an impact on the ability of these two systems to function optimally.

Ammonia

Ammonia is a common contaminant of swine buildings, with concentrations ranging from 0 ppm to 68 ppm measured on farms in Australia (Skirrow and Cargill, 1994; Banhazi *et al.*, 2000; Holyoake, 2002). Banhazi *et al.* (2000) reported that the mean concentration of ammonia on 141 herds was 3.33 ppm, with concentrations above the recommended threshold for optimal health of 7 ppm approximately 8% of the time. Ammonia is easily detectable at concentrations of 5 ppm to 10 ppm and is irritating to nasal membranes and to the upper respiratory tract at concentrations above 25 ppm (Robertson, 1994). Gaseous ammonia is highly soluble in water and is adsorbed from the inspired air by the respiratory mucus so it rarely reaches the lungs. However, ammonia may reach the lungs transported by dust particles that are themselves able to reach the lungs (Curtis *et al.*, 1975).

Ammonia is an irritant and induces inflammatory changes to the upper respiratory tract (nasal turbinates and trachea) in exposed animals. Drummond *et al.* (1980) reported cellular infiltrates in the trachea and nasal turbinates of pigs exposed to ammonia (Drummond *et al.*, 1980). Doig and Willoughby (1971) reported microscopic changes including loss of cilia, thickened epithelia and decreased number of goblet cells in the trachea and turbinates of pigs exposed to ammonia at 100 ppm. The impact that ammonia has on the lung depends on the concurrent presence of other atmospheric contaminants that can penetrate into the lower respiratory tract. Previous exposure to ammonia can enhance the effect of airborne bacteria through reduced ability of the lung to clear exogenous particulate matter (Jericho, 1968; Curtis *et al.*, 1976; Drummond *et al.,* 1978*).* Acute exposure to nebulised ammonium chloride slows particle transport by altering the viscosity of the mucous layer in the bronchial tree. (Jericho, 1968) Atmospheric ammonia at concentrations of 50 ppm and 75 ppm decreased the clearance rate of non-pathogenic *Escherichia coli* from the lungs of piglets by 51%, relative to non-exposed control pigs (Drummond *et al.*, 1978). In the absence of concurrent airborne bacteria, ammonia may have minimal effect on the lung (Strombaugh *et al.*, 1969).

The studies highlighted above suggest that ammonia is a primary irritant and that it decreases the clearance mechanisms of the lung, therefore, the effect that it has on the health and growth performance of pigs will depend on the concentration of ammonia and the presence of other infectious and non-infectious atmospheric contaminants. Clinical signs in pigs exposed to relatively high concentrations of ammonia (more than 50 to 100 ppm) under experimental conditions include coughing, lethargy and secretions from the eyes, nose and mouth, but it appears that animals are able to adjust to these exposures after 1-2 weeks with reduced symptoms (Strombaugh *et al.*, 1969; Drummond *et al.*, 1980). Both pigs and domestic fowl demonstrate a preference for fresh air over an ammoniated atmosphere, but when given a forced choice between thermal comfort and access to fresh air, cold-stressed pigs chose the former over the latter (Wathes *et al.*, 2004). At very high concentrations (280 ppm for 36 hours) of ammonia, exposed gilts showed frothing from the mouth, excessive secretions from the nose and mouth, shortened respiration and convulsions (Strombaugh *et al.*, 1969).

There are conflicting reports on the effect of ammonia exposure on the growth performance of pigs. Exposure of grower pigs to ammonia at concentrations of 10 ppm to 150 ppm had a highly significant effect on feed consumption

and average daily gain, with no significant effect on feed conversion (Strombaugh *et al.*, 1969). Exposure of 4-week-old pigs to ammonia at 50 ppm, 100 ppm and 150 ppm ammonia resulted in a percentage reduction in growth rate of 12%, 30% and 29%, respectively, relative to non-exposed pigs (Drummond *et al.*, 1980). In contrast, Curtis *et al.* (1975) conducted seven trials involving 92 pigs of varying age and found the growth rate and respiratory tract structure of pigs was not affected by exposure to ammonia at 50 ppm and above. Ammonia also had no effect on body weight gain and lung structure when combined with dust at concentrations of 10 mg/m^3. Performance was only affected when aerial dust was applied at 300 mg/m^3 in combination with ammonia (Curtis *et al.*, 1975). This is in agreement with Wathes *et al.* (2004), who suggested that dust had a larger effect on pig growth than ammonia. All experiments were conducted using pigs more than 4 weeks of age housed in exposure chambers for periods of approximately four to five weeks. It may be that the differences in performance between experiments were due to differences in the underlying health status of the pigs or to genetic differences that may have affected the pigs' physiological/immune response to ammonia exposure that were not measured.

Murphy and Cargill (2004) reported a significant reduction in the growth rate of pigs exposed to ammonia at 10 ppm, 25 ppm and 50 ppm compared with untreated controls (0 ppm), and the reduction increased as levels increased from 10 to 50 ppm (Murphy and Cargill, 2004b). Similar reductions were recorded in feed efficiency, but were only significant at the 50 ppm concentration. In this experiment, pigs were also dosed intranasally with a solution of *Viridans streptococcus* collected from the airspace of a shed that had housed growing pigs. Growth rate suppression and feed conversion efficiency of pigs were further worsened when pigs were exposed to bacteria after prior exposure to ammonia. In this study, systemic lymphocyte proliferation and phagocytic activity (% granulocytes – eosinophils and neutrophils with phagocytic potential) were significantly increased in response to both ammonia and bacteria, but not ammonia alone. The authors hypothesized that ammonia damaged the integrity of the respiratory mucosa allowing bacteria or their toxins better access to the animals' immune tissues (Murphy and Cargill, 2004b).

These results suggest that ammonia by itself has minimal effect on the respiratory health and growth performance of growing pigs. However, ammonia is likely to enable other pollutants and infectious pathogens commonly encountered in commercial swine production to have a greater effect on the respiratory tissues and the immune response of pigs. It also stresses the importance of airborne bacteria present at high levels where there is sub-optimal hygiene as a major risk factor for inducing poor performance in pigs previously exposed to ammonia.

Carbon dioxide

The concentration of carbon dioxide in the atmosphere is frequently measured during air quality studies on swine facilities. Banhazi *et al.* (2000) reported a mean carbon dioxide concentration of 860 ppm (range approximately 400 ppm to 2600 ppm) during a survey of 119 pig sheds. Similar concentrations of atmospheric carbon dioxide (up to 2400 ppm) were reported in controlled environment weaner sheds by Holyoake (2002). One study reported reduced growth rate and increased prevalence of respiratory disease in pigs exposed to concentrations of carbon dioxide above 1500 ppm together with high levels of ammonia (Donham *et al.*, 1989). Carbon dioxide is a by-product of the pigs' respiration and is linked to stocking density within a shed, ventilation rates and pig activity. Several studies have found a correlation between indoor carbon dioxide concentration and other atmospheric contaminants (Baekbo, 1990; Donham, 1991). Therefore, although carbon dioxide in isolation is not significant for disease development, high concentrations of carbon dioxide usually mean high concentrations of dust, ammonia, micro-organisms and endotoxin (Robertson, 1994).

Dust

Dust within piggery environments is usually classified according to size. Total dust refers to particles up to a maximum size of 20 μm. Dust particles that are too large to be respired into the lungs (greater than 5 to 10 μm diameter) are unlikely to cause respiratory disorders but the inhalation and deposition of animal protein of this size class into the nose, trachea and bronchi can cause allergic reactions and may lead to asthma in humans. Particles of less than 5 μm have a greater impact than larger particles on respiratory structures because of their ability to penetrate into the deeper respiratory system.

Dust particles can originate from many sources (feed, skin squames, particles of bedding, faeces, urine, bacteria, yeasts and fungi). Major biologically active agents in dust include endotoxin and beta 1,3-glucan, a cell wall component of molds (Jolie *et al.*, 1996).

Because of the complex nature of dust and the potential differences in its composition, it is difficult to standardize animal exposure studies that involve dust to determine its effects on health and performance. However, despite the technical difficulties involved in studying the effect of dust on animals under controlled conditions, the

importance of dust cannot be underestimated. Indeed, it appears that dust is the universal carrier of almost all airborne pollutants, including ammonia and bacteria (Dutkiewicz, 1997; Ikeguchi, 2001; Martin *et al*., 1996; Takai *et al*., 2002; Thedell *et al*., 1980). Bacteria do not survive very well in the air without the protection of dust particles and Danish results have demonstrated that dust-bound ammonia is potentially more important than aerial ammonia. Therefore it is highly unlikely that the importance of airborne bacteria and endotoxin can be demonstrated independent of dust particles. Indeed, when bacteria are measured using modern technology, a high correlation was found between dust particles and viable particles (Agranovski *et al*., 2002; Agranovski *et al*., 2004). Characteristics of dust that indicate its potential health hazard include: (1) particle size in the respirable range (less than 5 μm diameter), (2) high protein concentration, (3) high bacterial and fungal counts, (4) endotoxin activity, and (5) adsorbed irritating gasses (Donham and Leininger, 1984). Particles less than 5 μm have both adverse effects and indirect effects on pigs by acting as a carrier of potentially harmful gasses and micro-organisms.

It would appear that dust of any type can impact on the health and growth performance of pigs in a number of ways. Dust may act as a non-specific irritant of the upper respiratory tract, as demonstrated by lesions in the trachea and nasal turbinates of rabbits and guinea pigs housed in a swine confinement building for 12 months (Donham and Leininger, 1984). Lesions in these animals included loss of cilia and squamous metaplasia, and were suggestive of a chronic, non-specific, low-grade irritation. The authors reported that these lesions would make the lungs more vulnerable to aerosolized particles, gasses and infectious agents.

Dust has an impact on the clearance mechanisms of the lungs. In one study, exposure of guinea pigs to coal dust for 4 weeks reduced the clearance rate of non-pathogenic *E. coli* relative to control dust-free guinea pigs (Rylander, 1969). This suggests that a smaller dose of pathogenic bacteria may be sufficient to induce colonization and disease in animals housed in dusty environments.

There is ample evidence that bioaerosols carried on dust stimulate inflammatory changes in the lungs and the periphery of exposed animals, including humans. Jolie *et al.* (1999) attempted a 15-week experimental exposure of pigs to airborne dust (fine corn/soybean meal) with added endotoxin in a continuous flow chamber. A non-specific inflammatory response was found in exposed and control pigs, suggested by increased neutrophils in bronchoalveolar fluid (BAL) and small inflammatory areas of the lung tissue. The findings in the control pigs implied that even low dust concentrations and possibly endogenous peptidoglycan originating from the microbial flora of the pigs themselves, the feed and the environment, can induce cellular changes in the BAL and that a true control pig does not exist. In addition, exposed pigs developed a mild eosinophilia, indicating an allergic response-most likely to the soybean meal in the dust. In this experiment, only a small number (12) of pigs were used, however the results indicated that the control pigs grew faster than the exposed pigs (Jolie *et al*., 1999b). Donham and Leininger (1984) described histiocytic pneumonia in rabbits and a guinea pig housed in a swine confinement building for 12 months. Such lesions were thought to be due to inhaled particles and represented either the intermediate stages of bacterial, viral or mycotic infections or a toxic or immunological reaction. The presence of plasma cells in the lungs and antibodies specific to swine-house dust in the sera suggested that the immune system was involved and exposure may have resulted in a hypersensitivity reaction in the animals (Donham and Leininger, 1984).

Exposure of humans to dust in pig-rearing buildings results in airway irritation and may lead to malaise, chills and fever. Larsson *et al.* (1994) demonstrated acute airway inflammatory reactions in humans after short-term (2 to 5 hour) exposure to swine dust during weighing of pigs in a swine-confinement building. Exposed individuals showed a 75-fold increase in neutrophilic granulocytes, a two-to three-fold increase in mononuclear cells and a significant increase in eosinophilic granuolocytes in the bronchoalveolar fluid 1 day after exposure. The number of leukocytes in peripheral blood was almost doubled 6 hours after exposure. The authors suggested that this response was most likely due to the endotoxin present in the dust (Larrson *et al*., 1994). Zhang *et al.* (1998) used canola oil to reduce dust, endotoxin, ammonia and hydrogen sulphide in a swine confinement building and demonstrated an improvement in respiratory responses of human subjects exposed to the atmosphere. Improved air quality resulted in reductions in shift changes in forced expiratory volume (FEV1) and forced vital capacity (FVC), and the white blood count of the subjects was improved across the work shift (Zhang *et al*., 1998).

Reports on the effect of airborne dust on pig health and performance are variable. Wathes *et al.* (2004) reported that 'artificial' dust (feed, barley straw and faeces mixed by weight in the proportions of 0.5:0.1:0.4) adversely affected feed intake and live-weight gain of weaned pigs at concentrations greater than 5.1 mg/m^3 for 5.5 weeks. This result was observed across ammonia concentrations up to 37 ppm. Surprisingly, the severity of respiratory disease among these pigs was mild, despite the presence of potential pathogens (*Mycoplasma hyopneumoniae* infection (Done *et al*., in press). Chiba *et al.* (1985) reported a lower incidence and less severe lung lesions in pigs fed on tallow-supplemented diets in an attempt to reduce atmospheric dust than pigs fed on non-supplemented diets. In contrast to these, in a Canadian study, growing pigs (55 to 82 kg bodyweight) naturally infected with *Pasteurella multocida* and *Streptoccocus suis* were exposed to

feed dust or faecal dust (Jansen and Feddes, 1995). The test pigs were exposed to four times more dust than the control pigs. The study found no relationship between the dust concentration and the lung score or between dust concentration and the weight gain of pigs. The difference in the outcome of these studies may have been due to differences in the nature of the dust-particularly particle size and the content of biologically-active agents (e.g. endotoxin).

Micro-organisms

Most airborne microorganisms in piggery buildings are considered to be non-pathogenic, originating from the skin and faeces of the pig and from feedstuff and straw. Viable micro-organisms make up less than 1% of the total number of airborne micro-organisms and most microbes are enteric in origin (Baekbo, 1989). The vast majority of bacteria are gram-positive, with some gram-negatives (Donham, 1995). Molds make up only a small proportion of viable microorganisms, with approximately 80% of the molds being fungi and the remaining being yeasts (Baekbo, 1998). Air-borne bacteria may be distributed in three different ways: (1) attached to dust particles, (2) contained in gross droplets expelled from the nose and mouth, and (3) in droplet nuclei, produced as the result of evaporation of smaller droplets expelled from the nose and mouth. It is the bacteria attached to dust and the droplet nuclei that are of most concern in terms of long-range disease transmission, particularly in high humidity environments (Queensland and southern Australia in winter).

High humidity and high temperatures are favorable to the health and productivity of pigs due to reduced bacterial colonies in these conditions (Gordon, 1963). This was demonstrated during a study of 160 pig housing facilities undertaken in Australia where the concentration of airborne bacteria in pig sheds were negatively correlated with high humidity and temperature (Banhazi *et al.*, 2000). In this report, airborne bacteria concentrations averaged 117,000 colony-forming units (CFU)/m^3 and 41% were above the recommended threshold of 100,000 CFU/m^3. Straw-based shelters appeared to have the highest levels of airborne bacteria (Banhazi *et al.*, 2000; Holyoake, 2002). Anderson two-stage viable microbial particle sizing samplers are routinely used to quantify bacteria on-farm. This method of quantifying airborne bacteria is likely to under-estimate the true concentration, as it measures clumps of bacteria, rather than an individual bacterium and does not take into account non-viable bacteria (Murphy and Cargill, 2004a). In addition, bacteria concentrations are currently sampled using an "incorrect" flow rate (i.e. much lower than what Andersen samplers were designed for), which is likely to have an influence on the number of colony forming units (CFU) captured (Thorne *et al.*, 1992). This reduced flow rate has been used previously in Australia (Cargill *et al.*, 1998; Skirrow *et al.*, 1995) and for overseas studies (Seedorf *et al.*, 1998), so the results obtained by this methodology might be more relevant for comparative studies.

It is difficult to separate the effects of airborne bacteria on the health and performance of pigs from the impact of endotoxin (as a component of gram-negative bacteria) and dust (as a carrier of bacteria). Indeed, there is a strong relationship between airborne bacteria and particle concentrations, when measured simultaneously using advanced technology (Agranovski *et al.*, 2004). Skirrow *et al.* (1995) reported that the severity of pleurisy across 236 batches of pigs from 60 herds in Western and South Australia was positively correlated with the concentration of airborne viable bacteria, particularly *Streptococcus* species, airborne respirable dust and the number of pigs in an air space. The growth rate of pigs was negatively correlated with the severity of pleurisy, and was observed to fall by approximately 5% as the pleurisy grade increased from 0 to 4 (Skirrow *et al.*, 1995). Murphy *et al.* (2000) conducted studies on 11 farms in Australia that were naturally ventilated and operated on an all-in/all-out basis. In this study, the size of the shed and the number of pigs in the shed were measured, together with concentrations of inspirable and respirable particles and concentration of airborne bacteria. Growth rates from 10 weeks of age to 20-23 weeks of age were measured. In this study, there was a negative correlation between stocking density and the growth rate of pigs and a positive correlation between stocking density and airborne viable bacteria. The authors suggested that stocking density is a key risk factor for high concentrations of airborne viable bacteria which may in turn compromise the growth rate of pigs (Murphy, et al., Cargill, and Carr, 2000). However, this study did not take into consideration other environmental effects (temperature, humidity, ventilation rates, hygiene levels) and did not re-adjust the data set for these effects.

Endotoxin and beta (1,3)-D-glucan

Endotoxin concentrations in pig confinement buildings in Australia have been measured at approximately 33 EU/m^3 (range 0 EU/m^3 to 238 EU/m^3) (Banhazi *et al.*, 2000) In this study, dry and lactating sow accommodations tended to have low concentrations of endotoxin while bedded systems had very high endotoxin concentrations. Holyoake (2002) also reported very high concentrations of endotoxin in rice-hull bedded shelters. Endotoxin concentrations were positively correlated with humidity and biological loading of the shed (kg pig per shed), and negatively correlated with ventilation levels.

It would appear that endotoxin is a major risk factor for inducing inflammatory changes in livestock and humans, but it has very little effect on the nasal passages of pigs. In controlled exposure experiments, endotoxin had

no direct affect on the nasal mucosa, as determined by neutrophil counts and albumin concentrations in nasal lavage fluid of pigs (Urbain *et al*., 1996). In contrast, exposure of pigs to 50 ppm ammonia caused inflammation of the nasal mucosa, illustrated by an increased neutrophil count and albumin concentration. Endotoxin only elicited a change in the nasal mucosa after pigs had prior exposure to ammonia.

The impact that endotoxin has on the lower respiratory tract and the generalised inflammatory response are significant. Several *in vivo* and *in vitro* studies have demonstrated that endotoxin and/or organic dusts activate airway epithelial cells, alveolar macrophages and polymorphonuclear cells (Lopez *et al*., 1987; Schwartz *et al*., 1994). Inhaled lipopolysaccharide (LPS) initiates a complex interaction between alveolar macrophages and other inflammatory cells (primarily neutrophils), and this interaction appears to be mediated by specific cytokines. LPS also appears to be cytotoxic to pulmonary cells (possibly bronchial and bronchiolar epithelium and Type II pneumocytes), reflected by an increase in lactate dehydrogenase and alkaline phosphatase into the extracellular milieu of the bronchoalveolar space of exposed rats (Lopez *et al*., 1987). Schwartz *et al*. (1994) used two mouse-challenged models to demonstrate that endotoxin responsiveness was critical to the development of an acute inflammatory response to inhaled corn dust. These authors also demonstrated that endotoxin tolerance could develop, resulting in down-regulation of the inflammatory response of the lower respiratory tract.

Airflow among swine confinement workers has been associated with the concentration of inhaled endotoxin (Donham *et al*., 1989). In non-asthmatic humans, inhalation of endotoxin (lipopolysacharide) was able to induce a systemic inflammatory response (significantly increased total white blood cell and polymorphonuclear cell counts) in the absence of any effect on lung mechanics (no decrease in FEV1, no increase in airway resistance or specific conductance), while in asthmatics the same bronchial challenge has been reported to induce similar blood inflammation associated with a significant response in lung function (Michel *et al*., 1995). In this study, it would appear that the systemic neutrophil response was due to an alveolar cell-mediated inflammation.

Beta (1,3)-D-glucans are present in the cell wall of fungi and *Actinomyceter*. They are polyglucose compounds and are potent inducers of inflammation as well as modulators of the immune system (Fogelmark *et al*., 1994). Organic dusts are likely to contain beta (1,3)-D-glucans, particularly if they are derived from shelters where bedding is used. Aeroallergenic mold spores including *Cladosporium* and *Alternaria* have been recorded at high levels in 'eco-shelters' in Australia where pigs have been housed. Fungal counts reduced as the duration of occupancy of pigs within the sheds increased from 3 days to 2 weeks (Holyoake, 2002). It is likely that endotoxin and beta (1,3)-D-glucan can induce inflammation in isolation, but that the response is increased to induce pathology when these agents are given simultaneously. This was demonstrated in a study where guinea pigs were exposed to endotoxin and curdlan (a form of beta (1,3)-D-glucan) at the same time (Fogelmark *et al*., 1994). Concurrent exposure to agents in isolation caused mild inflammatory changes in the lungs, however, when given simultaneously, they had a significant effect on increasing lung lavage cells (macrophages, lymphocytes, neutrophils and eosinophils). In addition, simultaneous exposure to both agents induced significant histological changes to the lung, with a large influx of inflammatory cells into the alveolar space and the beginning of granuloma formation. The authors suggested that these changes were consistent with those seen in patients with hypersensitivity pneumonitis after repeated exposure to moldy hay. This study suggested that inhaled glucan sensitizes animals to endotoxin through a decrease in the normal function of alveolar macrophages.

The interaction between air quality and infectious agents

The most common infectious respiratory agents of pigs in Australia are *Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, *Pasteurella multocida*, *Streptococcus suis, Haemophilus parasuis* and *Bordetella bronchiseptica*. A number of studies have linked piggery environmental risk factors with the prevalence of disease and/or lung lesions in herds (Donham, 1991; Stark *et al*., 1998).

A study of 1117 pigs from 12 farms indicated that atmospheric ammonia, dust and microbial concentrations in the farrowing house, and dust in the first-stage weaner house, played a significant role in the severity of atrophic rhinitis (AR) (Robertson *et al*., 1990). Farms with low concentrations of these produced pigs with low AR snout scores, despite the presence of the causative agents on the farms. In this study, there were no significant relationships between air quality in the second-stage weaner and the finisher facilities, and the severity of snout lesions. This was likely to be because inflammation and colonization of the nasal mucosa is established in the young pig before it enters the latter growth stages. Drummond *et al*. (1981) reported that the growth of pigs experimentally challenged with *Bordetella bronchiseptica* was slower than non-challenged pigs, but that exposure to ammonia at 50 ppm and 100 ppm did not add to the growth rate decline. However, ammonia-exposed pigs showed muco-purulent nasal discharge that became more profuse and viscous over time. In addition, ocular discharge was observed in ammonia-exposed pigs leading to the development of dark patches under the eyes. This was not apparent in control pigs or pigs exposed to *B bronchiseptica* alone (Drummond *et al*., 1981). Pneumonic lesions and the inflammatory response in the turbinate epithelium appeared to be more severe in ammonia-exposed pigs.

It would appear that the impact that the environment has on the health and growth performance of animals is largely dependent on the magnitude of the environmental insult and the virulence of the pathogens present. One study in the US investigated the impact that fluctuating temperature and/or ammonia exposure had on *M. hyopneumoniae*-induced pneumonia (Clark *et al*., 1993). Four groups of 12 pigs per group were used in this study. Pigs in Groups 2 to 4 were mixed with *M. hyopneumoniae* seeder pigs (Group 1 pigs were not challenged with *M. hyopneumoniae*). In this study, fluctuating temperature (18° C to 21° C) and ammonia exposure (mean 17.9 ppm) did not result in an increase in seroconversion to *M. hyopneumoniae* or coughing among pigs, relative to control pigs. In addition, the growth performance of pigs in the four groups did not differ. Only when the results from Groups 2 to 4 were pooled did *M. hyopneumoniae* have an impact on coughing, seroconversion and growth relative to control pigs. The results of this study suggest that *M. hyopneumoniae* infection alone is not sufficient to induce pneumonia in pigs, despite adverse environmental conditions (ammonia and fluctuating temperature). In contrast, Andreasen *et al.* (1994) demonstrated that high levels of ammonia (50 ppm) increased the extent of pneumonic lesions caused by combined infection with *M. hyopneumoniae* and *P. multocida*, and reduced the pigs' weight gain. Feed conversion tended to increase with increased exposure to ammonia. Ammonia exposure had no effect on clinical disease or lung pathology due to *P. multocida* in isolation (Andreasen *et al.,* 1994). These high concentrations of ammonia are unlikely to occur on piggery operations.

An on-farm study undertaken in the Netherlands investigated the effectiveness of three interventions (filtration and recirculation of air in the weaner rooms, weekly vacuum cleaning of the finishing house and weekly washing of weaner pigs with water) to reduce the exposure of pigs to aerosols (Klooster *et al*., 1993). In this study, the concentration of aerosols in the atmosphere was reduced by 40%, 6% and 10%, respectively with no improvement in daily feed intake, daily weight gain or respiratory health of pigs.

Impact of air quality on respiratory diseases and productivity on-farm

It is difficult to apply the data from controlled exposure studies to actual livestock production units because of the numerous environmental and management variables that exist. Threshold limit values recommended for the environment may not be appropriate on-farm because they do not take into account the mixture of agents found in such environments. One of the first on-farm epidemiological studies associating air quality with swine health was undertaken by Donham in 1991 on 28 swine farms in Sweden. This study was important as it was the first paper to publish exposure guidelines in swine-confinement buildings, many of which are still adhered to today. The author found significant correlations between environmental measurements in the fattening house and disease at slaughter. The most consistent of these was the association between respirable and total microbes and pneumonia. There were no significant associations between environmental measures and disease outcomes in the farrowing house, however, pigs in the farrowing buildings that had poorer air quality had reduced growth rate and high mortalities. Performance in the finishing facilities was also generally poorer as dust, ammonia and carbon dioxide concentrations increased (Donham, 1991).

The impact that air quality has on respiratory disease and pig performance on-farm depends on the magnitude of the concentration of contaminants present and the pathogen load on the pigs. Baekbo *et al.* (1996) found no significant difference in the growth rate of pigs housed in poor air quality rooms with low ventilation (air change rate of $19m^3$/hour/pig) compared to those with improved air quality (air change rate of $52m^3$/hour/pig). In this study, the health and performance of 304 pigs were monitored during four repetitions of the experiment (Baekbo *et al.,* 1996). Three out of the four groups of pigs had a higher weight gain at good air quality, whereas one group had a lower weight gain. Pigs produced at the high air exchange rate showed a reduction in treatments for respiratory disease and in subclinical atrophic rhinitis than pigs produced at low air exchange rates. The authors speculated that the air exchange rates in piggery buildings would be lower during winter and that poor air quality may be responsible for the poor health and growth of pigs produced during the cold season in Denmark. Caution must be made with increasing ventilation rates excessively, as this results in drier airborne materials and drier bronchial mucosa, which may stop ciliary beating and transport, thus interfering with the primary clearance of the mucus blanket of the respiratory tract. A reduced efficiency of mucociliary apparatus creates favorable conditions for antigenic and non-antigenic substances to penetrate into the lower respiratory systems (Jericho, 1968).

Controlled intervention and epidemiological studies undertaken on-farm suggest that strategies that improve hygiene and/or ventilation and that reduce the stocking density of pig rearing units will improve the health and growth performance of pigs through an improvement in air quality. Pigs housed in dirty environments under commercial conditions grow slower and have poorer respiratory health than pigs housed in clean or isolated environments (Crowe *et al.,* 1996; Jolie *et al*., 1999a; Lee *et al.,* 2004 (in press). Systems that incorporate all-in/all-out (AIAO) management, and cleaning facilities between batches maximize hygiene and improve air quality (Cargill *et al*., 1998; Cargill *et al*., 2000). Pigs housed in AIAO sheds that were cleaned thoroughly between batches grew 39 g/day faster, and had significantly less lung damage and pleurisy at slaughter than pigs housed in adjacent AIAO sections that were not cleaned (Cargill and Banhazi, 1998). In New Zealand, the results of a questionnaire-based study demonstrated that frequent manure removal from the nursery, the use of wet/dry feeding and good hygiene were protective against lung

lesions at processing and that slatted floors in combination with liquid manure systems were risk factors (Stark *et al.*, 1998). Cargill *et al.* (1996) undertook a widespread epidemiological study of 32 naturally-ventilated farms in Australia and reported a significant negative correlation between the stocking density (m^3/pig) in the shed and the prevalence of pleurisy in pigs.(Cargill *et al.*, 1996) There were also positive correlations between the number of pigs in the shed and pleurisy prevalence, pneumonia prevalence and coughing rate. The concentration of *Streptococci* species were identified to be an important indicator of poor air quality and strongly correlated with the prevalence of pleurisy in a herd. These studies confirm the importance of optimising hygiene, maintaining adequate shed size and limiting the number of pigs housed in sheds to maximise air quality and hence pig health and performance. The experiment also demonstrated the importance of ventilation, which needs to be taken into consideration, when evaluating the suitability of the building environments.

It would seem that the environment may be growth-limiting in the presence of endemic disease. Straw (1991) found that pigs housed at an isolated research station grew faster and had slightly less pneumonia than pigs housed on their farm of origin. Test station pigs with mild pneumonia grew faster than pigs that were home grown. There were no significant differences in the growth rate of pigs with moderate or severe pneumonia housed at the test station or at the home farm (Straw, 1991). Similarly, pigs reared in isolated facilities with reduced dust and respirable endotoxin grew faster than a comparative group of pigs reared on-site in a commercial weaner room (Crowe *et al.,* 996). These results suggested that, rather than undertaking a pathogen eradication program, significant improvements in pig performance could be made by improving the environment of the pigs on-farm. Even pigs without major respiratory problems respond positively to improved environmental conditions (Banhazi and Cargill, 1998).

Pigs reared in a dirty environment may be under greater immunological challenge, resulting in reduced growth rate. Lee *et al.* (2004) conducted an experiment on a commercial farm in Australia where the growth performance and endocrine responses of male weaner pigs (3-8 weeks of age) were evaluated in two different environments (clean and dirty) and housing (single or groups of 10 pigs per pen). In this experiment, pigs housed in the clean environment with reduced ammonia, carbon dioxide and total dust particles grew faster and consumed more feed than pigs reared in the 'dirty' environment. Pigs housed in groups in the dirty environment had increased cortisol, beta-endorphin and decreased IGF-I concentrations compared to group-housed pigs in the clean environment. Pigs raised in an isolated disease-free research facility were found to have activated alveolar macrophages in bronchoalveolar fluid with no effect on total white blood cell counts compared to pigs from the same breeder facility but housed on-farm and exposed to higher concentrations of infectious and non-infectious agents (Jolie *et al.*, 1999a). The pigs housed on-farm had reduced growth rate, increased white blood cell and neutrophil concentrations in peripheral blood, increased macroscopic lung lesions at slaughter and seroconverted to *Mycoplasma hyopneumoniae* and Porcine Reproductive and Respiratory Syndrome (PRRS) virus. These observations and those of Lee *et al.* (in press) are consistent with Johnson (1997) who demonstrated that pro-inflammatory cytokines, IL-1, IL-6 and TNF-alpha, act directly on peripheral somatic tissues to induce metabolic responses, and act on the central nervous system to alter the neuroendocrine system, reducing growth hormone and increasing plasma corticosteroids. This results in a reduction in feed intake and a shift to partitioning nutrients away from skeletal muscle growth toward metabolic processes that support the immune system (Johnson, 1997).

Conclusions

It is evident that the air inside livestock buildings contains substances that are potentially hazardous to animals and workers. Dose-response guidelines for individual atmospheric contaminants have been suggested for pigs and humans (Donham, 1989; Donham *et al.,* 1989) to help ensure a safe and healthy environment. However, it must be remembered that no one atmospheric contaminant acts alone, and the effects of these pollutants on the health and productivity of animals will vary depending on the mixture and concentrations of the pollutants together and on the dose rate the individual animal is exposed to.

Atmospheric contaminants can affect the health of animals in a variety of ways. Clinical signs of poor air quality that have been observed in pigs during on-farm studies range from reduced growth rate and decreased appetite, to coughing, sneezing and excessive lacrimal secretions. These clinical signs result primarily from airway irritation (particularly due to ammonia and dust) and stimulation of local and generalised inflammatory responses (particularly due to endotoxin).

The ability to predict the effect of atmospheric contaminants on pig health and growth performance would be useful to gain insight into the economic impact of poor air quality. Turner *et al.* (1997) described the use of fuzzy logic to develop a mathematical/computer model for predicting the effects of air quality (ammonia and dust) and immunity level on the incidence, prevalence and severity of atrophic rhinitis in pigs. The data generated from this model compared favorably with real data on the incidence and severity of atrophic rhinitis snout lesions from pigs from four specific farrow-to-finish farms (Turner *et al.,* 1997). The incorporation of models such as this into "precision livestock farming systems" would be a useful tool for pork producers.

Numerous studies have demonstrated the importance of optimising hygiene, ventilation and stocking density on-farm to maximise the health and growth performance of pigs. The application of management strategies such as

all-in/all-out rearing coupled with good hygiene, improve air quality, pig health and growth performance. Assuming that the volume (kg/m^2 of floor space/year) of pigs can be maintained, improvements such as these that do not rely on antibiotics to lift pig productivity are profitable and sustainable.

It is recommended that buildings be evaluated using the existing dose-response guidelines on a semi-annual basis. Hence there is a need for easy-to-use equipment that is not cost-prohibitive to undertake these measurements. Given the increasing popularity of bedded systems in the pork production industry and the routine use of straw, rice hulls and sawdust in the broiler and racehorse industry, it would seem prudent to routinely measure the byproducts of the bedding (fungal hyphae, fungal spores, pollen) and to gather more data on safe exposure limits for the byproducts of this type of production. Routine air quality monitoring would indicate when there is a need for management changes, engineering changes, work practice changes or respiratory protection to keep exposure levels within safe limits for livestock and workers. Such a measurement system is currently being developed in Australia (Banhazi, 2005).

References

AGRANOVSKI, I.E., AGRANOVSKI, V., REPONEN, T., WILLEKE, K., AND GRINSHPUN, S.A. (2002) Development and evaluation of a new personal sampler for culturable airborne micro-organisms. *Atmospheric Environment* **36:**889-898

AGRANOVSKI, v., RISTOVSKI, Z., BLACKALL, P.J., and MORAWSKA, L. (2004) Size-selective assessment of airborne particles in swine confinement building with the UVAPS. *Atmospheric Environment*. **38:**3893-3901

ANDREASEN, M., BAEKBO, P. and NIELSEN, K. (1994). Effect of aerial ammonia on the MIRD-complex. Proceedings of the 13th IPVS Congress, Bangkok, Thailand: p. 429.

BAEKBO, P. (1989). Air quality and pig herd health, Royal Veterinary and Agricultural University, Copenhagen, Denmark.

BAEKBO, P. (1990). Air quality in Danish pig herds. *Proceedings of the 11th International Pig Veterinary Science Congress*, Lausanne, Switzerland: p. 395.

BAEKBO, P. (1998). Effects of noxious gases, dust and micro-organisms on the incidence and severity of respiratory diseases in pigs. *Proceedings of the 15th International Pig Veterinary Science Congress*, Birmingham, England: pp. 135-142.

BAEKBO, P., PEDERSEN, P. and THOMSEN, L. (1996). Impact of air quality on respiratory diseases and productivity. *14th International Pig Veterinary Society Congress*, Bologna, Italy: p. 522.

BANHAZI, T. (2005), Improved air quality measurement procedure - BASE-Q system. *Proceedings of the Australian Association of Pig Veterinarians*, Gold Coast pp. 71-75

BANHAZI, T., and CARGILL, C. (1998) An assessment of the benefits of age-segregated rearing and all-in all-out management in respiratory disease free herds. *Proceedings of the 15th International Pig Veterinary Society Congress*, Birmingham, UK p. 387

BANHAZI, T., CARGILL, C., MARR, G., KEFFORD, A., MOORE, K., KOCH, S., PAYNE, H. and NICHOLLS, R. (2000). Relating airborne pollution to management and housing factors. Final Report to PRDC.

BANHAZI, T., RUTLEY, D., and GLATZ, P. (2004a) Factors affecting thermal conditions in piggery buildings in South Australia. In New Trends in Pig Buildings. Evora, Portugal. CD publication

BANHAZI, T., SEEDORF, J., RUTLEY, D.L. and PITCHFORD, W.S. (2004b) Factors affecting the concentrations of airborne bacteria and endotoxins in Australian piggery buildings. *Proceedings of ISAH Conference*, St Malo, France pp. 197-198

BANHAZI, T., SEEDORF, J., RUTLEY, D.L. and PITCHFORD, W.S. (2004c) Factors affecting the concentrations of airborne particles in Australian piggery buildings. *Proceedings of the ISAH Conference*, St Malo, France pp. 193-194

BANHAZI, T.M., SEEDORF, J., RUTLEY, D.L. and PITCHFORD, W.S. (2005a) Statistical modeling of airborne bacteria and endotoxin concentrations in Australian piggery buildings. In ILES. Bejing, China (Ed. T. Brown-Brandl) pp.72-78

BANHAZI, T.M., SEEDORF, J., RUTLEY, D.L. and PITCHFORD, W.S. (2005b) Statistical modeling of gas concentrations in Australian piggery buildings. In ILES. Bejing, China. (Ed. T. Brown-Branl) p.64-71

BARTZ, J., and HARTUNG, J. (1993) Dust measurements on a horse using an "equine personal sampler". Livestock Environment IV pp.742-746

CARGILL, C. and BANHAZI, T. (1998). The importance of cleaning in all in all out management systems. *15th International Pig Veterinary Society Congress*, Birmingham, UK, **3:** 15.

CARGILL, C., SKIRROW, S. and BANHAZI, T. (1996). The relationship between pig population size, stocking density, air quality parameters and pleurisy in pig herds. *Proceedings of the 14th IPVS Congress*, Bologna, Italy p.521.

CARGILL, C., BANHAZI, T. and CONNAUGHTON, I. (1998). The influence of air quality on production increases associated with all in all out management. *15th International Pig Veterinary Society Congress*, Birmingham, UK, **2:**248.

CARGILL, C., MADEC, F. and BANHAZI, T. (2000). Hygiene, health and production. *Proceedings of the Australian Association of Pig Veterinarians*, Perth pp.77-84.

CLARK, L., ARMSTRONG, C., SCHEIDT, A. and ALSTINE, W.V. (1993). The effect of *Mycoplasma hyopneumoniae* infection on growth in pigs with or without environmental constraints. *Swine Health and Production* **1:** 10-14.

CROWE, C., HARRIS, D.H., ELLIOT, L., WILSON, E. and WISEMAN, B. (1996). A possible relationship between low facility dust and endotoxin levels and improved growth rates in pigs reared by Isowean(SM). *Swine Health and Production 4*: 231-236.

CURTIS, S., KINGDON, D., SIMON, J. and DRUMMOND, D. (1976). Effects of age and cold on pulmonary bacterial clearance in the young pig. *American Journal of Veterinary Research* **37:** 299-301.

CURTIS, S., ANDERSON, C., SIMON J., JENSEN, A., DAY, D. and KELLEY, K. (1975). Effects of aerial ammonia, hydrogen sulfide and swine-house dust on rate of gain and respiratory tract structure in swine. *Journal of Animal Science* **41:** 735-739.

DONE, S., GRESHAM, A., CHENNELLS, D., WILLIAMSON, S., HUNT, B., TAYLOR, L., BLAND, V., JONES, P., ARMSTRONG, S., WHITE, R., DEMMERS, T., TEER, N. and WATHES, C. (2005). Clinical and pathological responses of weaned pigs to atmospheric ammonia and dust. *Veterinary Record* **78:** 87-97.

DONHAM, K. (1989). Relationships of air quality and productivity in intensive swine housing. *Agri-Practice* **10:** 15-26.

DONHAM, K. (1991). Association of environmental air contaminants with disease and productivity in swine. *American Journal of Veterinary Research* **52:** 1723-1730.

DONHAM, K. (1995). A review - the effects of environmental conditions inside swine housing on worker and pig health In 'Manipulating Pig Production V', pp. 203-219, ed P.D. Cranwell. (Australasian Pig Science Association: Werribee).

DONHAM, K., and LEININGER, J. (1984). Animal studies of potential chronic lung disease of workers in swine confinement buildings. *American Journal of Veterinary Research* **45:** 926-931.

DONHAM, K., HAGLIND, P., PETERSON, Y., RYLANDER, R. and BELIN, L. (1989). Environmental and health studies of farm workers in Swedish swine confinement buildings. *British Journal of Industrial Medicine* **46:** 31-37.

DRIESEN, S. (2003). Assessing the respiratory health of piggery workers in Australia. APL Report.

DRUMMOND, J., CURTIS, S. and SIMON, J. (1978). Effects of atmospheric ammonia on pulmonary bacterial clearance in the young pig. *American Journal of Veterinary Research* **39:** 211-212.

DRUMMOND, J., CURTIS, S., SIMON, J. and NORTON, H. (1980). Effects of aerial ammonia on growth and health of young pigs. *Journal of Animal Science* **50:** 1085-1091.

DRUMMOND, J., CURTIS, S., MEYER, R., SIMON, J. and NORTON, H. (1981). Effects of atmospheric ammonia on young pigs experimentally infected with Bordetella bronchiseptica. *American Journal of Veterinary Research* **42:** 963-968.

DUTKIEWICZ, J. (1997). Bacteria and fungi in organic dust as potential health hazard. *Annals of Agricultural and Environmental Medicine*. **4:**11-16

FOGELMARK, B., SJOSTRAND, M. and RYLANDER, R. (1994). Pulmonary inflammation-induced by repeated inhalations of beta(1,3)-d-glucan and endotoxin. *International Journal of Experimental Pathology* **75:** 85-90.

GONYOU, H., LEMAY, S. and ZHANG, Y. (1999). Effects of the Environment on Productivity and Disease. In 'Diseases of Swine, pp.1017-1027, eds B. Straw, S. D'Allaire, W. Mengeling, and D. Taylor, 8th edition, (Iowa State University Press).

GORDON, W. (1963). Environmental studies in pig housing IV. The bacterial content of air in piggeries and its influence on disease incidence. *British Veterinary Journal* **119:** 263-273.

GUSTAFSSON, G. (1999). Factors affecting the release and concentration of dust in pig houses. *Journal of Agricultural Engineering Research*. **74:**379-390

HOLYOAKE, P. (2002). Developing a safe environment for people working in pig production systems. Australian Pork Limited. Project no. 1618.

HOLYOAKE, P.K., DRIESEN, S., KOCH, S., DANIEL, P., FAHY, V., GOLEC, R. CARGILL, C., BANHAZI, T., PATTISON, P. and GALE, A. (2002). Ensuring a safe environment for people working in pig production systems. *Proceedings of the Australian Association of Pig Veterinarians*, Adelaide pp. 59-65

IKEGUCHI, A. (2001). Livestock Environment IV. *Proceedings of the 6th International Symposium*, Louiseville, Kentucky pp. 564-570

JANSEN, A. and FEDDES, J. (1995). Effect of airborne dust on health and performance of growing pigs *Canadian Agricultural Engineering* **37:** 211-216.

JERICHO, K. (1968). Pathogenesis of pneumonia in pigs. *The Veterinary Record* **82:** 507-517.

JOHNSON, R. (1997). Inhibition of growth by pro-inflammatory cytokines: an integrated view. *Journal of Animal Science* 75: 1244-1255.

JOLIE, R., BACKSTROM, L. and OLSON, L. (1996). Inflammatory changes in the respiratory tract of pigs induced by airborne contaminants present in swine confinement units. *Proceedings of the 14th IPVS Congress*, Bologna, Italy pp. 518.

JOLIE, R., BACKSTROM, L., OLSON, L. and CHASE, C. (1999a). Respiratory and systemic health parameters in pigs raised in a conventional farm or in isolation. *Swine Health and Production* **7:** 269-275.

JOLIE, R., BACKSTROM, L., OLSON, L. and CHASE, C. (1999b). A 15-week experimental exposure of pigs to airborne dust with added endotoxin in a continuous flow exposure chamber. *Canadian Journal of Veterinary Research* **63:** 129-137.

KLOOSTER, C.V.T., ROELOFS, P. and HARTOG, L.D. (1993). Effects of filtration, vacuum cleaning and washing in pig houses on aerosol levels and pig performance. *Livestock Production Science* **33:** 171-182.

LARSSON, K., EKLUND, A., HANSSON, L., ISAKSSON, B. and MALMBERG, P. (1994). Swine dust causes intense airways inflammation in healthy subjects. *American Journal Respiratory Critical Care Medicine* **150:** 973-977.

LEE, C., GILES, L., BRYDEN, W., DOWNING, J., OWENS, P., KIRBY, A. and WYNN, P. (2005). Performance and endocrine responses of group housed weaner pigs exposed to the air quality of a commercial environment. *Livestock Production Science*. **93:** 255-262.

LOPEZ, A., ALBASSAM, M., YONG, S., SHARMA, A., LILLIE, L. and PRIOR, M. (1987). Profiles of type-II pneumocytes in rats inoculated intratracheally with bacterial lipopolysaccharide. *American Journal of Veterinary Research* **48:** 1534-1539.

MARTIN, W.T., ZHANG, Y., WILLSON, P., ARCHER, T.P., KINAHAN, C. and BARBER, E.M. (1996). Bacterial and fungal flora of dust deposits in a pig building. *Occupational Environmental Medicine*. **53:**484-487

MICHEL, O., DUCHATEAU, J., PLAT, G., CANTINIEAUX, C., HOTIMSKY, A., GERAIN, J and SERGYSELS, R. (1995). Blood inflammatory response to inhaled endotoxin in normal subjects. *Clinical and Experimental Allergy* **25:** 73-79.

MOORE-COLYER, M. (1996) Effects of soaking hay fodder for horses on dust and mineral content. *Animal Science* **63:**337-342

MURPHY, T., and CARGILL, C. (2004a). The effects of indoor air pollutants on the health and production of growing pigs. *Pig News and Information* **25:**35N-44N.

MURPHY, T., and CARGILL, C. (2004b). *Proceedings of ISAH Conference*, Saint Malo, France pp. 207-208.

MURPHY, T., CARGILL, C. and CARR, J. (2000). The effects of stocking density on air quality. *16th International Pig Veterinary Society Congress*, Melbourne, Australia pp. 326.

PABST, R., and BINNS, R. (1994). The immune system of the respiratory tract in pigs. *Veterinary Immunology and Immunopathology* **43:** 151-156.

ROBERTSON, J. (1994). Ammonia, dust and air quality: quantifying the problem. *Pig Journal* **33:** 113-125.

ROBERTSON, J., WILSON, D. and SMITH, W. (1990). Atrophic rhinitis: the influence of the aerial environment. *Animal Production* **50:** 173-182.

RYLANDER, R. (1969). Alterations of lung defense mechanisms against airborne bacteria. *Archives of Environmental Health* **18:** 551-555.

SCHWARTZ, D., THORNE, P., JAGIELO, P., WHITE, G., BLEUER S. and FREES, K. (1994). Endotoxin responsiveness and grain dust-induced inflammation in the lower respiratory tract. *American Journal of Physiology* 267: L609-L617.

SEEDORF, J., HARTUNG, J., SCHRODER, M., LINKERT, K.H., PHILLIPS, V.R., HOLDEN, M.R., SNEATH, R.W., SHORT, J.L., WHITE, R.P., PEDERSEN, S., TAKAI, H., JOHNSEN, J.O., METZ, J.H.M., GROOT KOERKAMP, P.W.G., UENK, G.H. and WATHES, C.M. (1998). Concentrations and emissions of airborne endotoxins and micro-organisms in livestock buildings in *Northern Europe Journal of Agricultural Engineering Research*. **70:**97-109

SKIRROW, S., and CARGILL, C. (1994). Risk factors for pleurisy in pigs. PRDC Report

SKIRROW, S., CARGILL, C., NICHOLLS, R., MASTERMAN, N. and BANHAZI, T. (1995). Causes and consequences of pleurisy - how much does it cost? *Proceedings of the Australian Association of Pig Veterinarians* pp. 47-52.

STARK, K., PFEIFFER, D. and MORRIS, R. (1998). Risk factors for respiratory diseases in New Zealand pig herds. New Zealand *Veterinary Journal* **46:** 3-10.

STOMBAUGH, D., TEAGUE, H. and ROLLER, W. (1969). Effects of atmospheric ammonia on the pig. *Journal of Animal Science* 28: 844-847.

STRAW, B. (1991). Performance measured in pigs with pneumonia and housed in different environments. *Journal of the American Veterinary Medical Association* **198:** 627-630.

TAKAI, H., JACOBSON, L.D. and PEDERSEN, S. (1996). Reduction in dust concentration and exposure in pig buildings by adding animal fat in feed. *Journal of Agricultural Engineering Research*. **63:**113-120

TAKAI, H., NEKOMOTO, K., DAHL, P., OKAMOTO, E., MORITA, S. and Hoshiba, S. (2002). Ammonia contents and desorption from dusts collected in livestock buildings. *CIGR Ejournal* **4:** 1-11.

THEDELL, T.D., MULL, J.C. and OLENCHOCK, S.A. (1980). A brief report of gram-negative bacterial endotoxin levels in airborne and settled dusts in animal confinement buildings. *American Journal of Industrial Medicine*. **1:**3-7

THORNE, P.S., KIEKHAEFER, M>S>, WHITTEN, P. and DONHAM, K.J. (1992) Comparison of bioaerosol sampling methods in barns housing swine. *Applied and Environmental Microbiology* **58**:2543-2551

TURNER, L., GATES, R., WATHES, C. and AUDSLEY, E. (1997). Using fuzzy logic for assessment of dust and ammonia effects on swine respiratory disease. *Proceedings of the 5th international symposium of the American Society of Agricultural Engineers*, 2: 638-646.

URBAIN, B., GUSTIN, P., BEERENS, D. and ANSAY, M. (1996). Acute effects of endotoxin inhalation on the nasal mucosa on pigs: interaction with ammonia. *Proceedings of the 14th IPVS Congress*, Bologna, Italy p.519.

WATHES, C., DEMMERS, T., TEER, N., WHITE, R., TAYLOR, L., BLAND, V., JONES, P., ARMSTRONG, D., GRESHAM, A., HARTUNG, J., CHENNELLS, D. and DONE, S. (2004). Production responses of weaned pigs after chronic exposure to airborne dust and ammonia. *Animal Science* **78:** 87-97.

ZHANG, Y., TANAKA, A., DOSMAN, J., SENTHILSELVAN, A., BARBER, A., KIRYCHUK, S., HOLFELD, L. and HURST, T. (1998). Acute respiratory responses of human subjects to air quality in a swine building *Journal of Agricultural Engineering Research* **70:** 367-373.

Role of dietary non-starch polysaccharides in pig nutrition

W. J.J. Gerrits and M.W.A. Verstegen

Wageningen University and Research Centre, Wageningen, The Netherlands.

Introduction

The interest in the role of fibre in pig nutrition is increasing (Noblet and Le Goff, 2001). Fibre rich feed ingredients improve gut transit and reduce stomach ulcers (see Low, 1985). Dietary fibre (DF) or non-starch polysaccharides (NSP) are the component of plants that are resistant to hydrolysis by animal digestive enzymes and requires microbial enzymes for its digestion. Aspects of fibre or NSP have been reviewed by Bach Knudsen (2001) and will not be dealt with in this paper. In this paper, the components of dietary carbohydrates and DF, in relation to the commonly applied Van Soest method (Van Soest, 1963), will be discussed (Figure 1).

Feed ingredients differ widely in their NSP content, composition and digestibility (faecal disappearance). Some examples are shown in Table 1. During the last decade, many research groups have investigated the digestibility of the NSP fraction by pigs as affected by level of NSP intake, the composition of NSP or live weight of the pigs. Generally, the effect of NSP intake on its digestibility depends on the source (Schrama *et al.*, 1998; Rijnen, 2003). With regard to the effect of NSP composition, there is abundant information in the literature. Generally, the soluble part of the NSP fraction is more readily digested (Graham *et al.*, 1986; Noblet and Bach Knudsen, 1997; Noblet and Le Goff, 2001), whereas the insoluble part is not completely and more slowly digestible (Graham *et al.*, 1986; Noblet and Bach Knudsen, 1997). It has been well established that digestibility of NSP increases with development and thus with weight or the age of pigs (Fernandez and Jørgenson, 1986; Noblet and Shi, 1994; Le Goff *et al.*, 2002). This may be related to the increased mean retention time in the pig (Le Goff *et al.*, 2002).

In this review some actions of NSP are briefly summarised. Subsequently, attention focuses on a description of studies on some aspects of NSP on physical activity, behaviour and possible implications for energy evaluation and for emission to the environment are discussed.

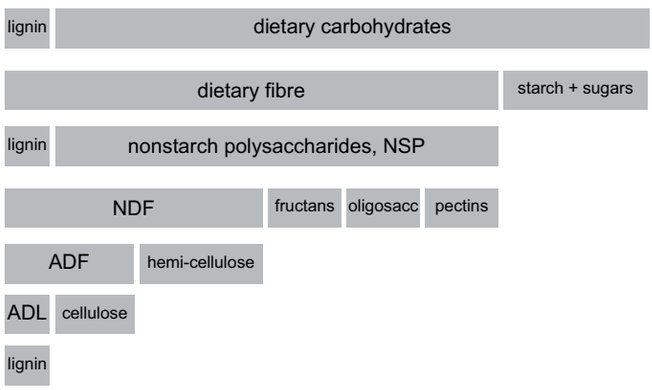


Figure 1. *Schematic representation of dietary carbohydrate, fibre and nonstarch polysaccharide composition with reference to the Van Soest analysis (NSP = non starch polysaccharides; NDF = neutral detergent fibre; ADF = acid detergent fibre; ADL = acid detergent lignin*

Table 1 Examples of fibrous feed ingredients, their NSP content and digestibility in growing pigs.

	NSPg/kg DM	NDFg/kg DM	ADFg/kg DM	ADLg/kg DM	Total tract digestibility NSP, %
Sugar beet pulp[a]	705	447	215	30	65
Wheat bran[a]	432	425	127	126	44
Maize bran[a]	482	495	42	18	43
Soy bean hulls[b]	758	626	511		55
Wheat straw[c,d]	850	815	516	85	16

Data from [a]Le Goff *et al.* (2002); [b]CVB, 2000; [c]Oosting, (1993); [d]Chabeauti *et al.* (1991), only digestibility.

Actions of non-starch polysaccharides

The variation in fermentation properties of various NSP has many consequences for their mode of action in the GI tract. A study by Houdijk (1998) showed the importance of GIT location for the fermentation of the fructo-oligosacharides (FOS) used as pre-biotics. Various approaches have been used to study - Minekus *et al.* (1999) used an *in vitro* model to simulate the transport and breakdown of feed components, such as chyme hydrolyses, in the various parts of the GIT of humans in combination with absorption of the hydrolysed components. Using the large intestinal section of the model, they showed that some NSP, such as inulin, soy polysaccharides and FOS, were already partly fermented at the beginning of the large intestine. Inulin and arabic gum were completely fermented mid-way along the large intestine and resistant starch and α-cellulose were fermented only in the distal part of the large intestine. Houdijk (1998) discussed why a continuous fermentation throughout the large intestine may be beneficial. In each segment of the GIT there has to be an appropriate carbon to nitrogen (C:N) ratio (Borg-Jensen, 1993).

There is considerable evidence that under non-optimal conditions of husbandry, NDF may have a beneficial effect on young animals. Bauer *et al.* (2001) performed a large study investigating the fermentation characteristics of a range of different carbohydrates. Using a modified cumulative gas production technique (Williams *et al.*, 2001b), fermentability was assessed according to the kinetics of gas production, Volatile fatty acids (VFA) and ammonia production, and pH of the medium at the end of fermentation. The inoculum was obtained from pigs that did not have any of the substrates to be tested as an ingredient of their diet. Table 2 shows the half-time (T½) of maximum gas production (a measure of fermentation kinetics), and the ratio of branched to straight-chain fatty acids (BCR) of various tested carbohydrates as substrates in vitro. Some other actions of NSP have been recently reviewed by Bach Knudsen (2001), Longland and Low (2001), Pluske *et al.* (2001), Wenk (2001), Mosenthin *et al.* (2001) and Williams *et al.* (2001). Briefly, the physical properties of NSP, in combination with its chemical composition, determine its physiological effect.

Table 2. $T_{½}$ (half-time for asymptote of gas production -h) during fermentation of some products and ratio of branched to straight-chain fatty acids (BCR) (Bauer *et al.*, 2001).

Feed ingredient	T ½ (h)	BCR
Arabic gum	24.5^{ab}	0.079^{bc}
Guar gum	16.4^{b}	0.082^{c}
Xylan	29.5^{a}	0.112^{b}
FOS	16.8^{b}	0.062^{c}
TOS (Trans-galacto-oligosaccharide)	16.1^{b}	0.137^{b}
Sugarbeet pulp	15.1^{b}	0.094^{c}
Jerusalem artichoke inulin	22.9^{ab}	0.127^{b}
Chicory inulin	21.6^{ab}	0.143

a,b,c Superscripts which differ in the same column are significantly different ($P<0.05$)

The data in Table 2 illustrate the large differences in the fermentability of different carbohydrates, both in terms of the rate of fermentation and also in the formation of end-products. From this it is thought that a stable supply of NSP throughout the distal part of the GI tract may help to maintain a stable microbiota. This prebiotic function of fermentable NSP has been the basis for a considerable research effort into its potential role as a replacement for the preventive use of antibiotics in animal diets. While most authors claim positive effects of fermentable NSP on the stability of the intestinal microflora or an improved gut health (Williams *et al.*, 2001a), others report negative effects of increased intakes of NSP on disease resistance (Pluske *et al.*, 2001; swine dysentery). Despite the latter, the effects of NSP on the microflora and gut health are generally perceived as positive. These assumed positive affects are all based on lower numbers of pathogens and lower pH. It is, however, important that the modes of action of different types of NSP are unravelled to successfully apply some of the NSP as an alternative for antibiotics.

Soluble NSP can increase viscosity in the upper part of the gastrointestinal tract, thus reducing the gastric emptying and passage rate. As discussed by Mosenthin *et al.,* (2001), however, gastric emptying is not a simple function of soluble fibre intake. Moreover, the retention time in the stomach and small intestine is much shorter compared with that of the large intestine. Therefore, changes in the rate of gastric emptying are not necessarily reflected in total retention time. Insoluble fibrous components are known to increase passage rate in the large intestine by physical stimulation and increase of bulk. After introduction of a significant quantity of a fermentable NSP in a pig's diet, the change in substrate for the intestinal microflora causes a shift in microbial activity (Pluske *et al.*, 2001; Williams *et al.*, 2001a). It seems, however, that fermentable carbohydrates alter the activity of the existing microbial population, rather than changing the presence of specific bacterial strains. The latter appears to be largely determined by the genotype of the animal (B.A. Williams, personal communication).

The end products of fermentation, mainly VFA and ammonia, can be readily absorbed through the intestinal wall and the short-chain fatty acids contribute to the energy supply of the animal. Furthermore, fermentation end products can have specific positive (e.g. stimulation of colonocyte proliferation by butyric acid (Brouns *et al.*, 2002)), or negative (e.g. cytotoxic effects of ammonia) effects in the animal (Williams *et al.*, 2001a).

Dietary fibre and physical activity

The interest in effects of NSP on pig behaviour and physical activity originates from work done with sows, which started in the 1970s (Fraser, 1975; Robert *et al.*, 1993; Brouns and Edwards, 1994). Generally, feedstuffs that induced a reduction in physical activity had a high fibrous content. Schrama *et al.* (1996) quantified the effect of NSP from sugar-beet-pulp silage (SBPS) on the energy expenditure on physical activity in group-housed pigs (Table 3). In this experiment, tapioca was exchanged for SBPS, based on its calculated net energy content. Heat production was measured using indirect calorimetry and physical activity was recorded using radio-Doppler meters as described by Wenk and van Es (1976). Schrama *et al.* (1996) found that the energy expenditure on physical activity in the SBPS group was reduced by 24%, mainly occurring during the dark phase of the day. Later Schrama *et al.* (1998) exchanged tapioca for SBPS in four steps (0, 5, 10, and 15% SBPS on a dry matter basis). In this experiment (Table 3), they found that activity-related heat production decreased by 3.9 kJ/g of fermented DF intake from SBPS. Schrama *et al.* (1998) hypothesised the reduction in physical activity to be caused by either: (i) the fermentative activity in the gastrointestinal tract, mainly the hindgut. This could be related to either fermentation end products, or by a more gradual availability of the dietary energy within the day; (ii) physical satiety, caused by increased dietary bulkiness, and/or iii) it could be a specific effect related to the chemical composition of the NSP fraction of SBPS.

To investigate the latter two hypotheses, Schrama and Bakker (1999) added wheat straw to a diet containing either gelatinised cornstarch or raw potato starch in a 2 x 2 factorial design (Table 3).

Table 3. Effects of various treatments on activity related heat production of growing barrows of 40-60 kg live weight.

Reference	Treatment	GE intake[a]	ME:GE[b]	P[e]	Total HP[c]	P[e]	Activity related HP[d]	P[e]
Schrama *et al.* (1996)	Control (13% tapioca)	1133	79	†	618	NS	118	†
	SBPS[h], 17% (no tapioca)	1172	78		631		90	
Schrama *et al.* (1998)[f]	Control (36% tapioca)	1268	77	**	673	NS	106	†
	SBPS[h], 17% (19% tapioca)	1280	74		665		80	
Schrama & Bakker (1999)[g]	Gelatinised maize starch (35%)	1325	78	**	659	NS	122	**
	Native potato starch (35%)	1345	71		644		100	
Schrama & Bakker (1999)[g]	No added wheat straw	1253	80	**	656	NS	112	NS
	15% added wheat straw	1420	70		647		111	
Rijnen (2003)[g]	Individual housing	1168	85	**	657	NS	71	NS
	Group housing	1192	81		641		79	
Rijnen (2003)[g]	Gelatinised cornstarch (%)	1175	85	**	653	NS	80	†
	Dried sugarbeet pulp (%)	1185	81		645		69	

[a]Gross energy intake, $kJ.kg^{-0.75}.d^{-1}$; All animals were fed restrictedly; [b]Metabolisability of the dietary gross energy intake, %; [c]Total heat production, $kJ.kg^{-0.75}.d^{-1}$, measured by indirect calorimetry; [d]Activity related heat production, $kJ.kg^{-0.75}.d^{-1}$; [e]Probability: NS, $P > 0.10$; †, $P < 0.10$; *, $P < 0.05$; **, $P < 0.01$; [f]Statistical significances taken from regression analyses of 0, 5, 10 and 15% SBPS on dry matter basis; this table only reports the two extreme treatments; [g]Means taken from least squares estimated from a 2 x 2 factorial design; interactions on the reported parameters were not present; [h]SBPS = sugar beet pulp silage.

They concluded that replacing enzymatically degradable starch (gelatinised maize starch) by a starch source known to be highly resistant to enzymatic degradation (raw potato starch) also reduced energy expenditure on physical activity. The amount of starch fermented in the hindgut was not quantified. Dietary bulkiness (added wheat straw) did not affect energy expenditure on physical activity in this experiment. There were no interactions found between type of starch and dietary bulkiness, except for methane emission. Methane emission was highest at the groups of pigs receiving both fermentable starch and milled straw, indicating that fermentation of resistant starch is affected by straw, even though the straw addition did not contribute to ME intake.

With the effects of NSP on physical activity being quantitatively significant, it became important to investigate whether housing systems could affect the potential of the dietary fibrous fraction to reduce physical activity. Rijnen *et al.* (2003) investigated the effect of individual as compared to group housing on the potential of NSP from sugar-beet pulp (SBP) to reduce physical activity. They hypothesised that the reduction in physical activity, obtained by replacing gelatinised maize starch by digestible NSP from SBP, would be lower when pigs are housed individually in metabolism crates compared with conventional group housing systems. This hypothesis was based on 1). the physical restriction of pigs housed in metabolism crates, decreasing the opportunity for altering physical activity, and 2). the absence of social interactions between pigs when individually housed. Results from this study (Table 3), however, indicated an absence of this hypothesised interaction. Individual housing numerically reduced energy expenditure on physical activity when compared with group housing, but not significantly. Dried SBP reduced energy expenditure on physical activity in a similar way to the studies of Schrama *et al.* (1996, 1998).

In summary, inclusion of sugar pulp in diets for growing pigs reduces energy expenditure on physical activity. This effect is not specific for SBP, as it was also observed for resistant starch. The effect is likely due to fermentation processes, either directly (e.g. due to changes in rate of passage and in physical conditions) or indirectly (by absorbed fermentation end products) and not to dietary bulkiness (by adding wheat straw). It should be further investigated whether rate of fermentation and or products of fermentation are related to the effects found. The reduction in physical activity was observed for both individually and group-housed pigs.

Dietary fibre and pig behaviour

An interesting question is whether NSP also affects the behaviour of pigs, which in turn can be used to assess their welfare. The supposed positive effect of NSP on behaviour is the basis for the European guidelines on inclusion of NSP in diets for various species (sows, veal calves). Therefore, behavioural observations are essential. The observations described above illustrate the effect of NSP on physical activity, and have been made in sows as well as in growing pigs. For example, Ramonet *et al.* (1999) found that increased crude fibre (CF) content of the diet reduced standing activity and non-feeding oral behaviour. They hypothesised that a change in feeding motivation was a driving force behind these observations. However, they found no effect of NSP from SBP and wheat bran on feeding motivation, which was measured using operand conditioning (Ramonet *et al.*, 2000). Danielsen and Vestergaard (2001) recently concluded in a large-scale study that both SBP and a mixed fibre diet reduced activity in first parity sows. Aggressive behaviour was reduced by SBP, but not by the mixed fibre diet. De Leeuw *et al.* (2004) studied how abnormal stereotyped behaviour and activity in restrictedly fed sows was related to fermentation in the gastrointestinal tract. He found that fermentation, induced by increased intakes of NSP from SBP reduced stereotyped behaviour and physical activity, and observed more stable blood glucose levels compared with low NSP diets. They hypothesised that stabilisation of blood glucose and insulin levels may indicate a prolonged feeling of satiety. Rijnen *et al.* (2003) also observed a reduction in physical activity when dietary starch was replaced by NSP from SBPS. Sows fed 20 or 30% SBPS spent less time and energy on physical activity than sows fed 0 or 10% SPBS.

For growing pigs, information concerning effects of NSP on behaviour is scarce. Below, the results of a pilot experiment on the effect of replacing starch with NSP from SBP on the behavioural and physiological responses of pigs in an open field test are presented. An open field test, such as social isolation in an unfamiliar environment, elicits complex behaviour patterns in various animal species that may reflect, among other things, exploratory motivation and fear (e.g. Andersen *et al.*, 2000; Candland and Nagy, 1969). For group-housed pigs, isolation from their pen mates appears to be the most stressful aspect of the test (Fraser, 1974).

Twenty-six of the group-housed pigs on either a SBP or a starch-based diet from the study of Rijnen *et al.* (2003) (Table 3) were subjected to an open field test (Doornhegge, Van den Borne, Rijnen and Bolhuis, unpublished data). Briefly, pigs were exposed to the dietary treatments for four weeks and then individually introduced into an open field of 4.8 x 5.5 m for 20 minutes. Ten minutes after the start of the test, a novel object was introduced into the open field. Pig behaviour was recorded using focal sampling. Saliva samples were taken from all pigs immediately before and after the open field test and analysed for cortisol. Heart rate was monitored continuously during the open field test using Polar devices (Polar Electro OY, Kimpele, Finland). Pigs were accustomed to the experimental procedures prior to the start of open field test.

Table 4. Effect of replacing starch by NSP from SBP on behavioural and physiological responses of growing pigs in an open field test (selected parameters)[a].

Variable	Starch	Sugar-beet pulp	P value
Posture and locomotion			
Locomotion (% of time)	27.4	24.4	0.06
Standing (% of time)	69.9	73.6	NS
Other behaviours			
Nosing wall/floor (% of time)	37.7	38.2	NS
Escape behaviour (% of time)	2.8	0.9	0.10
Vocalisations, latency (s)	60.0	102.0	0.02
Vocalisations, frequency	258.9	200.4	NS
Defecation, latency (s)	355.0	283.0	NS
Increase in cortisol during test (ng/ml saliva)	1.3	1.0	NS
Heart rate during test (bpm)	129.2	123.9	0.11
Novel object			
Snout contact, latency (s)	31.1	201.3	0.11
Snout contact (% of time)	12.6	6.5	0.06
Change in heart rate (bpm)	16.2	15.9	NS

[a]From Doornhegge, van den Borne, Rijnen & Bolhuis, unpublished data.

Selected parameters are presented in Table 4. Interestingly, pigs on the SBP diet not only displayed a reduced activity in their home pen (see the results from the study of Rijnen *et al.* (2003) (Table 3), but also tended to show a lower locomotor activity than starch-fed pigs during the open field test. This reduced physical activity of SBP pigs was not at the expense of general exploratory behaviour, as diet did not affect the percentage of time spent on nosing the walls or floor of the open field. The other observations reported in Table 4 show that SBP pigs spent less time exploring the novel object than starch-fed pigs. This could indicate that SBP pigs were more fearful or less motivated to investigate the object. Also, additional data analyses revealed no signs of a more fearful reaction of SBP pigs to the novel object. For instance, high amplitude calls, such as squeals and screams, which are generally associated with fear-eliciting situations (Kiley, 1972), were not particularly occurring in response to introduction of the novel object.

In summary, these results illustrate that NSP from SBP reduces physical activity, but not at the expense of explorative behaviour, and escape behaviour of pigs in an unfamiliar environment. Locomotion, escape behaviour and vocalisations in an open field have been related to the level of 'excitement', arousal, 'reactivity' or 'emotionality' of an individual pig during the test (Buchenauer, 1990; Fraser, 1974; Von Borell and Ladewig, 1992). Locomotion accompanied by escape attempts has also been interpreted as flight motivation (cf. Thodberg *et al.*, 1999). Pigs fed the SBP diet appeared less aroused by the test situation and less motivated to escape from the environment, which may indicate reduced levels of stress or fear. Alternatively, although a link between SBP or other NSP sources in the diet and feeding motivation is not always found (Meunier-Salaün *et al.*, 2001; Ramonet *et al.*, 2000; Whittaker *et al.*, 1998, 1999), the lower locomotion and reduced attention for the novel object of SBP pigs, as compared to starch-fed pigs, could be explained in terms of feeding motivation and foraging behaviour.

In rats, restricted feeding and food deprivation, which both enhance feeding motivation, have been reported to increase locomotion in open field tests (Heiderstadt *et al.*, 2000), a measure of activity and exploration in this species (Walsh and Cummins, 1976) and exploratory behaviour directed towards objects (Kamback, 1966), respectively. Also, in pigs, physical activity, when expressed as a percentage of maintenance energy expenditure, decreased when feed intake increased (Susenbeth and Menke, 1991). It should be noted, however, that in the open-field test described above, pigs on both dietary treatments were fed at similar levels of intake. Further research, which should include thorough (behavioural) observations on group-housed pigs, is necessary to elucidate how and why a SBP diet affects behavioural responses and, possibly, welfare of pigs. Additionally, it remains to be investigated if the results presented are related to fermentable NSP in general, or are specific for NSP from SBP.

The energy value of dietary NSP for growing pigs

The energetic evaluation of NSP has been of interest to several research groups, and the results of research efforts are widely adopted in practice. Dietary fibre is usually regarded as a poor energy source in diets for grower pigs, especially due to the low digestibility of NSP when compared with starch (Table 1; with faecal starch disappearance usually close to 100%). With regard to the energetic utilisation of digested NSP, an efficiency of around 70% of that of starch is generally adopted (CVB, 2000; Noblet and Le Goff, 2001; Longland and Low, 2001; Bach Knudsen, 2001). This assumption is usually based on biochemical calculations, classical energy evaluation studies using indirect calorimetry or infusion studies (Just *et al.*, 1983; Noblet *et al.*, 1994, Jørgenson *et al.*, 1996, 1997). As illustrated throughout the

present paper, however, NSP potentially reduces the energy expenditure on physical activity, thereby saving dietary energy for other purposes. In this section, the Dutch net energy (NE) system (CVB, 2000) is briefly explained as a representative example of a NE system. Subsequently, the implications of the previously mentioned effects on physical activity for the net energy value of NSP are discussed.

The Dutch net energy system for growing pigs

The Dutch NE system (described by CVB, 2000) can be characterised in three steps:

- The Dutch NE system is a marginal feed evaluation system. This means that the NE value (NEv, in MJ/kg) of a feedstuff indicates how much the energy retention of a pig will increase when the intake of that feedstuff increases with one kg. It implies that the energy expenditure on maintenance processes is constant.
- The first step within the system is the correction for faecal losses. The system uses (apparent faecal) digestible nutrients, and thereby corrects for differences in nutrient digestibility between feedstuffs. The nutrients considered are digestible crude protein, digestible crude fat, enzymatically digestible starch and sugars and fermentable DF (defined as dry matter-[ash+protein+fat+starch+sugars]).
- The second step is the conversion of digestible nutrients into net energy NE (i.e. energy retention). For this conversion, fixed partial efficiencies for each nutrient are assumed. Some of the values used by the NE system are presented in Table 5.

Obviously, a NE system has some advantages over DE or ME based systems. The main advantage over both other systems is that the energetic utilisation is made dependent on the nutrient composition of the feedstuff.

Table 5. Enthalpy and NE values for digestible nutrients used in the Dutch net energy system (based on CVB, 2000).

Nutrient	Enthalpy (MJ/kg)	NE value (MJ/kg)	Partial Efficiency (NE/ enthalpy, %)
Digestible crude protein	23.6	10.8	46[b]
Digestible crude fat	39.3	36.1	92
Enzymatically digestible starch	17.5	13.5	77
Enzymatically digestible sugars[a]	15.8	12.2	77
Fermentable DF	17.5	9.5	54[c]
Fermentable starch	17.5	9.5	54[c]

[a]sugars expressed in glucose equivalents [c]Including energy losses as combustible gases [b]Including energy losses as urinary urea

There are, however, serious drawbacks of a NE system as well. These include 1). the assumption that maintenance energy expenditure is constant and can therefore be bypassed by assigning marginal NE values to feedstuffs. There is abundant evidence of factors related to animal, environment or diet that affect biological processes that are considered to be a part of maintenance energy, illustrating the weakness of this assumption. Especially, dietary factors that affect maintenance energy can affect the true NE value of a feedstuff. Examples are anti-nutritional factors in the diet and the above-mentioned effects of NSP on physical activity; 2). the absence of biological mechanisms in the system that allow representation of interactions, for example (but not only) at the level of digestion. Bakker (1996), for instance, showed that increasing NSP depressed fat digestibility. Inclusion of this type of interaction is impossible to represent in a system assigning a feeding value to individual feedstuffs; 3). there are some difficulties in connecting feeding values with animal requirements. Although animal requirements are not part of a feed evaluation system, there has to be a close relation between the two. Feeding values are assigned to individual feedstuffs and are therefore expressed in units/kg feedstuff. Nutrient requirements, however, are ideally expressed in units/d but at best are expressed in units/kg of compound feed (not feedstuff). In practice, nutrient requirements and feeding values are matched using least cost formulation software, based on linear programming techniques.

Although true theoretically, for practical reasons, the NE system has proven its value in several countries, and the problems mentioned above are not specific for NE systems.

The NE value of dietary NSP

As shown in Table 3, energy expenditure on physical activity can be reduced by fermentable DF. Schrama *et al.* (1998) calculated that the decrease in energy expenditure on physical activity amounted to 3.9 kJ NE per gram of fermentable NSP intake. In their experimental set-up, this was similar to about 8% of the estimated ME requirements

for maintenance. The NE value for NSP, calculated from these data was 14.1 MJ/kg. Apparently, the reduction in physical activity induced by SBPS completely compensated for the lower theoretical NE value of DF (9.5 MJ/kg) when compared with starch (13.5 MJ/kg). As discussed previously, the data of Schrama and Bakker (1999) demonstrated a similar reduction in physical activity for resistant starch as for SBP. The NE value of resistant starch (in terms of fate in the GI tract being like a NSP source), however, cannot be calculated because the distinction between enzymatically digested and fermented starch could not be made. It can only be approached by measuring starch disappearance at the terminal ileum, assuming that no fermentation occurs in the small intestine. Data from experiments of Rijnen (2003) showed that the NE value of fermented NSP from solvent-extracted coconut meal and soybean meal was numerically higher than the theoretical value of 9.5 MJ/kg, used in the Dutch NE system (see Table 5), but not significantly different. In these experiments, no effect of fermented NSP from these sources on physical activity was found. Considering the wide variation in chemical composition of NSP sources from different botanical origin ic monocotyledons versus dicotylodons it is questionable whether one NE value for fermentable NSP is sufficiently precise for any good feed evaluation system. There can be quite big differences in estimations of the NE value of fermentable NSP between various fibre sources, even though similar procedures and equipment were applied (Table 5).

Three ways to account for diet-induced effects on physical activity in NE systems

Physical activity is usually considered to be a part of maintenance energy expenditure, although Susenbeth and Menke (1991) and Verstegen *et al.* (1982), for example, showed it to depend on feeding level and age, respectively. Theoretically, the design of most NE systems for growing pigs, being marginal systems and therefore assuming maintenance energy to be constant, does not allow for the diet-induced effects on maintenance energy expenditure. There are (at least) three ways to account for the diet-induced effects on physical activity in NE systems:

- One could argue that these effects should be accounted for on the requirement side rather than on the feed evaluation side. The advantage of this approach is that it bears a closer relationship with physiological processes occurring in the animal. There will, quite likely, be a biological explanation for these effects on physical activity, related to (the timing and/or type of) nutrient absorption, for example VFAs or other products that are produced during fermentation. This needs further investigation. The practical disadvantage is that, within the current systems, it is very unattractive to deal with animal requirements varying with diet-induced effects on physical activity. The use of biologically based, mechanistic models within feed evaluation could be a solution to this problem.
- Another possibility is to separate the effects of physical activity on NE (positive or negative) from the NE value of a feedstuff. In the case of SBPS (Schrama *et al.*, 1998), the NE value of the fermentable NSP could be similar to that of other feedstuffs, but a positive correction could be made on NE for physical activity, thus assigning a higher total NE value to SBPS. Analogously, other diet-dependent energy consuming processes could be dealt with, for example the energetic costs of digestion. A serious drawback with this approach is that it would involve detailed knowledge on the factors triggering processes like physical activity and energetic costs of digestion.
- The third, and most practical solution is to assign feedstuff-specific NE values to the fermentable NSP fraction. This can be done, as long as the NE value is truly dependent on feedstuff characteristics and independent from its nutrient composition. If, for example, the reduction of physical activity by SBP depends on the genotype, housing system (not the case for individual *vs.* group housing; Rijnen *et al*, unpublished) or feeding level, this approach is not valid.

Considering the above, it is important to increase knowledge of the biological mechanisms behind the effects of NSP on physical activity. Quite likely, it depends on the chemical and physical properties of the NSP fraction and also the fermentative capacity of the microbiota. For these reasons, further characterisation of the DF fraction of the diet in an NE system is essential.

Dietary NSP and environment

With regard to environment, research focus in the EU is on reducing total N excretion and ammonia emission from animals. To date, emphasis with regard to environment and nutrition has been on reducing total N excretion in manure (Henry and Dourmad, 1993). Recent studies have shown that, in pigs, ammonia emission can be reduced by including fermentable NSP in the diet. It is well known that by allowing proper microbial fermentation in the large intestine, more microbial biomass will be present in the excreta and less in urine (Canh *et al.*, 1998), while ileal disappearance of N may remain unaltered (Bakker, 1996). So it can be expected that by combining both a reduced level of N in feed and an increase in fermentable NSP this will result in a shift in N excretion to from urinary to faecal excretion. In addition, it has been reported that an increase in fermentable NSP will decrease the pH in excreta and reduce ammonia emission from slurry (see Figure 2, Canh *et al.*, 1998).

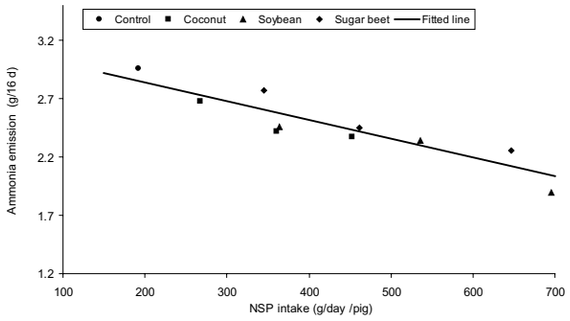


Figure 2. *Ammonia emission from the slurry as affected by daily intake of NSP (Canh et al., 1998).*

Conclusions

Results from various studies demonstrate that, compared with enzymatically digestible starch, inclusion of NSP in pig diets causes changes in the GI tract with regard to retention time, rate and type of fermentation and physical conditions. This can alter microbial activity and this has consequences for pH and number of pathogens in the gut. In sows and in growing pigs, some (e.g. SBP and resistant starch), but not all NSP sources reduce energy expenditure on physical activity. The effect could not be demonstrated by an increase in gut fill achieved by adding wheat straw to the diet. The reduction in physical activity was observed for both individually and group-housed pigs. A behavioural study (open-field) revealed that pigs fed SBP showed reduced locomotor activity, but not at the expense of explorative or escape behaviour.

Further research, which should include (behavioural) observations on group-housed pigs, is necessary to elucidate how and why a NSP diet affects behavioural responses and, possibly, the welfare of pigs. Additionally, it remains to be investigated if the results found in literature are related to fermentable NSP in general, or are specific for NSP from SBP.

The observed effects of fermentable NSP on physical activity have an impact on its feeding value. Accounting for the effect of NSP from SBP on, for example, physical activity in a NE system would increase its NE value to a value close to that of starch. Dietary effects on physical activity are, however, difficult to account for in a NE system, because they are generally considered to be a part of maintenance energy expenditure. It is, however, important to account for this effect in a feed evaluation system, because it represents an economic value of a feedstuff, and several options are discussed. For future feed evaluation, an increase in knowledge on the biological mechanisms behind the effects of DF on physical activity are important. Quite likely, these effects depend on the chemical and physical properties of the NSP fraction.

References

ANDERSEN, I.L., BOE, K.E., FOEREVIK, G., JANCZAK, A. M., BAKKEN, M. (2000). Behavioural evaluation of methods for assessing fear responses in weaned pigs. *Applied Animal Behaviour Science* **69**(3): 227-240.

BACH KNUDSEN, K.E., (2001) The nutritional significance of 'dietary fibre' analysis. *Animal Feed Science and Technology* **90**: 3-20.

BAKKER, G.C.M. (1996) Interaction between carbohydrates and fat in pigs. PhD thesis Wageningen University, Wageningen, the Netherlands, 193pp.

BAUER E., WILLIAMS B.A., VOIGT C., MOSENTHIN R. and VERSTEGEN M.W.A. (2001). Microbial activities of faeces from unweaned and adult pigs, in relation to selected fermentable carbohydrates. *Animal Science* **73**:313-322

BORG JENSEN, B. (1993). The possibility of manipulating the microbial activity in the digestive tract of momogastric animals paper 44th EAAP Meeting Aug 16-19 Foulum Denmark p1-15

BROUNS, F., EDWARDS, S.A. (1994). Social rank and feeding behaviour of group-housed sows fed competitively or *ad libitum*. *Applied Animal Behaviour Science* **39**: 225-235.

BROUNS, F., KETTLITZ, B., and ARRIGONI, E. (2002). Resistant starch and the butyrate revolution. *Trends in Food Science and Technology* **13**:251-261.

BUCHENAUER, D. (1990). Genetic behavioral aspects of agricultural animals (abstract). Deutsche Tierarztliche Wochenschrift **97**(6): 247-249.

CANH, T.T., VERSTEGEN M.W.A., Aarnink and A.J.A. Schrama J.W. (1997). Influence of dietary factors on nitrogen partitioning and composition of urine and feces of fattening pigs. *J. Animal Science* **75**:700-706 .

CANDLAND, D.K. and NAGY, Z.M. (1969). The open field: some comparative data. *Annals New York Academy of Sciences* **159**(3): 831-851.

CHABEAUTI, E., NOBLET, J. and CARRÉ, B. (1991). Digestion of plant cell walls fromo different sources in growing pigs. *Animal Feed Science and Technology* **32**: 207-213.

CVB (2000) Veevoedertabel. Centraal Veevoederbureau Nederland, Lelystad, The Netherlands.

DANIELSEN, V., VESTERGAARD, E.M. (2001). Dietary fibre for pregnant sows: effect on performance and behaviour. *Animal Feed Science and Technology* **90**: 71-80.

LEEUW J A DE, ZONDERLAND J.J.,ALTENA. SPOOLDER H.A.M., JONGBLOED A.W. and VERSTEGEN M.W.A. (2005). Effects of levels and sources of dietary fermentable Non Starch Polysaccharides on blood glucose stability and behaviour of group-housed pregnant sows. *Applied Behaviour Science* **91**:15-29.

FERNANDEZ, J. A. and JØRGENSEN, J.N. (1986). Digestibility and absorption of nutrients as affected by fibre content in the diet of the pig. Quantitative aspects. *Livestock Production Science* **15**(1): 53-71.

FRASER, D. (1974). The vocalizations and other behaviour of growing pigs in an 'open field' test. *Applied Animal Ethology* 1: 3-16.

FRASER, D. (1975) The effect of straw on the behaviour of sows in tether stalls. *Animal Production* **21**: 59-68.

GRAHAM, H., HESSELMAN, K., AMAN, P. (1986). The influence of wheat bran and sugar beet pulp on the digestibility of dietary components in a cereal-based pig diet. *Journal of Nutrition* **116:** 242-251.

HEIDERSTADT, K.M., MCLAUGHLIN, R.M., WRIGHT, D.C., WALKER, S.E. and GOMEZ-SANCHEZ, C.E. (2000). The effect of chronic food and water restriction on open-field behaviour and serum corticosterone levels in rats. *Laboratory Animals* **34:** 20-28.

HOUDIJK, J.G.M., (1998). Effects of non-digestible oligo sacharides in young pig diets. PhD Thesis Wageningen University, The Netherlands.

JØRGENSEN, H., ZHAO, X.Q., EGGUM, B.O. (1996). The effect of dietary fibre and environmental temperature on the development of the gastro-intestinal tract, digestibility, degree of fermentation in the hindgut and energy metabolism in pigs. *British Journal of Nutrition* **75**: 365-378.

JØRGENSON, H., LARSEN, T., ZHAO, X.Q. and EGGUM, B.O. (1997). The energy value of short-chain fatty acids infused into the caecum of pigs. *British Journal of Nutrition* **77**: 745-756.

JUST, A., FERNANDEZ, J.A. and JØRGENSEN, H. (1983). The net energy value of diets for growth in pigs in relation to the fermentative processes in the digestive tract and the site of absorption of the nutrients. *Livestock Production Science* **10**: 171-186.

KAMBAK, M.C. (1966). Effects of food deprivation on object and nonobject directed behavior in the rat. *Psychon Sci* **5**:47-53.

KILEY, M. (1972). The vocalizations of ungulates, their causation and function. *Zeitschrift für Tierpsychologie* **31**: 171-222.

LE GOFF, G., VAN MILGEN, J. and NOBLET, J. (2002). Inluence of dietary fibre on digestive utilization and rate of passage in growing pigs, finishing pigs and adult sows. *Animal Science* **74**:503-515.

LONGLAND, A.C. and LOW, A.G. (2001). Prediction of the energy value of alternative feeds for pigs. In: Recent Developments in Pig Nutrition [Garnsworthy, P.C. and Wiseman, J., eds] vol 3, pp 239-260.

LOW, A.G. (1985). Role of dietary fibre in pig diets. In: Recent advances in Animal Nutrition, pp 87-112. Haresign, W., Cole, D.J.A., eds), Butterworths, London.

MEUNIER-SALAÜN, M.C., EDWARDS and ROBERT, S. (2001). Effect of dietary fibre on the behaviour and health of the restricted fed sow. *Animal Feed Science and Technology* 90(1-2):53-69.

MINEKUS, M. (1998). Development and validation of a dynamic model of the gastrointestinal tract. Ph D Thesis. University of Utrecht The Netherlands.

MOSENTHIN, R., HAMBRECHT, E. and SAUER, W.C. (2001). Utilization of different fibres in piglets feeds. In: Recent Developments in Pig Nutrition [Garnsworthy, P.C. and Wiseman, J., eds] vol 3, pp 293-322.

MUL, A.J. (1997). Application of oligosacharides in animal feeds. In: Hartemink (1997). Non digestible oligosacharides. P. 106-124.

NOBLET. J. and BACH KNUDSEN, K.E. (1997). Comparative digestibility of wheat, maize and sugar beet pulp nonstarch polysaccharides in adult sows and growing pigs. Proceedings of the 7th international symposium on digestive physiology in pigs, Saint Malo, France, 26-28 May 1997, 571-574.

NOBLET J., FORTUNE, H. and SHI, X.S. and DUBOIS, S. (1994). Prediction of net energy value of feeds for growing pigs. Journal of *Animal Science* **72**: 344-354.

NOBLET, J. and LE GOFF, G. (2001). Effect of dietary fibre on the energy value of feeds for pigs. *Animal Feed Science and Technology* **90**: 35-52.

NOBLET, J., and SHI, X.S. (1994). Effect of body weight on digestive utilization of energy and nutrients of ingredients and diets in pigs. *Livestock Production Science* **37**: 323-338.

OOSTING, S.J. (1993). Wheat straw as a ruminant feed. PhD thesis Wageningen University, Wageningen, The Netherlands.

PLUSKE, J.R., PETHICK, D.W., DURMIC, Z., HAMPSON, D.J. and MULLAN, B.P. (2001) Non-starch polysaccharides in pig diets and their influence on intestinal microflora, digestive physiology and enteric disease. In: Recent Developments in Pig Nutrition (Garnsworthy, P.C. and Wiseman, J., eds) vol 3, pp 123-160.

RAMONET, Y., BOLDUC J., BERGERON R., ROBERT S, and MEUNIER-SALAUN, M.C. (2000). Feeding motivation in pregnant sows: effects of fibrous diets in an operant conditioning procedure. *Applied Animal Behaviour Science* 2000, **66:**21-29.

RAMONET Y., MEUNIER-SALAUN, M.C., and DOURMAD J.Y. (1999). High-fiber diets in pregnant sows: digestive utilization and effects on the behavior of the animals. *Journal of Animal Science* **77:** 591-599.

RIJNEN M.M.J.A. (2003). Energetic uitilisation of dietary fibre in pigs. PhD thesis Wageningen University The Netherlands.

RIJNEN, M.M.J.A., VERSTEGEN, M.W.A., HEETKAMP, M.J.W., HAAKSMA, J., SCHRAMA, J.W. (2003). Effects of dietary fermentable carbohydrates on behavior and heat production in group-housed sows. *Journal of Animal Science* **81**:1822-190.

ROBERT, S., MATTE, J.J., FARMER, C., GIRARD, C.L. and MARTINEAU, G.P. (1993). High-fibre diets for sows: effects on stereotypes and adjunctive drinking. *Applied Animal Behaviour Science* **37**: 297-309.

SCHRAMA, J. W. and BAKKER, G.C.M. (1999). Changes in energy metabolism in relation to physical activity due to fermentable carbohydrates in group-housed growing pigs. *Journal of Animal Science* **77**: 3274-3280.

SCHRAMA, J.W., VERSTEGEN, M.W.A., VERBOEKET, P.H.J., SCHUTTE and J.H., HAAKSMA, J. (1996). Energy metabolism in relation to physical activity in growing pigs as affected by type of dietary carbohydrate. *Journal of Animal Science* **74**: 2220-2225.

SCHRAMA, J. W., BOSCH, M.W., VERSTEGEN, M.W.A., VORSELAAR, A.H.P.M., HAAKSMA, and J., HEETKAMP, M.J.W. (1998). The energetic value of nonstarch polysaccharides in relation to physical activity in group-housed, growing pigs. *Journal of Animal Science* **76**: 3016-3023.

SUSENBETH, A. and MENKE, K.H. (1991). Energy requirement for physical activity in pigs. In: Energy Metabolism of Farm Animals, Wenk, C. and Boessinger, M (eds) 416-419. Schriftenreihe aus dem Institut für Nutztierwissenschaften, Gruppe Ernahrung, ETH-Zurich, Zurich.

THODBERG, K., JENSEN, K.H. and HERSKIN, M.S. (1999). A general reaction patterns across situations in prepubertal gilts. *Applied Animal Behaviour Science* **63**:103-119.

VAN SOEST, P.J. (1963). Use of detergents in the analysis of fibrous feeds. II. A rapid method for the determination of fiber and lignin. *J. AOAC* **46**: 829-835.

VERSTEGEN, M.W.A., VAN DER HEL., W., BRANDSMA, H.A. and KANIS, E. (1982). Heat production in groups of growing pigs as affected by weight and feeding level. In: Energy Metabolism of Farm Animals (Ekern, A. and Sunstøl, F., eds) pp218-221. Agricultural University of Norway, Aas, Norway.

VON BORELL and E., LADEWIG, J. (1992). Relationship between behaviour and adrenocortical response pattern in domestic pigs. *Applied Animal Behaviour Science* **34**(3): 195-206.

WALSH, R.N, CUMMINS, R.A. (1976). The open field test: a critical review. Psychological Bulletin **83(3)**: 482-504.

WENK, C. (2001) The role of dietary fibre in the digestive physiology of the pig. *Animal Feed Science and Technology* **90**: 21-33.

WENK, C. and VAN ES, J.H. (1976). Eine Methode zur Bestimmung des Energieauswandes für die körperliche Aktivität von Wachsenden Küken. Schweiz. Landwirtsch. Monatsh. **54**: 232-236.

WHITTAKER, X. EDWARDS, S.A. SPOOLDER, H.A.M, LAWRENCE, A.B. and CORNING, S. (1999). Effects of straw bedding and high fibre diets on the behaviour of floor fed group-housed sows. *Applied Animal Behaviour Science* **63**(1): 25-39.

WHITTAKER, X. SPOOLDER, H.A.M, EDWARDS, S.A., LAWRENCE, A.B. and CORNING, S. (1998). The influence of dietary fibre and the provision of straw on the development of stereotypic behaviour in food restricted pregnant sows. *Applied Animal Behaviour Science* (61)**2**: 89-102.

WILLIAMS, B.A., VERSTEGEN, M.W.A. and TAMMINGA, S. (2001). Fermentation in the large intestine of single-stomached animals and its relationship to animal health. Nutrition Research Reviews **14**: 201-227.

WILLIAMS B.A., VOIGT C., MOSENTHIN R., and VERSTEGEN M.W.A. (2001). Microbial activities of faeces from unweaned and adult pigs, in relation to selected fermentable carbohydrates. *Animal Science* **73**:313-322

Organic acids for pigs: mode of action and new strategy

R. Gauthier* and A. Piva**

Jefo Nutrition Inc., Quebec, Canada. *University of Bologna, Bologna, Italy.

Organic acids have been used successfully in pig nutrition to control post-weaning diarrhoea and facilitate growth through a reduction in pH of the gastrointestinal tract (GIT). Organic acids modify the population of GIT bacteria causing a change in fermentation pattern that subsequently causes sensitive bacteria to be killed. Undissociated organic acids diffuse passively through bacterial cell membranes and following dissociation inside the bacteria impact strongly on bacterial metabolism, which leads to an accumulation of toxic anions. We tested the hypothesis that by protecting an organic acid via a patented triglyceride matrix process (Piva and Tedeschi, 2004), the acid would be released along the GIT and the populations of specific bacteria modified. Three groups of piglets (20 kg live weight), housed in an accredited research facility were fed *ad libitum* a regular corn-soy diet (16.4% crude protein, 5.9% crude fibre, 10.4 MJ/kg net energy) for two weeks before being slaughtered. One group received no organic acids in their diet, a second group received a mix of protected organic acids (0.5% of the diet including sorbic acid not naturally found in pigs) and a third group received the same mix of organic acids as group two but without the protection matrix.

Analyses were done on the GIT for sorbic acid concentration, pH, lactic acid bacteria and *E. coli*. All data were evaluated by ANOVA (GraphPad Prism Software). Significance was based on $P < 0.05$. Pigs fed the protected organic acid diet had significantly higher sorbic acid concentration along the entire GIT (with the exception of the stomach) than pigs fed the control or non-protected organic acid diets (Figure 1). There was no pH change in the GIT in any feed treatment following the addition of organic acid, except for a significant pH increase in the distal jejunum and ileum for the group fed non-protected acid. The population of lactic acid bacteria in the jejunum was not altered by any feed treatment. Pigs fed protected acid had significantly fewer *E. coli* in their jejunum than pigs in the control group or the group fed non-protected acid (Figure 2).

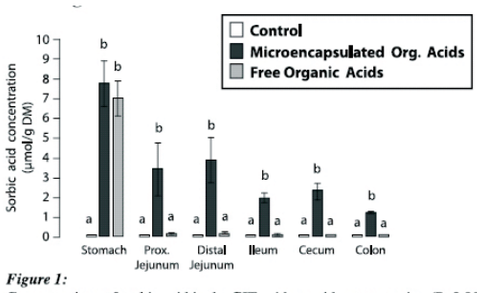


Figure 1:
Concentrations of sorbic acid in the GIT, with or without protection (P<0.05)

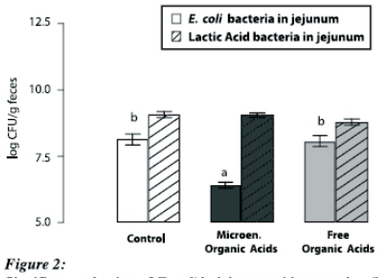


Figure 2:
Significant reduction of *E. coli* in jejunum with protection (P<0.05) vs. stability of lactic acid bacteria in same conditions.

When properly protected, organic acids can be liberated along the GIT of pigs and reduce the *E. coli* population significantly without disturbing the gut pH or the so-called beneficial lactic acid bacteria.

References

PIVA, A. and TEDESCHI, M. (2004). European Patent Office # EP1391155B1.

HMB can be used as a dietary acidifier for piglets

Y. G. Liu*, Q. Cui ** and Z. S. Wang**

* Adisseo Asian-Pacific P/L, Singapore. **Sichuan Agricultural University, Yaan, China.

Liquid methionine hydroxy analogue, known as 2-hydroxy-4-(methylthio) butanoic acid (HMB), has been used widely as a methionine source, but little attention has been paid to its potential side effects as a short chain organic acid. Our previous study demonstrated the efficacy of HMB in inhibiting pathogenic microbes such as *E. coli*, *C. perfringens, S. pullorum, fungi A. flavus* and *F. graminum* (Liu *et al.,* 2004). Commercial HMB contains 88% organic acid and has a pH of about 1.0. HMB is used mostly in poultry diets while little or none is added to swine feeds. We tested the hypothesis that because HMB is a short chain organic acid it would function as a dietary acidifier. We investigated whether HMB could reduce dietary pH and acid binding capacity (ABC) and therefore enhance the performance of piglets.

One hundred and twenty crossbred (Duroc × Landrace × Yorkshire) piglets were weaned at 28 days and allocated randomly to six treatments: 1. Control; 2. DL-Methionine (DLM) added at 1.0 kg/t; 3. Rhodimet AT88 (AT88) added at 1.14 kg (and containing HMB 1.0 kg); 4. AT88 added at 2.28 kg; 5. AT88 added at 3.42 kg/t; 6. Commercial acidifier (mainly fumaric and lactic acids) added at 3.0 kg/t. Basal diet formulation followed local industry practice (in %): corn 58.5; soybean meal 11.9; wheat middling 6.7; fish meal 6.7; expanded soybean 3.5; rapeseed meal 2.7; corn gluten meal 2.2; yeast 2.4; and sugar 3.0. There were no antibiotics in the diets. The trial lasted for six weeks and results were analysed using ANOVA and ranked according to LSD and mean.

Adding DLM at 1.0 kg/t did not affect dietary acidity but tended to increase feed intake (Table 1). However, DLM did not improve daily gain, feed conversion or incidence of diarrhoea ($P>0.05$). This indicates that the basal diet contained sufficient level of methionine to meet animal requirements for methionine and cysteine. Adding the commercial acidifier reduced the ABC value and improved performance ($P>0.05$). Adding three levels of HMB tended to reduce dietary pH and significantly reduced ABC values. Adding HMB at 2.28 kg/t improved feed intake, daily gain and FCR and significantly reduced the incidence of diarrhoea ($P<0.05$). Curiously, piglets receiving 3 kg HMB did not perform as well as those receiving 2.28 kg/t of HMB. This was unexpected and requires further investigation. We conclude that HMB functions as a typical acidifier and can improve the performance of weaned piglets, and that including HMB at about AT88 2.0 kg/t achieves good production responses.

Table 1. Impact of HMB, DLM and acidifier on the performance and dietary acidity of weaned piglets.

	Start wt (kg)	FI (g/d)	Daily gain (g)	FCR	Diarrhoea (%)	Diet pH	ABC (ml)
1. Control	8.02	815^{a*}	418^{a}	1.89^{ab}	15.12^{ab}	6.09^{a}	26.0^{a}
2. DLM 1	8.02	852^{a}	434^{a}	1.97^{b}	16.05^{a}	6.05^{a}	24.8^{ab}
3. HMB 1	8.00	862^{a}	453^{ab}	1.86^{ab}	13.58^{bc}	5.98^{a}	23.4^{b}
4. HMB 2	8.03	890^{a}	486^{b}	1.81^{a}	13.27^{c}	5.92^{a}	22.8^{b}
5. HMB 3	8.01	879^{a}	443^{ab}	1.95^{b}	14.81^{ab}	5.85^{a}	22.4^{b}
6. Acidifier	8.00	878^{a}	464^{ab}	1.87^{ab}	13.27^{c}	6.14^{a}	23.4^{b}

*Values in the same column carrying the same letter indicate $P>0.05$.

References

LIU Y.G., Z.S. WANG and X.Q. NI (2004). In: *Proceedings of Australian Poultry Science Symposium* Sydney, NSW, Australia. **16**:189-192.

Changes in pH of fermented sorghum liquid feed: natural fermentation compared with *Lactobacillus plantarum* inoculation

M. Swanson*, M. Choct** and R.J. van Barneveld***

*University of New England, Armidale, NSW 2351. **Australian Poultry CRC, Armidale, NSW 2351. ***Barneveld Nutrition Pty Ltd, South Maclean, Qld. 4280.

The rising incidence of antibiotic resistant bacteria in commercial piggeries has forced producers to seek alternatives to in-feed antibiotics. Fermented liquid feed (FLF) could offer such an alternative as it contains high numbers of lactic acid bacteria and yeast, a pH below 4.5 and high concentration of organic acids (Brooks *et al.,* 1996). The acidic environment of FLF can help to reduce or eliminate the number of enteropathogens in the feed (Brooks *et al.,* 2003). Jensen and Mikkelsen (1998) concluded that if the required pH at feeding needs to be lower than 4.5, then liquid feed needs to be fermented at least at 20°C for 8 hours with a back-slop of 50% remaining in the tank before the addition of fresh feed. Although several lactic acid bacteria inoculants have been identified for possible use in piggery FLF systems (including *Lactobacillus plantarum*), they have previously only been tested in diets where wheat or barley was the primary cereal. In this study we investigated the effect of back slopping and an inoculant of *L. plantarum* on the pH of a FLF based on sorghum.

Hammer-milled sorghum (2 mm screen) was mixed with water in sterile bottles at a cereal to water ratio of 1:3.4 (total volume 1 L of FLF) and fermented at 35°C. The experiment consisted of the following treatments: natural fermentation (control) or feed inoculated with different concentrations of *L.plantarum* per ml of liquid feed using either 0 or 50% back-slopping residue (see Table 1). Back slopping was applied at 24 and 48 hours when feed was removed from each bottle until only 50% of the original volume remained and fresh feed was added at the appropriate cereal to water ratio. There were three replicates per treatment. The pH was measured at 0, 4, 8, 12, 16, 20, 24, 48 and 72 hours. Data obtained at each time interval were subjected to a one-way ANOVA.

Adding *L.plantarum* at 10^7 per ml of liquid feed significantly lowered the pH compared to all other treatments. The results suggest that an eight-hour fermentation time for sorghum is insufficient to maintain a pH below 4.5, regardless of whether *L. plantarum* or natural fermentation is used. Fermenting sorghum for at least 16 hours at 35°C is more likely to maintain a pH below 4.5. In addition, fermenting sorghum for 24 hours before using back slopping would be sufficient to maintain a low pH, irrespective of treatment. Although the increasing concentration of lactic acid bacteria in the inoculant hastened the fermentation process over the first 12 hours and reduced the pH, it was not necessary to use an inoculant to achieve a pH below 4.5. It is possible this strain of *L. plantarum* was unable to compete effectively with other endogenous lactic acid bacteria in the feed and this may have compromised its ability to maintain a low pH. Thus, while sorghum could be used effectively as an alternative cereal grain in FLF systems, it may require a longer fermentation time and/or higher fermentation temperature than other grains, such as wheat or barley.

Table 1. Influence of *Lactobacillus plantarum* and back slopping on the pH of fermented liquid feed.

	Fermentation time (hours)								
Treatments	0	4	8	12	16	20	24	48	72
Control 1 (BS)	6.41^{cd}	6.23^{bc}	5.73^{a}	4.13^{a}	3.78^{ab}	3.74^{a}	3.65^{b}	3.68^{ab}	3.73^{a}
LAB 10^4/ml (BS)	6.44^{b}	6.24^{ab}	5.71^{a}	4.00^{c}	3.79^{a}	3.75^{a}	3.66^{ab}	3.68^{ab}	3.73^{ab}
Control 2	6.42^{cd}	6.19^{d}	5.66^{b}	4.05^{b}	3.77^{b}	3.70^{b}	3.66^{b}	3.66^{bc}	3.72^{ab}
LAB 10^4/ml	6.44^{ab}	6.24^{bc}	5.63^{bc}	4.01^{c}	3.79^{ab}	3.70^{b}	3.68^{a}	3.70^{a}	3.74^{a}
LAB 10^5/ml	6.46^{a}	6.25^{a}	5.72^{a}	3.99^{c}	3.75^{c}	3.71^{b}	3.64^{b}	3.66^{bc}	3.71^{ab}
LAB 10^6/ml	6.40^{d}	6.23^{c}	5.60^{c}	4.00^{c}	3.67^{d}	3.62^{c}	3.62^{c}	3.64^{c}	3.68^{b}
LAB 10^7/ml	6.42^{bc}	6.23^{c}	5.47^{d}	3.83^{d}	3.56^{e}	3.53^{d}	3.51^{d}	3.52^{d}	3.54^{c}
SEM	0.007	0.004	0.014	0.012	0.006	0.008	0.007	0.009	0.016

$^{a\,b\,c\,d\,e}$ Means within columns with different superscripts are significantly different ($P < 0.05$) LAB: lactic acid bacteria inoculate (*Lactobacillus plantarum*) per ml/liquid feed added at time 0 BS: back slopping treatment

References

BROOKS, P.H., GEARY, T.M., MORGAN, D.T. AND CAMPBELL, A. (1996). *The Pig Journal* **36:**43-64.

BROOKS, P.H., BEAL, J.D., NIVEN, S. AND DEMEČKOVÁ, V. (2003). *Animal Science Papers and Reports* **21:**23-39.

JENSEN, B.B. AND MIKKELSEN, L.L. (1998). In: *Recent Advances in Animal Nutrition* (eds PC Garnsworthy and J. Wiseman). Nottingham University Press, Thrumpton, Nottingham, pp. 107-126.

Insoluble non-starch polysacchrides fed as oat hulls reduce protein fermentation in the large intestine of newly-weaned pigs

J.C. Kim*, B.P. Mullan**, D.J. Hampson* and J.R. Pluske*

*Murdoch University, Murdoch, WA 6155. **Department of Agriculture, South Perth, WA 6151.

A less than optimal amount of fermentable carbohydrates and excessive undigested nitrogen (N) entering the large intestine (LI) of newly-weaned pigs has been postulated as a cause of post-weaning diarrhoea (PWD). This is because the microbiota in the LI generate biogenic amines that have been implicated in the aetiology of PWD (Porter and Kenworthy, 1969; Bolduan *et al*., 1988; Aumaitre *et al*., 1995). The purpose of this study was to observe whether adding insoluble non-starch polysaccharides (NSP) (20 g oat hulls kg^{-1}) reduced the potential negative effects of more protein than carbohydrate entering the LI on PWD and digestibility.

Forty-eight weaned male pigs (Large White X Landrace) weighing 5.5 kg ± 0.05 (SEM) were fed either (1) extruded rice plus an animal protein supplement (whey powder, meat and bone meal, blood meal, fish meal; RAP) or (2) diet (1) with 20 g oat hulls kg^{-1} (RAPOH). The pigs were offered their respective experimental diet *ad libitum* for three weeks. Titanium dioxide (TiO_2) was added as an inert marker for estimation of the coefficient of total tract apparent digestibility. The diets contained 700 g and 680 g /kg extruded rice, respectively, and contained similar concentrations of calculated digestible energy (14.4 MJ/kg) and available lysine (0.80 g/MJ DE). The diets did not contain antibiotics and ZnO. Occurrence of diarrhoea was visually assessed three times daily. Pigs showing clinical diarrhoea were injected with Trisoprim-480 (trimethropin 80 mg/ml, sulfadiazine 400 mg/ml; 1.5 ml/30 kg body weight) until clinical signs disappeared. Blood samples were taken from the jugular vein on days 7 and 14 for urea and creatinine analyses. Faecal 'grab' samples were collected from each pen on day 7 and 14 of the experiment for estimation of dry matter, energy digestibility and biogenic amines content. The ANOVA analysis and Chi-Square test were used for statistical analyses.

Table 1. Effect of oat hulls on PWD, digestibility and plasma and faecal metabolites in weaner pigs fed extruded rice and animal protein-based diets.

	RAP	RAPOH	Pooled mean	s.e.m.	Significance
Incidence of diarrhoea (No. of pigs)	9 (6)	2 (2)			0.083
No. of antibiotic treatments	21	6			
DM digestibility (%)	90.2	87.2	88.7	0.40	0.001
GE digestibility (%)	89.3	86.7	88.0	0.40	0.001
DE (MJ/kg DM)	17.0	16.5	16.7	0.07	0.001
Plasma urea (mmol/L)	3.5	2.6	3.03	0.16	0.016
Plasma creatinine (μmol/L)	74.1	76.4	75.2	1.38	0.498
Total biogenic amines (mg/kg)	431	292	362	60	0.257

Oat hulls decreased the number of pigs with PWD and the number of antibiotic treatments. Due to the dilution effect of oat hulls, the digestibilities of DM and GE were significantly decreased ($P<0.001$). Oat hull inclusion decreased plasma urea concentration ($P=0.016$) suggesting a decrease in ammonia production in the LI. This decrease was also supported by a decrease in the biogenic amines concentration, albeit not statistically significant due to the high variation between animals (SEM=60). The results suggest that insoluble NSP in a highly digestible carbohydrate diet based on extruded rice for weaner pigs reduces PWD, although reductions in digestibility were also noted. The protective effect of oat hulls was most likely due to the modification of the intestinal microbiota away from a predominantly protein fermenting populations to a biota having more saccharolytic bacteria.

The Rice Sub-program of the Rural Industries Research and Development Corporation is thanked for funding this research.

References

AUMAITRE, A., PEINIAU, J. AND MADEC, F. (1995). *Pig News and Information* **16**:73N-79N.

BOLDUAN, G., JUNG, H., SCHNABLE, E. AND SCHNEIDER, R. (1988). *Pig News and Information* **9**:381-385.

PORTER, P. AND KENWORTHY, R. (1969). *Research in Veterinary Science* **10**:440-447.

Combined strategies to deactivate mycotoxins in swine feed

D. Moore*, U. Hofstetter**, S. Nitsch**, G. Schatzmayr**, D. Schatzmayr**, A.P. Loibner***, E.M. Binder**** and M. Mézes*****

*Biomin Australia Pty Ltd., Berwick, Vic. 3806. **Biomin GmbH, Herzogenburg, Austria. ***Department of Environmental Biotechnology, Tulln, Austria. ****Erber AG, Herzogenburg, Austria. *****Szent Istvan University, Gödöllö, Hungary.

When swine ingest the mycotoxin ochratoxin A (OTA) they can develop kidney problems, drink more water and produce more urine, and reduce their feed intake and daily weight gain. The most common way to counteract the impacts of mycotxins has been to add clay minerals to feed to prevent aflatoxicosis. However, clay minerals have had variable success against some mycotoxins. Since mycotoxins vary widely in structure, it is unlikely that a single treatment will counteract all possible mycotoxins in feed. In this study we investigated the ability of a feed additive containing clay minerals BBSH797 and *Trichosporon mycotoxinivorans* (MTV) to deactivate mycotoxins in the feed of weaning piglets. The amount of OTA in feed was quantified using an HPLC method with UV detection. MTV was produced in a five-litre fermenter and cells harvested by centrifugation and subsequently lyophilised. Seventy-two Hungarian Large White x Hungarian Landrace F1 genotype mixed sex pigs (sex ratio 1:1) were used with an age of five to six weeks and an initial average live weight of about 10 kg. Treatments included a base feed with: 1). No added OTA or additive (group A); 2). OTA alone added at 500 µg/kg (group B); 3). OTA combined with the feed additive at 10^5/g of feed (group C); 4). OTA added at 10^6/g of feed (group D); and 5. MTV added alone at the level of 10^5/g of feed (group E). The feed additive and OTA were incorporated in a standard ration (mashed feed – two feeding phases, Feed I was consumed for four weeks and Feed II for three weeks) and fed to piglets for a period of 49 days. OTA had a negative impact on live weight (LW), average daily weight gain (ADWG) and feed conversion ratio (FCR) of weaning piglets (Table 1). A concentration of 10^5 /g MTV improved live weight after 28 days (group C). Adding MTV improved live weights after 49 days and FCR after 28 days compared to pigs fed the toxin.

Table 1. Impact of MTV and OTA on live weight, ADWG, FCR, deaths and incidence of diarrhoea in piglets.

Design	A	B	C	D	E
CFU/g	-	-	10^5	10^6	10^5
OTA µg/kg	-	500	500	500	500
LW (kg) 0d	10.23 ± 1.19	10.20 ± 1.14	10.23 ± 0.94	10.23 ± 1.02	10.25 ± 0.99
LW (kg) 28d	22.76 ± 3.02	20.24 ± 3.71	21.86 ± 2.35	21.79 ± 2.88	21.78 ± 2.02
LW (kg) 49d	32.65 ± 4.75	30.78 ± 5.13	31.53 ± 4.55	33.81 ± 4.32	33.10 ± 4.95
ADWG (g)	457.7 ± 85.9^a	379.8 ± 103.1^b	445.4 ± 111.0^{ab}	476.7 ±78.5^a	465.5 ± 98.5^a
FCR 28d	1.88	2.15	1.94	1.83	1.99
FCR 49d	2.44	2.35	2.30	2.13	2.24
Losses (No.)	0	2	0	0	0
Diarrhoea (No.)	2	4	0	1	0

^{a,b}Means within a column with no common superscript are significantly different (P<0.05).

After 28 days, adding MTV alone did not improve live weight, but after 49 days live weight of these animals was slightly higher than in the control group. Fewer animals were lost to diarrhoea in the control and trial groups than in the toxin group. When animal losses in Group B were considered in the live weight measurement, the most objective evaluation criterion was average daily weight gain (ADWG). ADWG in the toxin group were significantly lower than in the other groups. We conclude that adding *Trichosporon mycotoxinivorans* to feed can alleviate the negative effects of OTA on swine.

References

FUCHS, E., BINDER, E.M., HEIDLER D. and KRSKA R. (2002). *Food Additives and Contaminants* **19**:379-386.

MOLNAR, O., SCHATZMAYR, G., FUCHS, E. and PRILLINGER, H. (2004). *Systematic and Applied Microbiology* **27**:661-671.

RAMOS, A.J., FINK-GREMMELS, J. and HERNANDEZ, E. (1996). *Journal of Food Protection* **59**:631-641.

Particle size influences the incidence of stomach ulcers but has no effect on performance in barley-based diets for pigs

P.C.H. Morel

Massey University, Palmerston North, New Zealand.

Particle size can influence nutrient digestibility, growth rate, feed intake, feed conversion ratio and also gut health especially in terms of gastric ulceration. Reducing particle size generally has a positive effect on growth performance and a negative effect on gut health (Wondra *et al.*, 1995; Guillou and Landeau, 2000). However, most published experimental work is based on corn-based diets, which are not typical in New Zealand. The aim of this study was to investigate the effect of particle size in barley-based diets on the growth rate, energy digestibility and gastric ulceration in pigs.

The grower and finisher diets contained barley grain (70.5% and 80.7% respectively), soybean meal and animal byproducts. The barley was ground with a hammer mill equipped with 1 mm (Fine), 4 mm (Medium) or 7 mm (Coarse) screens. Four diets were mixed: Coarse ($d_{gw} \pm S_{gw}$, 1100 μm ±2.19μm), Medium (78 5μm ±2.23μm), Fine (434 μm±1.97μm) and Mixed (789 μm±2.45 μm). The Mixed diet was a blend of 1/3 Coarse, 1/3 Medium and 1/3 Fine and was used to assess the impact of a variable particle size.

Sixty-four entire male pigs (31.1 kg live weight (LW) ± 2.70 kg (sd)) were housed in eight pens of eight pigs and fed *ad libitum*, with water available at all times. Each pen was allocated randomly to one of four treatment groups (Fine, Medium, Coarse or Mixed) with two pens of eight pigs per treatment. The grower diet was fed for five weeks (up to 61.1 kg ± 5.6 kg) and the finisher diet from five weeks until slaughter at 87.2 kg ± 5.7 kg. Individual growth rates were recorded. Following slaughter, P2 was measured and the *pars-oesophagea* part of the stomach was scored for gastric ulceration on a scale of 0 to 3 (Kavanagh, 1994). Twenty-four entire male pigs, weighing 34.3 kg (± 2.4 kg) were allocated randomly to one of the four grower diets to determine faecal apparent energy digestibility (total collection method). The effect of dietary particle size on growth rate, back fat thickness and energy digestibility was statistically analysed with a simple analysis of variance. Stomach scores were analysed using a categorical data modelling procedure.

Table 1. Effect of particle size on energy digestibility and pig growth, back fat and stomach ulceration score.

	Fine	Medium	Coarse	Mixed	SE	P
Growth rate (g/d)	940	964	937	994	23.9	0.462
P2 back fat (mm)	10.7	10.9	10.2	10.9	0.59	0.448
Ulceration score (0-3)	1.87^{z}	0.20^{x}	0.25^{x}	0.69^{y}	0.156	
Energy digestibility (%)	81.2	78.9	81.7	82.1	0.93	0.103

^{xyz}- row with different letter superscript are significantly different (Chi^2, $p<0.05$)

There were no statistically significant differences in growth rate, P2 back fat or energy digestibility between pigs fed diets of different average particle size. Pigs fed the Medium and Coarse diets had lower stomach ulceration scores (0.20 and 0.25 respectively) than those fed the Mixed diet (0.69) or the Fine diet (1.87). All animals fed the Fine diet had stomach lesions and ulcerations. It is concluded that, with barley based diets, a variation in average particle size between 400 μm and 1100 μm has practically no effect on growth performances and apparent faecal energy digestibility. However, the incidence of stomach lesion and ulceration increases greatly when the average particle size is reduced to 434 μm (Fine).

This work was supported by the New Zealand Pork Industry Board.

References

GUILLOU, D. and LANDEAU, E. (2000). *Production Animale* **13**:137-145

KAVANAGH, N. (1994). *In Practice* **16**:209-213.

WONDRA, K.L., HANCOCK, J.D., BEHNKE, K.C. HINES, R.H. and STARK, C.R. (1995). *Journal of Animal Science* **73**:757-763.

Evaluation of cottonseed meal for pigs between 20-90 kg live weight

D.N. Singh* and M. Evans**

*Department of Primary Industries and Fisheries, Cleveland, Qld. 4163. **Applied Nutrition Pty Ltd, Alexandra Hills, Qld. 4161.

While cottonseed meal (CSM) has potential for greater use as a protein source it is often included only at low levels in pig diets (4-10%) because of the suspected adverse effects of anti-nutritional factors on feed digestibility. This experiment examined the impact of increasing levels of a solvent-extracted CSM on the performance of pigs from 20-90 kg live weight. The experiment was arranged as a randomised block layout of 36 individually penned Large White cross pigs, with six dietary treatments (0, 50, 100, 150, 150 with Fe and 200 g/kg CSM) and two sexes (males and females), replicated three times. Pigs weighing about 20 kg were randomly allocated within sex and initial weight. Diets were formulated to 14 and 13 MJ DE/kg and 0.63 g and 0.55 g available lysine/MJ DE for growers and finishers, respectively. The major sources of dietary protein replaced by CSM were soybean meal, meat and bone meal and blood meal. All diets were offered *ad libitum*. Parametric exercises on CSM (CSM46% @ $418/t; CSM37% @ $352/t) were carried out for each growth phase to establish the cost/inclusion relationship (breakpoints) and key replacement raw materials in diets for grower and finisher pigs up to an inclusion level of 20%.

Table 1. Effect of feeding graded levels of CSM on pigs growing from 20 to 90 kg.

	Grower 20-50kg			Finisher 50-90kg			
Diets CSM (g/kg)	ADG (kg/d)	FCR (feed/gain)	DFI (kg/d)	ADG (kg/d)	FCR (feed/gain)	DFI (kg/d)	P2 back fat (mm)
0	0.83[b]	2.21	1.84[b]	1.08	2.62	2.81	14.3
50	0.75[c]	2.52	1.87[b]	1.07	2.67	2.76	13.3
100	0.89[ab]	2.37	2.12[a]	1.19	2.80	3.34	15.2
150	0.91[a]	2.31	2.07[a]	1.05	2.90	3.01	12.8
150 +Fe	0.90[a]	2.31	2.08[a]	1.01	2.86	2.90	13.0
200	0.90[a]	2.37	2.13[a]	1.05	3.02	3.17	14.0
l.s.d. (0.05)	0.07	0.30	0.19	0.20	0.44	0.53	4.5

Means within columns not followed by a common superscript differ significantly.

The performance of pigs fed more than 100 g/kg CSM in the grower phase was significantly greater than those on the control diet ($P<0.05$). This suggests that the DE value of CSM was underestimated in the formulation of the diets. The diet containing 50 g/kg of CSM yielded the lowest ADG (0.747 kg/d), although the DFI at this inclusion level was not significantly different to the control diet. The FCR did not change significantly as the level of CSM was increased. In the finisher phase there was no significant difference in ADG, FCR, DFI and back fat. The mean ADG was 1.08 kg/d, FCR 2.83 and DFI 3.02 kg/d. Adding iron salt did not have any effect on growth performance at 150 g/kg.

The data show that up to 200 g/kg of CSM can be fed to pigs growing from 20 to 90 kg without any deleterious effect on performance. The parametric exercise showed that, in the grower diet, as the cost of CSM46% increased from $412 to $435, CSM inclusion decreased from 20 to 2.4% while CSM37% inclusions decreased from 20 to 8% as cost increased from $346 to $358. In finisher diets CSM46% inclusion decreased from 20 to 9.8% as cost increased from $356 to $418 while there was no gradual parametric profile for CSM37%. Once the cost of CSM37% reached $353/t up to 20% CSM was included in the diet.

Supported in part by Australian Pork Ltd.

Assessment of agronomic practices on triticale variety W19

A. Murphy, A.C. Philpotts and D.J. Henman

QAF Meat Industries, Corowa, NSW 2646.

Triticale is a cereal grain with specific application as a stock feed. Evaluation of several triticale varieties for grain yield and their effects on weaner growth performance identified a high-yielding variety (W19) suitable for feeding to pigs (unpublished data). In this study we hypothesised that growing conditions would alter the chemical composition of W19 triticale grain and that this would subsequently affect pig performance.

Eighty, 21-day-old Landrace x Large White male weaner pigs (live weight 7 kg ± 0.5 kg) were housed in individual cages and assigned randomly to one of four treatments based upon initial live weight. Treatments were assigned as: triticale fertilised with pig effluent (PE); two separate sources of triticale from dryland farms using convention fertiliser application (DL1 and DL2); and triticale irrigated (I). All triticale crops were grown within a 100 km radius of Corowa, NSW. Diets were formulated to a standard triticale specification (65% of the diet) and 14.2 MJ DE/kg and 10.5% protein. All other dietary components were kept the same for each treatment. Pigs were fed the dietary treatments for 21 days and average daily gain (ADG), average daily intake (ADI) and feed conversion ratio (FCR) recorded at 7, 14 and 21 days. The statistical analysis was done using SPSS for windows version 12. Performance data were analysed using general linear model, analysis of variance. The analysis was done using a multi-variant approach with treatment as the fixed factor and weight at each time period, rate of gain, feed intake and feed conversion during each period as dependant variables.

Table 1. Effect of growing conditions on the chemical composition of triticale W19.

Growing conditions / source farm	Protein (%)	Starch (%)
Fertilised with pig effluent (PE)	17.2	55.6
Grown with irrigation (I)	10.5	60.1
Dryland grown with fertiliser (DL 2)	11.7	57.4
Dryland grown with fertiliser (DL 1)	13.5	60.1

Table 2. Effect of W19 triticale grown under different conditions on the performance of male weaners.

Growing conditions /source farm	Start weight (kg)	Final weight (kg)	Rate of gain (kg/day)	Feed conversion ratio	Average daily intake (kg/day)
PE*	6.99	19.80	0.539	1.23	0.664
I	7.03	19.67	0.530	1.21	0.643
DL 2	7.00	20.15	0.545	1.23	0.668
DL 1	6.93	19.44	0.519	1.22	0.632
SEM	0.036	0.284	0.01	0.014	0.015
Signifcance[1]	NS	NS	NS	NS	NS

[1]NS not significant; *Refer to Table 1 for details of growing conditions.

Triticale grain sourced from different farms or growing conditions showed some differences in chemical properties (Table 1). Each source received the same application of mineral fertiliser while the triticale grown at the QAF farm was fertilised with pig manure. The increased level of nitrogen supplied by the pig manure was incorporated into grain protein at the expense of starch. There was no significant difference in the growth performance of the pigs due to the different treatments (Table 2). Thus, while agronomic practices altered some chemical characteristics of triticale, there was no effect on growth performance.

6 Nutrition, feed additives, productivity and reproduction

A step-up ractopamine (Paylean) program increases lean tissue in all sexes and decreases fat tissue in boars and immunocastrates

F.R. Dunshea*, C. Rikard-Bell**, M.A. Curtis**, A.C. Edwards***, N.J. Gannon****, D.J. Henman*****, B.P. Mullan****** and ******* R. J. van Barneveld

* Department of Primary Industries, Werribee, Vic. 3030, **Elanco Animal Health, Macquarie Park NSW 2113, ***ACE Consulting, Cockatoo Valley, SA 5440. ****Ridley AgriProducts, Toowoomba, Qld. 4352, *****QAF Meat Industries, Corowa, NSW 2646. ******Western Australian Department of Agriculture, South Perth, WA 6151. ******* Barneveld Nutrition, South Maclean Qld. 4280.

The ß-agonist ractopamine (Paylean®[1], RAC) has recently been approved in Australia for use as a dietary ingredient to improve lean tissue mass and growth in finisher pigs (Dunshea *et al.,* 1993). In gilts and barrows it appears that RAC increases lean mass without changing fat mass (Dunshea *et al.,* 1993). However, further analyses of the limited data that exist for boars (Dunshea *et al.,* 1993) suggest that RAC may increase lean tissue and decrease fat deposition. There are no data on the effect of RAC on body composition in immunocastrated boars (IM, boars treated with Improvac®[2] at 11 and 17 weeks of age). The aim of this investigation was to determine if a step-up RAC feeding program would increase lean mass in all sexes and decrease fat mass in entire and IM male pigs.

The study involved 286 commercial crosses and pure line pigs randomised and proportionally allocated by breed into 24 groups of 11 or 12 pigs, with equal groups of boars, IM boars and gilts. At 17 weeks of age (ca. 71 kg) pigs were stratified on live weight, allocated to pens and offered diets containing either 0 or 5 ppm of RAC for 14 days. After 14 days the dose of RAC was increased to 10 ppm and diets were fed for a further 17 days (Rikard-Bell *et al.,* 2005). After slaughter, carcasses from the five median live weight pigs from each pen were split in half and then scanned for regional body composition using dual energy X-ray absorptiometry (DXA) (Suster *et al.,* 2004). Data were analysed using ANOVA.

Table 1. Effect of sex and step-up dietary ractopamine program on composition of the carcass (head off) of finisher pigs as determined by DXA.

Sex (S)	Boar		Gilt		IM			Significance		
Diet (D)	Control	RAC	Control	RAC	Control	RAC	s.e.d[a]	S	D	SxD
Total (kg)	71.3	73.4	68.4	73.6	72.1	75.6	1.12	<.001	0.008	0.18
Lean (kg)	49.5	53.5	44.5	49.3	46.6	53.1	1.99	<.001	0.014	0.66
Fat (kg)	9.2	6.5	13.0	12.1	14.6	9.3	1.87	0.013	0.004	0.28
Bone (kg)	12.2	12.5	11.5	12.3	12.2	12.7	0.20	<.001	0.003	0.15
Lean (%)	69.5	72.8	65.1	67.0	64.4	70.2	2.03	0.006	0.006	0.40
Fat (%)	12.9	8.9	18.9	16.4	20.4	12.3	2.58	0.004	0.004	0.31
Bone (%)	17.1	17.0	16.8	16.7	16.9	16.8	0.18	0.33	0.053	0.97

[a]SED for Sex x Diet;

Dietary RAC increased (P=0.018) lean tissue mass by 4.0, 4.8 and 6.5 kg in the boars, gilts and IM boars, respectively. In the boars and IM boars the increase in lean tissue was accompanied with a decrease (P=0.004) in fat mass, particularly in the latter group. Consistent with previous studies there was little effect of dietary RAC on fat mass in gilts. However, carcass percent fat was decreased (P=0.004) and percent lean increased (P=0.006) in all sexes. Immunocastration caused a decrease in lean tissue mass and an increase in fat mass in response to the absence of male steroids and a dramatic increase in feed intake, particularly over the last half of the study (Rickard-Bell *et al.,* 2005). It appears that unlike for gilts and surgical barrows, dietary RAC may decrease fat mass in entire and IM boars. In conclusion, these data demonstrate that a dietary RAC step-up program can be used to increase lean tissue yields in all sexes and offers an excellent means of minimising the increase in fat mass sometimes observed in IM boars.

References

DUNSHEA, F.R., KING, R.H. and CAMPBELL, R.G. (1993). *Journal of Animal Science* **71**: 2931-2941.

RIKARD-BELL, C., CURTIS, M.A., VAN BARNEVELD, R.J., MULLAN, B.P., EDWARDS, A.C., GANNON, N.J., HENMAN, D.J., HUGHES, P.E. and DUNSHEA, F.R., (2005). In 'Manipulating Pig Production X', p. 151. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

SUSTER, D., LEURY, B.J., HOFMEYR, C.D. and DUNSHEA, F.R. (2004). *Australian Journal of Agricultural Research* **55:** 973-982.

A step-up dietary ractopamine (Paylean) program improves growth performance and carcass traits in all sexes

C. Rikard-Bell*, M.A. Curtis*, R. J. van Barneveld**, B.P. Mullan***, A.C. Edwards****, N.J. Gannon*****, D.J. Henman******, P.E. Hughes******* and F.R. Dunshea********

* Elanco Animal Health, Macquarie Park NSW 2113, ** Barneveld Nutrition, South Maclean Qld. 4280, *** Department of Agriculture, South Perth, WA 6151. **** ACE Consulting, Cockatoo Valley, SA 5440. ***** Ridley AgriProducts, Toowoomba, Qld. 4352, ****** QAF Meat industries, Corowa, NSW 2646. ******* South Australia Research and Development Institute, Roseworthy SA 5371. ******** Department of Primary Industries, Werribee, Vic. 3030.

The ß-agonist ractopamine (Paylean®, RAC) has recently been approved in Australia for use as a dietary ingredient to improve production efficiency in pigs. Dietary RAC increases lean tissue deposition and daily gain in pigs fed protein-adequate diets (Dunshea *et al.,* 1993) when fed at 20 ppm. The effects of RAC are most pronounced in the first two weeks of feeding and decline thereafter due to down-regulation of ß-receptors (Dunshea 1993). A possible way to counter down-regulation is to increase the dose of RAC during the treatment period. The aim of this investigation was to determine if the stimulation in growth performance could be maintained over 31 days by using a step-up RAC feeding program.

The study involved 286 commercial crosses and pure line pigs randomised and proportionally allocated by breed into 24 groups of 11 or 12 pigs, with equal groups of boars, immunocastrated boars (IM, boars treated with Improvac® at 11 and 17 wk of age) and gilts. At 17 wk of age (ca. 71 kg) pigs were stratified on liveweight, allocated to pens and offered diets containing either 0 or 5 ppm of RAC for 14 days. After 14 days the dose of RAC was increased to 10 ppm and diets were fed for a further 17 days. Diets were formulated to contain 0.56 g available lysine/MJ DE and 13.2 MJ/kg. Pigs were weighed on d 0, 14 and 31 and voluntary feed intake (VFI) determined on days 14 and 31. Back fat at the P2 site was determined with an ultrasound on day 0 and day 31. At the end of the study pigs were slaughtered and carcass weight and P2 back fat determined. Data were analysed by ANOVA with pen as the experimental unit.

Table 1. Effect of sex and step-up dietary ractopamine program on voluntary feed intake (VFI), average daily gain (ADG), feed conversion ratio (FCR) and carcass characteristics in finisher pigs.

Sex (S)	Boar		Gilt		IM			Significance		
Diet (D)	Control	RAC	Control	RAC	Control	RAC	SED[a]	S	D	SxD
VFI d 0-14, kg/d	3.01	2.79	2.83	2.82	3.04	2.82	0.080	0.19	0.005	0.14
VFI d 15-31, kg/d	3.15	3.21	3.00	3.11	4.15	3.96	0.099	<0.001	0.87	0.10
ADG d 0-14, kg/d	1.19	1.17	0.94	1.04	1.20	1.19	0.065	<0.001	0.57	0.37
ADG d 15-31, kg/d	1.03	1.16	0.96	1.06	1.26	1.35	0.031	<.001	<.001	0.67
FCR d 0-14	2.55	2.39	3.03	2.72	2.52	2.38	0.136	<0.001	0.019	0.64
FCR d 15-31	3.05	2.78	3.13	2.93	3.30	2.95	0.100	0.036	<.001	0.56
Carcass[b], kg	79.6	81.1	78.0	80.9	82.6	84.5	1.15	0.001	<.001	0.33
Delta P2[c], mm	3.0	2.8	2.8	2.8	5.6	4.4	0.42	<0.001	0.076	0.12

[a]SED for Sex x Diet; [b]initial live weight used as a covariate; [c] calculated by difference between d 0 and 31.

Dietary RAC decreased VFI during the first two weeks particularly in the entire and IM boars with the reduction in VFI being maintained in the IM boars after the step-up in RAC dose. Daily gain was not altered by dietary RAC during the first two weeks but was increased after the step-up in dose. The FCR was decreased in all sexes with the response being greater after the increase in dietary RAC dose. Carcass weight was increased (P<0.001) by dietary RAC in all sexes while P2 tended (P=0.076) to be reduced, particularly in the IM boars. These effects on fat deposition were confirmed using dual energy X-ray absorptiometry (Dunshea *et al.,* 2005). These data demonstrate that a step-up RAC program can be used to maintain or even improve the growth promoting effects of dietary RAC in all sexes and offers an excellent means of maximising the effects of immunocastration while minimising the increase in P2 sometimes observed in IM boars.

References

DUNSHEA, F.R., KING, R.H. and CAMPBELL, R.G. (1993). *Journal of Animal Science* **71:** 2931-2941.

DUNSHEA, F.R. (1998). *Journal of Animal Science* **71:** 1966-1977.

DUNSHEA, F.R., RIKARD-BELL, C., CURTIS, M.A., EDWARDS, A.C., GANNON, N.J., HENMAN, D.J., MULLAN, B.P. and VAN BARNEVELD RJ (2005). In 'Manipulating Pig Production X', p. 150. ed J.E.Paterson. (Australasian Pig Science Association: Werribee).

Effect of Allzyme SSF on the growth performance of grower pigs

Q. Lei *, C. Miao **, R. Wang ***, D. Lin * and S. Chen **

*Henan Agricultural University, China. ** Alltech Asia-Pacific Bioscience Center, Beijing, China. ***China Agricultural University, China.

Commercial use of phytase and other enzymes has been widely accepted by the feed industry. However, the purified enzymes produced by submerge-liquid fermentation might not always match the matrix value for specific raw materials and also could have issues regarding GMO status. Recently, a GMO-free enzyme product has been developed (Allzyme SSFTM, Alltech Inc, USA), which is produced from a fungal strain via solid-state fermentation (SSF) and contains seven major enzyme activities including phytase. Efficacy of Allzyme SSF on phosphorus excretion and ileal digestibility for phosphorus, calcium, crude protein and energy has been demonstrated (Park *et al*., 2003; Wu *et al*., 2004). However, data on the effects of lower doses of Allzyme SSF (0.2 kg/ton) in the grower and finisher pig fed wheat-based diets are relatively unknown. The aim of the following experiment was to investigate the effect of Allzyme SSF on the performance of grower pigs fed wheat-based diets containing low crude protein (CP) or low available phosphorous (aP) and digestible energy (DE).

Forty crossbred pigs (Durcoc×Landrace×Yorkshire, average live weight 21.6 kg) were allocated randomly to five treatments with four replicates of two pigs (male and female) per pen per treatment. All pigs were allowed a 7-day adaptation period and the experiment lasted for 12 weeks. Five experimental diets were formulated (calculated analysis shown in Table 1): positive control, low CP (-6%), low CP plus 0.2 kg/t Allzyme SSF, low- aP/CP/DE (-42%, -6% and –0.37 MJ/kg respectively), and low aP/CP/DE plus 0.2 kg/t Allzyme SSF. The data were subjected to analysis of variance by the General Linear Models procedures of SPSS 11.5 (SAS Institute, 1997) using pen as the experimental unit.

Table 1. Calculated analysis of the experimental diets.

Diets	Control	Low CP	Low CP+SSF	Low aP/ CP/DE	Low aP/ CP/ DE + SSF
DE (MJ/kg)	13.29	13.29	13.29	12.91	12.91
CP (%)	18.14	17.07	17.07	17.07	17.07
Ca (%)	0.71	0.70	0.70	0.70	0.70
aP (%)	0.334	0.320	0.320	0.188	0.188
Lysine (%)	0.95	0.95	0.95	0.95	0.95
Allzyme SSF (kg/t)	-	-	0.2	-	0.2

Table 2. Effect of dietary treatments on the growth performances of growing pigs (20-50 kg).

Treatments	Initial body weight (kg)	Final body weight (kg)	Average daily gain (g)	Daily feed intake (g)	FCR
Positive control	21.39±2.25	42.31±3.91	675.0±58.43	1491.1±104.63	2.21±0.01
Low-CP	21.53±2.56	45.53±4.28	662.3±81.27	1440.7±202.30	2.18±0.18
Low-CP + Allzyme SSF	21.44±2.87	41.97±5.23	774.2±71.60	1563.5±144.81	2.02±0.17
Low aP/CP/DE	21.63±2.67	41.34±4.36	636.1±103.35	1442.7±110.49	2.30±0.27
Low aP/CP/DE + Allzyme SSF	21.78±2.80	43.03±4.99	686.5±86.04	1377.6±209.76	2.01±0.22

Dietary treatments had no significant effect (P>0.05) on daily gains, feed intake and FCR of growing pigs (Table 2). However there was a trend for the down-specified diets to increase FCR as a consequence of poorer ADG. Addition of Allzyme SSF to both down-specified diets tended to improve ADG and FCR to levels exceeding the positive control performance, the benefits of which are of commercial relevance. The results suggest that adding Allzyme SSF to diets low in CP or low in aP, CP and DE tended to improve ADG and FCR in growing pigs.

References

PARK, J.S., CARTER, S.D., SCHNEIDER, J.D. and MORILLO, T.B. (2003). *Journal of Animal Science* (suppl 1): 35.

WU, Y.B., RAVINDRAN, V. and HENDRIKS, W.H. (2003). *British Poultry Science* **44**:710-718.

Effect of Allzyme SSF on the growth performance of finisher pigs

Q. Lei *, C. Miao **, R. Wang ***, D. Lin * and S. Chen**

*Henan Agricultural University, China. ** Alltech Asia-Pacific Bioscience Center, Beijing, China. ***China Agricultural University, China.

We examined the benefits of supplementing pig feed with Allzyme SSF (Alltech Inc, USA) - a novel enzyme preparation produced from a single strain of fungi via solid-state fermentation (SSF) and containing seven major enzyme activities including phytase. The aim was to investigate the effect of Allzyme SSF on the performance of finisher pigs fed wheat-based diets containing low crude protein (CP) or low available phosphorous (aP) and digestible energy (DE).

Forty crossbred pigs (Durcoc×Landrace×Yorkshire with an average live weight of 42.6 kg) were allocated randomly to five treatments with four replicates of two pigs (male and female) per pen per treatment. All pigs were given a 7-day adaptation period and the experiment lasted for 12 weeks. Five experimental diets were formulated (calculated analysis shown in Table 1): positive control, low CP (-6%), low CP plus 0.2 kg/t Allzyme SSF, low- aP/ CP/DE (-42%, -6% and –0.37 MJ/kg respectively), and low aP/CP/DE plus 0.2 kg/t Allzyme SSF. The data were subjected to analysis of variance by the General Linear Models procedures of SPSS 11.5 (SAS Institute, 1997) using pen as the experimental unit.

Table 1. Calculated analysis the experimental diets.

Diets	Control	Low CP	Low CP+SSF	Low aP/ CP/DE	Low aP/ CP/ DE + SSF
DE (MJ/kg)	13.29	13.29	13.29	12.91	12.91
CP (%)	18.14	17.07	17.07	17.07	17.07
Ca (%)	0.71	0.70	0.70	0.70	0.70
aP (%)	0.334	0.320	0.320	0.188	0.188
Lysine (%)	0.95	0.95	0.95	0.95	0.95
Allzyme SSF (kg/t)	-	-	0.2	-	0.2

Table 2. Effect of dietary treatments on the growth performances of finisher pigs (50-100 kg).

Treatments	Initial body weight (kg)	Final body weight (kg)	Average daily gain (g)	Daily feed intake (g)	FCR
Positive control	42.31±3.91	81.25±0.31 b	812.1±11.74c	2651.0±20.07c	3.26±0.02c
Low-CP	45.53±4.28	65.94±0.30 e	501.7±14.57a	1680.5±32.66ab	3.36±0.03b
Low-CP + Allzyme SSF	41.97±5.23	85.56±0.25 a	834.4±10.42c	1647.4±30.94a	1.97±0.05d
Low aP/CP/DE	41.34±4.36	72.65±0.22 d	653.4±12.46b	2643.2±20.05c	4.05±0.05a
Low aP/CP/DE + Allzyme SSF	43.03±4.99	76.67±0.38 c	701.2±18.26c	1731.7±40.2b	2.47±0.01e

Pigs fed the low CP and the low aP/CP/DE diet gained significantly less weight ($P<0.05$) than positive control animals. In addition, these pigs had a significantly better FCR ($P<0.05$) than positive control animals. Adding Allzyme SSF to the down-specified diets significantly improved ($P<0.05$) ADG, feed intake and FCR compared to negative control animals. Pigs fed down-specified diets supplemented with Allzyme SSF exhibited significantly better FCR than positive control animals.

The results showed that adding Allzyme SSF to diets low in CP or low in aP/CP/DE improved growth performance significantly compared to positive and negative control animals.

The apparent digestibility of phosphorus from a range of inorganic sources is less than 100% in pigs

R.H. King, J. Cauduro and P.J. Eason

Department of Primary Industries, Werribee, Vic. 3030.

Phosphorus (P) pollution from intensive animal agriculture has received much attention in recent times. Although P is an essential nutrient for the pig, 60-70% of P found in common pig feed ingredients is bound as phytate. As pigs lack the endogenous enzyme phytase to break down phytate P, inorganic P is sometimes added to pig diets to meet P requirements. While several feeding standards assume that the availability of P from all inorganic sources is 100%, the review by Jongbloed *et al.* (1993) showed there were marked differences in digestibility of P ranging from 65 to 90%, depending on the type and origin of feed phosphates. The aim of this experiment was to determine the digestibility of six sources of P that were included in a basal diet low in P.

Eight male pigs of 41.6±0.4 kg live weight (mean±SE) were allocated randomly to seven diets. Six pigs received one of the test diets containing supplemental P, while the remaining two pigs received a basal diet containing no supplemental P. The basal diet based upon corn, soybean meal and blood meal contained 7.4 g Ca/kg, 3.1 g P/kg and an estimated 0.8 g available P/kg. To this basal diet, 1.7 g P/kg was added from one of the six sources of supplemental P (Table 1) to create the six experimental diets. The digestibility of P in the various sources was determined by difference. Pigs were fed 1.5 kg/day for 14 days and total faecal output was collected daily for the last five days of this period after sufficient acclimatisation to the diet. At the end of the first 14-day experimental period, pigs were reallocated to the experimental diets and subsequently assigned to another three 14-day experimental period to obtain four replicates of each test diet and eight replicates of the basal diet. Digestibility data were subjected to analysis of variance for treatments in a blocked design with block being the experimental period.

The P content of urine of pigs fed the experimental diets was low (5.7 ± 1.3) mg/L and certainly well less than the maximum 150 mg/L previously observed in pigs fed below P requirement (Jongbloed, 1987). The apparent digestibility of P in the basal diet was 23.0 ± 3.4%.

Table 1. Composition (g/kg) and apparent digestibility of P in supplemental P sources.

Phosphorus Source	Phosphorus	Calcium	P digestibility (%)
Rock Phosphate	172	386	45.9^{c}
Dicalcium phosphate	183	245	48.7^{bc}
Dicalcium phosphate (anhydrous)	187	288	72.3^{a}
Monocalcium/Dicalcium phosphate	211	168	71.1^{ab}
Dicalcium phosphate (dihydrate)	183	243	73.2^{a}
Meat and bone meal	32	59	85.1^{a}
s.e.d			11.8

^{ab} means with different superscripts are significantly different (P<0.05).

The apparent digestibility of P in rock phosphate was the lowest of all P sources and significantly less than in most of the other inorganic sources of P. The apparent digestibility of P in most of the mono or dicalcium phosphates was similar and within the range Jongbloed *et al.* (1993) reported for a wide range of feed phosphates. Furthermore, Jongbloed *et al.* (1993) concluded that the apparent P digestibility in meat and bone meal samples ranged from 74- 85%, which is similar to the results of our study. These results suggest that there are differences in the apparent digestibility of P from various inorganic P sources and these differences should be considered when formulating diets to minimise excretion of P by pigs.

Supported in part by Australian Pork Limited.

References

JONGBLOED, A.W. (1987). Phosphorus in the Feeding of Pigs. PhD Thesis. Wageningen Agricultural University.

JONGBLOED, A.W., EVERTS, H. and KEMME, P.A. (1993). In 'Recent Developments in Pig Nutrition 2'. pp. 163-178, eds D.J.A. Cole, W.Haresign and P.C. Garnsworthy. (Nottingham University Press, Loughborough, UK).

Australian Pork

Haematological indices of piglets provided with parenteral iron dextran and creep feed or soil prior to weaning

H.G. Payne*, B.P. Mullan*, R.R. Nicholls*, S.M. McCulloch*, J.R. Pluske** and P. Clark**

Department of Agriculture, South Perth, WA 6151. *Murdoch University, Murdoch, WA 6150.

There have been recent field reports from Western Australia of anaemia in 21-day-old piglets from conventional production systems. This study investigated haematological indices of piglets that were farrowed indoors but offered substrates commonly available to piglets produced outdoors.

Two replicates of 12 multiparous crossbred sows were batch-farrowed in an environmentally controlled room maintained at 22ºC in slotted-floored farrowing crates with a creep area equipped with a solid floor, hover, heat lamp, 600 mm long feeder and nipple drinker. Piglets were weighed, ear-notched, tooth-clipped, tail-docked and injected intramuscularly with 200 mg of iron dextran on day two after birth. Litters were equalised at 10 piglets per sow. Following stratification for birth date, litters were allocated randomly to one of three pre-weaning nutritional treatments: 1). control - no creep feed (NC), 2). commercial creep (CF) fed as crumbles and 3). an 'outdoor' mix (OM) of 25 parts soil, 5 parts sow feed and 1 part straw. Treatments were offered from 7 days until weaning at 28 days of age. Blood samples were taken from median weight piglets from six randomly selected litters per treatment. Differences between treatment means (six pigs per treatment) were tested using analysis of variance.

Table 1. Haematological indices (mean, ±SEM) of 28-day-old piglets that received iron dextran on day two after birth and subsequently offered no creep feed (NC), ad libitum creep feed (CF) or an 'outdoor mix' of soil, sow feed and straw (OM) from seven days of age until weaning at 28 days of age.

	NC	CF	OM	s.e.d	P value
Serum Fe (µmol/l)	13.2 ±7.56	11.4 ±5.25	33.0 ±7.32	9.48	0.073
Red Blood Cells (RBC) (x 10^{12}/l)	5.37 ±0.217	5.59 ±0.170	6.16 ±0.076	0.229	0.011
Haemoglobin (Hb) (g/l)	80.0 ±3.57	87.5 ±7.35	110.5 ±5.54	8.33	0.007
Packed cell volume (PCV) (l/l)	0.29 ±0.019	0.29 ±0.019	0.36 ±0.017	0.025	0.025
Mean corpuscular volume (MCV) (fl)	49.3 ±1.14	52.4 ±2.59	59.4 ±2.23	3.03	0.014
Mean corpuscular Hb (MCH) (pg)	14.9 ±0.29	15.6 ±0.96	17.9 ±0.83	1.095	0.042
Mean corpuscular Hb conc. (MCHC) (g/l)	303 ±2.4	297 ±4.9	301	5.2	0.490

Mean serum Fe and blood Hb values of NC and CF piglets were lower than for OM piglets (P = 0.073 and 0.007, respectively). For NC and CF piglets, two thirds of serum Fe values were within or below a marginal band of 2.7 – 10.7 µmol/l suggested by Underwood and Suttle (1999) for assessing the risk of iron deficiency, while half of the Hb values were below a threshold of 80 g/l used by Egeli *et al.* (1998) to differentiate between normal and anaemic piglets. Mean PCV values of NC and CF piglets were significantly lower than for OM piglets (P = 0.025) which, combined with low Hb values, also indicated that some of the NC and CF piglets were mildly anaemic. Mean growth rates from birth to 28 days of NC, CF and OM sample piglets (251 ±12.8, 250 ±10.4, 280 ±10.2 g/d, respectively; mean ±s.e.m.) were not significantly different (P > 0.05). These results suggest that, under the conditions of this experiment, the Fe supplement administered in accordance with current industry practice did not prevent Fe deficiency in all piglets. However, OM piglets apparently derived sufficient additional Fe from soil in the OM to prevent Fe deficiency and to maintain haematological indices within normal ranges. Low Fe stores at birth may have contributed to the Fe deficiency seen in NC and CF piglets at 28 days but this possibility was not tested in this experiment. Our results are in disagreement with many other studies, e.g. Hill *et al.* (1999) who found that one injection of 200 mg of iron dextran was sufficient to maintain adequate growth and haemoglobin concentration of >100 g/l at weaning (21 days). Therefore further work is required to determine whether our results are repeatable and if so to investigate the causes of the observed Fe deficiency.

Supported in part by Australian Pork Limited.

References

EGELI, A.K., FRAMSTAD, T., and MORBERG, H. (1998). *Acta Vet. scand* **39**:381-393.

HILL, G.M., LINK, J.E., MEYER, L. and FRITSCHE, K.L. (1999). *Journal of Animal Science* **77**:1762-1768.

UNDERWOOD, E.J. and SUTTIE, N.F. (1995). 'The Mineral Nutrition of Livestock', 3rd edn (CABI International: Wallingford, UK).

Piglet exposure to soil before weaning reduces the post-weaning growth check and increases carcass weight

H.G. Payne*, B.P. Mullan*, R.R. Nicholls*, S.M. McCullough* and J.R. Pluske**

Department of Agriculture, South Perth, WA 6151. * Murdoch University, Murdoch, WA 6150.

Weaner pigs produced outdoors have been reported to experience less of a growth check after weaning and to dress out heavier than their indoor counterparts reared in conventional or deep-litter pens (Payne *et al.*, 2003). Pre-weaning nutritional differences that occur between indoor and outdoor production systems may influence subsequent performance. This study was carried out to investigate, in isolation from other physical and behavioural variables, the effects of offering substrates to piglets farrowed indoors that are commonly only available to piglets farrowed outdoors.

Two replicates of 12 multiparous crossbred sows were batch-farrowed in an environmentally controlled room maintained at 22ºC in conventional slotted-floor crates with a creep area equipped with a solid floor, hover, heat lamp, 600 mm long feeder and nipple drinker. Piglets were weighed, ear notched, tooth-clipped, tail-docked and injected intramuscularly with 200 mg of iron dextran at 1-2 days of age. Litters were equalised at 10 piglets per sow, although insufficient piglets were available to balance litters for birth weight and sex. After stratification for birth date, litters were allocated randomly to one of three pre-weaning nutritional treatments: 1) control - no creep feed (NC), 2) commercial creep (CF) fed as crumbles and 3) an 'outdoor' mix (OM) of 25 parts soil, five parts sow feed, and one part straw. Treatments were offered from 7 to 28 days of age when litters were weaned and transferred into conventional weaner and grower/finisher pens where they were maintained in litter groups and managed identically until slaughter at 105 kg live weight. Pelleted feed was offered *ad libitum* in a six diet, phase-feeding program during the grow-out phase. Male pigs were immunocastrated using Improvac™ (Pfizer Australia). Six pigs were slaughtered from each treatment on days 28 and 35 and sections taken from the small intestine for histological examination. Differences between treatment means were tested using analysis of variance (Genstat 8th Edition, Lawes Agricultural Trust 2005) with litter as the experimental unit and birth weight, sex and fostering as covariates.

Table 1. Growth and carcass characteristics of pigs offered no (NC), ad libitum creep feed (CF) or an 'outdoor mix' of soil, sow feed and straw (OM) from 7 days of age until weaning at 28 days of age.

	NC	CF	OM	s.e.d.	P value.
Gain birth to weaning at 28 d (g/d)	265	268	272	10.6	0.764
Gain from 28 d to 35 d (g/d)	70	118	112	16.7	0.011
Gain birth to finish (g/d)	677	677	693	16.7	0.238
Hot carcass weight – Trim 13 (kg)	70.4	70.3	71.8	0.385	0.055
Dressing %	66.0	65.9	66.7	0.358	0.047
Carcass P2 (mm)	12.5	12.8	13.2	0.486	0.357
Feed disappearance 7 to 28 d (g/pig)	nil	449	530	53.0	0.171
Feed disappearance 28 to 35 d (g/pig)	1221	1381	1467	143.9	0.257

Piglets offered CF or OM grew faster (P=0.011) in the week after weaning. Overall performance was similar for all treatments (p >0.05) but OM pigs had heavier carcasses and higher dressing percentages than the other treatments (P=0.055 and 0.047, respectively). Villous height and crypt depth were not significantly different between NC, CF and OM piglets at 28 days (425, 499, 461 μm ± 38.3 and 175, 173,187 μm ± 21.8, respectively; mean ± s.e.m.) or at 35 days (280, 357, 288 μm ± 27.1 and 319, 308, 312 μm, respectively; mean ± SEM). However, the decrease in villous height and increase in crypt depth at day 35 compared to day 28 were significant (P <0.001), indicating that the pre-weaning treatments imposed did not prevent major changes to gut architecture from occurring during the week after weaning. A potentially confounding factor in this study was the lower Hb content at weaning in the blood of pigs not fed the OM during lactation (Payne *et al.*, 2005), despite all pigs receiving parenteral iron dextran after farrowing. The implication of the difference in Hb levels at weaning on subsequent performance is unknown.

Supported in part by Australian Pork Limited.

References

PAYNE, H., MULLAN, B., NICHOLLS, R., McCULLOUGH, S. and PLUSKE, J. (2003). In 'Manipulating Pig Production IX', pp. 125, ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

PAYNE, H., MULLAN, B., NICHOLLS, R., McCULLOUGH, S., PLUSKE, J. and CLARK, P. (2005). In 'Manipulating Pig Production X', p. 155, ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

Australian Pork

The impact of repeated out-of-feed events on grower pig performance

M.C. Brumm and S. Colgan

University of Nebraska Haskell Agricultural Laboratory, Concord, NE 68728, USA.

Growing pigs are frequently faced with empty feeders in facilities designed and managed for *ad libitum* access to feed. These facilities generally have feed storage devices and automatic feed delivery mechanisms designed to keep feed in feeders at all times. Automated feeding systems (Robertson *et al.,* 2002) and repeated out-of-feed events have been related to ulcers (Ayles *et al.,* 1999) and gastric torsion (Morin *et al*., 1984). The causes of major out-of-feed events include human errors in the ordering and delivery of feed from commercial mills and bridging of feed in feed storage devices. Diets based on corn and soybean meal tend to have ingredients of low micron size stimulate improvements in feed efficiency (Wondra *et al.,* 1995). In addition, these feeds have relatively high levels of added fat, particularly during warm summer weather. Both low micron size and high fat content increase the angle of repose for diets in meal form and could be a major reason for increased bridging in feed storage devices (Groesbeck *et al*., 2003).

An experiment was carried out to examine the impact of repeated out-of-feed events and feed particle size on grower pig performance in a fully slatted wean-to-finish facility with 15 pigs and one two-hole wean-to-finish feeder (35 cm wide hole) per pen using a 2 x 2 factorial design with four pens per treatment combination. Out-of-feed events occurred either never or once per week from 1200 h to 0800 h on a random day each week. Complete diets had a mean particle size of 900 microns (fine) or 1200 microns (coarse). At each bi-weekly weighing, pigs were evaluated by two observers for skin lesions, tail biting and lameness. There was no effect of any treatment combination on any of these welfare indices.

Table 1. Effect of diet particle size and out-of-feed (OOF) events on grower pig performance.

	Grind		OOF			P-value		
Pig wt, kg	Coarse	Fine	Never	Weekly	SE	Grind	OOF	Grind x OOF
0 d	23.7	23.7	24.1	23.3	0.3	0.889	0.098	0.041
39 d	55.7	55.3	57.5	53.6	0.7	0.661	0.001	0.170
CV of pig wt within pen, %								
0 d	16.3	17.3	17.6	16.0	1.5	0.635	.470	0.704
39 d	11.9	12.9	12.5	12.2	0.9	0.456	.824	0.797
ADG (g)	820	812	856	776	12	0.625	.0005	0.625
ADF (kg)	1.806	1.771	1.861	1.716	0.021	0.270	.0003	0.255
G:F	0.454	0.459	0.460	0.453	0.004	0.505	.192	0.483

References

AYLES, H.L., FRIENDSHIP, R.M., BUBENIK, G.A. and BALL. R.O. (1999). *Canadian Journal of Animal Science* **79**:179-185.

GROESBECK, C.N., GOODBAND, R.D., TOKACH, M.D., NELSSEN, J.L., DRITZ, S.S., LAWRENCE, K.R. and HASTAD, C.W. (2003). *Kansas State University Swine Day*, Manhattan, Kansas, USA. pp. 203-208.

MORIN, M., SAUVAGEAU, R., PHANEUF, J.B., TEUSCHER, E., BEAUREGARD and LAGACE., A. (1984). *Canadian Veterinary Journal* **25**:440-442.

ROBERTSON, I.D., ACCIOLY, J.M., MOORE, K.M., DRIESEN, S.J., PETHICK, D.W. and HAMPTON, D.J. (2002). *Preventative Veterinary Medicine* **53**:293-303.

WONDRA, K.J., HANCOCK, J.D., BEHNKE, K.C., HINES, R.H. and STARK, C.R. (1995). *Journal of Animal Science* **73**:757-763.

Evaluation of wireless sensors to measure the effect of microclimate on pig body temperature

I. McCauley, B. Matthews, L. Nugent, A. Mather and J. A. Simons

Department of Primary Industries, Attwood, Vic. 3049.

Intensive monitoring of agricultural systems is now possible with the development of *ad-hoc* wireless sensor network (AWSN) technology and the decreasing size and cost of sensors. The problems associated with gathering real-time data from sensors placed on mobile animals can be addressed by adding wireless capability to the monitoring devices. Such intensive, real-time monitoring of complex systems can reveal valuable information. This study was carried out to evaluate the suitability of AWSN software and hardware for monitoring the microclimates and body temperature of pigs fitted with wireless-enabled thermistor sensors.

Four boars (45-50 kg) were each surgically implanted with two thermistors (YSI44106, Omega, Manchester, UK) in the side of the neck, one placed just under the surface of the skin (Surface) the other, about five centimetres into the neck muscle (Core). Sensor wires were externalised and connected to a Mica2Dot mote (Crossbow Technologies, San Jose, USA) mounted in a collar on the back of the pig. The animals were housed individually at two locations at extreme ends of a set of pens in a grower/finisher facility. Wireless temperature sensors connected to Mica2Dot motes were placed next to each pen at pig level and a combined temperature and humidity sensor (Crossbow) was placed in a central, elevated location. The management software for the pig sensors was the open-source application TinyDB (Madden *et al.*, 2003). The environment sensors were managed by Sensicast Developers Version (Sensicast, Needham MA, USA). Sensor data were gathered every 32 seconds from the pig sensors and every five minutes from the environment sensors. Data were collected for nine days from three of the pigs and for six days from the fourth pig. The environment network functioned for 15 days before being shut down. The average core temperature from all pigs was 39.0 ± 0.3°C. The surface temperature was on average 0.6°C lower than the core. While the core temperatures of individual pigs fluctuated, there was no discernible pattern with the normal environmental fluctuations in temperature or humidity. Core and surface temperatures were closely correlated (R^2 =0.947, n=9490). However, on one occasion, a sharp increase in the gradient between the core and surface sensors representing a 3-4°C drop in the surface temperature was noted in two of the pigs at one end of the shed (Figure 1A).

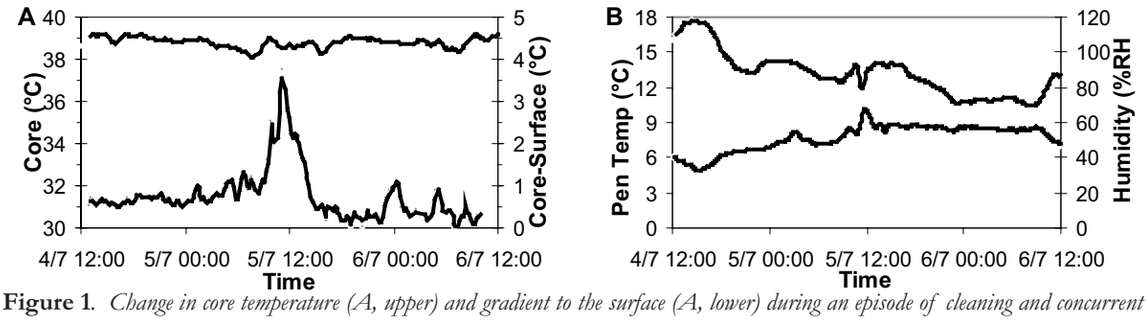


Figure 1. *Change in core temperature (A, upper) and gradient to the surface (A, lower) during an episode of cleaning and concurrent changes in shed humidity (B, lower) and pen temperature (B, upper).*

The temperature difference was associated with an elevated humidity noted in the shed sensor node and by a sharp drop in the local temperature in both pens at one end of the shed (Figure 1B). Retrospective analysis showed that this event occurred following an episode of shed cleaning and presumably reflected chilling following wetting. Environment data from both pens and measures of body temperature from the pig at the other end of the shed showed no evidence of local cooling. This study showed that for healthy pigs in standard housing the welfare of the animals, as indicated by the consistent relationship between core and surface temperature, was unaffected by normal environmental variation. However, the instrumentation enabled changes in the relationship between core and surface temperature that were related to occasional extreme environmental events to be determined, even when these events were not monitored directly. This study shows the capacity of sensor networks to monitor pigs and their local environment intensively to detect environmental interactions with pig physiology.

References

MADDEN, S., FRANKLIN, M., HELLERSTEIN, J. and HONG, W. (2003). *Proceedings of the ACM SIGMOD International Conference on Management of Data*, San Diego, USA, pp. 491-452.

Exogenous progesterone fails to increase circulating levels in pregnant sows

L.J. Johnston,* R.J. Smits** and W.C. Morley**

*West Central Research and Outreach Centre, University of Minnesota, Morris, MN 56267, U.SA. **QAF Meat Industries, Corowa, NSW 2646.

Reproductive failure is an important cause of culling females in a swine breeding herd, contributing 14% of sows not culled for age (Hughes and Smits, 2002). We have found that plasma progesterone (P_4) concentration is unaffected by housing system or sow age when measured in mid-gestation in sows confirmed pregnant (Johnston *et al.,* 2005). The average plasma P_4 concentration recorded was 19.1 $\pm$ 0.58 ng/ml. This was recorded during mid-gestation in spring, a time known as a high fertility period for breeding sows in Australia. Social stresses are associated with increased rate of pregnancy failure (Peltoniemi *et al.,* 2000) and may be mediated through a decline in plasma P_4. This may occur during seasonal infertility or when sows are relocated or mixed in groups after pregnancy diagnosis as part of a management system. Sows may benefit from supplementation of P_4 during these stressful periods. Lopez-Gatius *et al.* (2004) reduced pregnancy loss in dairy cattle with supplemental P_4. Our long-term aim is to reduce pregnancy failure during stressful periods with P_4 supplementation. Before supplementation protocols can be established, the influence of exogenous P_4 on plasma P_4 and reproductive performance must be determined.

We hypothesised that supplemental oral and injectable P_4 would increase plasma levels in pregnant sows. Thirty-two sows (Large White x Landrace F1) of mixed parity were housed in individual stalls and confirmed pregnant by real-time ultrasound 41 $\pm$ 3 days after mating. The experiment began in February during the period of expected suboptimal fertility. Sows received: no supplementation (CON); 20 mg altrenogest daily (LOW); 40 mg altrenogest daily (HI) or 125 mg P_4 in one intramuscular injection (INJ; Jurox Progesterone®). Oral doses of altrenogest were supplied by Regumate® which began 42 days after mating and continued for seven days. Sows assigned to INJ received one injection on day 42. Blood was sampled on days 41, 48, 51, and 55 after mating for plasma P_4 levels. Data were analysed by least squares analysis of covariance with repeated measures in time (plasma P_4) and minimum chi-square (farrowing rate).

Table 1. Effect of exogenous P_4 on plasma levels (ng/ml)[1] and farrowing rate.

	Day post-mating[2]					No. sows	No. preg.	
Treatment	41	48	51	55	Mean[3]	farrowed[4]	failures[4]	Other[4]
CON	20.1	16.1	15.5	17.3	16.3	6	2	0
LO	14.7	9.9	14.1	14.2	12.8	7	0	1
HI	18.3	11.2	16.1	11.6	13.0	5	3	0
INJ	19.0	16.5	12.9	13.2	14.2	8	0	0

[3](LO, HI < CON, INJ; P < 0.10; Pooled SE for treatment = .92); [4](Minimum chi-square = 16.27; P < 0.01).

Oral P_4 depressed (P < 0.10) mean plasma P_4 compared with CON and INJ (Table 1). LOW and HI sows had lower (P < 0.01) P_4 concentrations on day 48, the last day of oral supplementation, compared with CON and INJ sows. Progesterone concentrations following the withdrawal of oral P_4 (days 51 and 55 after mating) were not influenced by treatment. Mean concentration of P_4 in CON sows (16.3 ng/ml) was less than 19 ng/ml, the level we hypothesised was a stable physiological P_4 level in the spring, a season of high fertility. Supplementation regimen influenced (P < 0.01) proportion of sows that farrowed. These results suggest that oral doses of altrenogest depress plasma P_4 concentrations from 42 to 55 days after mating and can have detrimental effects on maintenance of pregnancy in sows. The efficacy of injectable P_4 to influence plasma P_4 is not clear since blood was not collected until 6 days after P_4 injection. A more rigorous sampling protocol is necessary to clearly determine the influence of injectable P_4 on plasma concentrations. Oral dosing with altrenogest is not an effective means of supplementing pregnant sows with P_4. Injectable and other methods of exogenous supplementation merit further investigation.

References

HUGHES, P.E. and SMITS, R.J. (2002). APL Report no. 1611.

JOHNSTON, L.J., SMITS, R.J. and MORLEY, W.C. (2005). In 'Manipulating Pig Production X', p. 160. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

LOPEZ-GATIUS, F., SANTOLARIA, P., YANIZ, J.L. and HUNTER, R.H.F. (2004). *Theriogenology* **62**:1529-1535.

PELTONIEMI, O.A.T., TAST, A., and LOVE, R.J. (2000). *Anim Reprod Sci* 60-61:173-184.

Neither housing system nor age change circulating progesterone concentrations in pregnant sows

L.J. Johnston,* R.J. Smits** and W.C. Morley**

*West Central Research and Outreach Centre, University of Minnesota, Morris, MN 56267, U.S.A. **QAF Meat Industries, Corowa, NSW 2646.

Reproductive failure is an important cause of culling females in a swine breeding herd. Pregnancy failure characterised by irregular returns to oestrus and sows non-pregnant at expected farrowing can account for 14% of non-age based culling of sows (Hughes and Smits, 2002). While several factors could be responsible for this high incidence of pregnancy failure, progesterone (P_4) status of the sow may be an important physiological control point. Mixing sows following positive pregnancy diagnosis 35 days after mating may depress plasma P_4 due to social stress and increase incidence of pregnancy failure (Peltoniemi *et al.*, 2000). Lopez-Gatius *et al.* (2004) supplemented lactating dairy cattle with P_4 and reduced pregnancy loss during the early fetal period. Sows may benefit from supplementation of P_4 during periods of known stress. Our ultimate aim is to develop a regimen of P_4 supplementation that might prevent spontaneous declines in P_4, such as may occur during stress or seasonal infertility. Before supplementation protocols can be established for evaluation, normal circulating concentrations of P_4 during pregnancy must be determined.

We hypothesised that the concentration of plasma P4 would not be different in pregnant sows housed either in individual stalls, or small and large groups. Sixty Large White x Landrace F1 cross sows were studied that were 42 $\pm$ 3 days after mating and confirmed pregnant by real-time ultrasound. Twenty sows were housed in each of three types of gestation housing: individual stalls; small groups (10/pen); and large groups (80/pen). All sows were individually stalled from weaning to mating. Sows were moved into either stalls in a separate location or mixed into small groups or large groups on day 40, day 40 or day 7 of gestation, respectively. Within each housing type, young sows (parity 0; n = 10) and old sows (parity 2-3; n = 10) were selected randomly. All sows received 2.4 kg/day of a pelleted diet containing 13 MJ DE/ kg and 140 g crude protein/kg. Blood was collected by jugular venipuncture from sows on days 42, 49 and 56 after mating into non-clot vacutainer tubes for analysis of plasma P_4 concentration. Progesterone concentration of plasma was analysed statistically by least squares analysis of variance in a 3 x 2 factorial arrangement of treatments with repeated measurements in time.

Plasma P_4 concentration was not influenced by housing type, sow age, or stage of gestation (Table 1). Plasma P_4 concentrations of individual sows ranged from 16.9 to 21.6 ng/ml. The average P_4 concentration over all sows and sampling days was 19.1 $\pm$ 0.58 ng/ml. It appears that sows from 42 to 56 days after mating closely regulate plasma P_4 to stay very near 19 ng/ml. This experiment was conducted during a season of high fertility (spring). Incidence of pregnancy failure was observed in only two of 60 sows and was unrelated to treatment.

Table 1. Plasma P_4 concentration (ng/ml) of pregnant sows by housing type and age.

	Day after mating[1]			
Treatment[2]	42	49	56	Significance
Housing type:				P < 0.90
Individual	18.4	19.7	17.8	
Small group	19.0	19.2	20.0	
Large group	19.3	18.7	19.6	
Age:				P < 0.50
Parity 0	18.7	18.1	19.0	
Parity 2-3	19.1	20.2	19.3	

[1](P < 0.80; Pooled SE = .31) [2](Pooled SE = 1.42)

We conclude that age and housing type during mid-gestation have no influence on plasma P_4 concentration of sows, thereby supporting our hypothesis. Furthermore, plasma P_4 levels during mid-gestation are stable with a concentration of 19 ng/ml in pregnant sows.

References

HUGHES, P.E. and SMITS, R.J. (2002). Final Report 1611, Australian Pork Limited, Canberra.

LOPEZ-GATIUS, F., SANTOLARIA, P., YANIZ, J.L. and HUNTER, R.H.F. (2004). *Theriogenology* **62**:1529-1535.

PELTONIEMI, O.A.T., TAST, A., and LOVE, R.J. (2000). *Anim Reprod Sci* 60-61:173-184.

Paylean® improves feed conversion efficiency of entire and immunocastrated male pigs

K.L. Moore*, B.P. Mullan*, D.P. Hennessy**, F.R. Dunshea*** and D.N. D'Souza****

*Department of Agriculture, South Perth, WA 6151. **Pfizer, Parkville, Vic. 3052. ***Department of Primary Industries, Werribee, Vic. 3030. ****Alltech Biotechnology, Dandenong South, Vic. 3175.

Paylean® (ractopamine) is an in-feed β-agonist that increases the rate of protein deposition in pigs. It is absorbed into the blood stream and binds to specific beta receptors on the cell membrane of muscles where its primary response is to increase the size of muscle fibres (Dunshea and Walton, 1995). Paylean® is added to the diet of finisher pigs for the last 28 days before slaughter. It is currently included at a rate of 5 ppm for the 28 days however, it is known that after three to four weeks there is a down-regulation of the beta-receptors. This has driven interest in 'step-up' programs where the concentration of Paylean is periodically increased (eg. 5 ppm Paylean® for the first two weeks, followed by 10 ppm Paylean® for the next two weeks). Most of the research on Paylean® has been carried out on females and surgically castrated male pigs. However, little is known about the effect of Paylean® supplementation (both normal and step-up programs) on the growth performance and carcass quality of pork from immunologically castrated male pigs. The immunocastration of entire male pigs is a standard practice in many Australian commercial piggeries. The aim of this experiment was to determine the effect of two levels of Paylean® supplementation on the growth performance and carcass quality of entire and immunocastrated male pigs.

Sixty Large White x Landrace x Duroc crossbred entire male pigs were used in a 2x3 factorial experiment. The main treatments were (i) sex (S) – entire male pigs and immunological castrate male pigs (Improvac® vaccination was administered at 13 weeks of age and at five weeks before slaughter) and (ii) Paylean® dose (P) – control (0 ppm Paylean), Paylean 5 (5 ppm Paylean for 26 days before slaughter) and Paylean 5/10 (5 ppm Paylean for 14 days followed by 10 ppm Paylean for a further 12 days). The pigs were housed individually from 45 kg live weight. The pigs were weighed and voluntary feed intake was recorded weekly. At about 114 kg live weight the pigs were slaughtered using standard commercial practices and the carcass weight, depth of back fat (Hennessy probe) and dressing percentage were determined. All data were analysed by ANOVA.

Table 1. Effect of Paylean® supplementation for 26 days before slaughter on the growth performance and carcass quality of entire and immunocastrated male pigs (n=10).

Sex	Entire			Improvac				Significance		
Paylean (ppm)	0	5	5/10	0	5	5/10	SED[a]	S	P	SxP
Daily gain (kg/day)	1.08	1.19	1.21	1.21	1.26	1.31	0.076	0.026	0.102	0.856
Feed intake (kg/day)	3.31	3.55	3.29	3.92	3.61	3.78	0.169	<0.001	0.809	0.064
FCR (kg/kg)	3.07	3.08	2.75	3.27	2.87	2.91	0.186	0.680	0.046	0.242
Dressing %	63.6	65.9	64.5	63.8	63.9	64.1	1.61	0.450	0.582	0.606
Carcass weight (kg)	71.9	75.2	74.6	72.6	74.6	74.2	2.45	0.942	0.278	0.916
P2 (mm)	12.9	14.3	14	15.3	14.3	14.5	0.31	0.204	0.975	0.395

[a]s.e.d. for Sex x Paylean dose

Immunocastrated male pigs had a higher daily gain and a higher feed intake than entire males (Table 1). The inclusion of Paylean® in the diet significantly improved the feed conversion ratio for both entire and immunocastrated male pigs. There was also a trend for Paylean® supplementation to improve the average daily gain (P=0.102).

These findings indicate that Paylean® supplementation improved feed conversion efficiency in entire and immunocastrated male pigs. The use of a step-up program of Paylean® did not appear to improve growth performance and carcass quality further when compared to normal Paylean® supplementation. However, the behaviour and feed intake of individually housed pigs are known to differ from those of group-housed pigs and so group-housed pigs may respond differently to a step-up program of Paylean® compared to pigs housed individually.

References

DUNSHEA, F.R. and WALTON, P.E. (1995). In 'Manipulating Pig Production V', pp. 42-51, eds D.P. Hennessy and P.D. Cranwell. (Australasian Pig Science Association: Werribee).

Influence of the form and level of copper and zinc suplementation on mineral status of grower and finisher pigs

A. Hernández*, B. P. Mullan**, K. Turnley***, D.N. D'Souza*** and J.R. Pluske*

*Murdoch University, Murdoch, WA 6150. **Department of Agriculture, South Perth, WA 6151. ***Alltech Biotechnology Pty Ltd, Dandenong South, Vic. 3175.

Organic forms of copper (Cu) and zinc (Zn) induce higher growth rates than inorganic forms. The higher growth rates of pigs fed organic Cu and Zn are thought to be because organic forms of the minerals are absorbed better than inorganic forms (Coffey *et al*., 1994) and that they may result in greater plasma mineral concentrations (Hahn and Baker, 1993) and/or higher organ mineral stores in pigs (Apgar *et al*., 1995). However, an effect of mineral form on these indices has not always been demonstrated (Wedekind *et al.,* 1994). The aim of this experiment was to compare the effect of Cu and Zn fed in the form of Bioplex® or sulphate at two levels of dietary inclusion on the mineral status of growing and finishing pigs. The experiment was designed as a 2x2 factorial arrangement of treatments, with two mineral forms (organic and inorganic) and two inclusion levels (low and high). The study used 160 female pigs (Large White x Landrace) through the growing and finishing phases (25-107 kg live weight). The 'low' levels aimed to provide 25 ppm of Cu and 40 ppm of Zn per kg, while the 'high' levels aimed to provide 160 ppm of Cu and 160 ppm of Zn per kg. These levels were fed in diets formulated for the growing and finishing phases of growth. The mineral supplement incorporated in the diets contained Cu and Zn sulphate or Bioplex® Cu and Zn (Alltech Biotechnology P/L, Victoria, Australia) according to their required levels in each diet. Pigs were fed *ad libitum*. At 36 and 97 kg live weight (growing and finishing phases, respectively) blood samples were taken from a random sub-sample of four pigs per pen (five pens per treatment). At the end of the experiment the pigs were slaughtered as per commercial procedures, and one foretrotter per pig along with samples of liver and kidney were collected from the same pigs that blood was sampled from during the trial. Data were analysed by two-way analysis of variance.

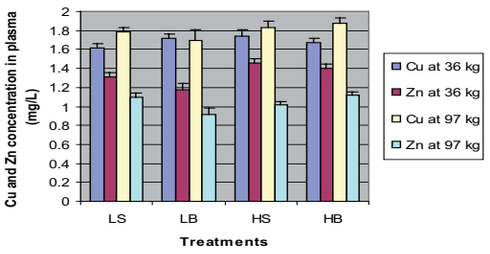
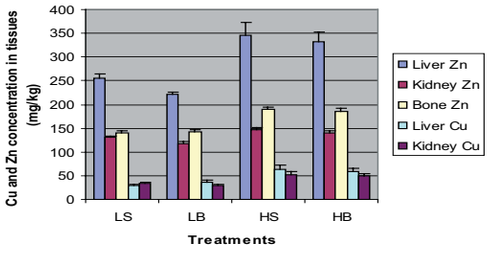


Figure 1. *Cu and Zn plasma levels (±SEM).*



*Cu and Zn levels in liver and kidney measured in 20 samples/ treatment, Zn in bone in 10 samples/treatment.

Figure 2. *Cu and Zn tissue levels* (±SEM).*

Levels of Cu and Zn in plasma were within the homeostatic ranges for pigs of this weight. At 36 kg, there were no differences in Cu levels between treatments but there was a significant main effect of inclusion level for Zn (P=0.001). At 97 kg, there was no effect of the diets on Cu levels but there was a significant interaction for Zn (P=0.002), with pigs fed LB having the lowest Zn levels (0.92 mg/L), which was similar to pigs fed HS (1.02 mg/L). Concentrations of Cu and Zn in the livers of pigs in all treatments and Zn in the bone of pigs in the low treatments were within the normal range. In addition, the levels of Cu and Zn in the kidneys of pigs in all treatments and Zn in the bone of pigs in the high treatments were high compared to normal levels (26-29 ppm, 56-112 ppm and 95-146 ppm dry weight for Cu and Zn in kidneys and Zn in bone, respectively). Cu and Zn in tissues were affected significantly by inclusion level (P<0.0001). Based on the indicators of Cu and Zn status analysed, a higher absorption for Bioplex® Cu and Zn compared to that of the sulphates was not evident. However a proper bioavailability study is required as factors like initial pig mineral status, assessed indicators of mineral status and composition of the diet may have influenced the response (Wedekind *et al*., 1994).

The financial support of Alltech Biotechnology P/L and Murdoch University are acknowledged.

References

HAHN, J.D. and BAKER, D.H. (1993). *Journal of Animal Science* **71**:3020-3024.

WEDEKIND, K.J., LEWIS, A.J., GIESEMANN, M.A., and MILLER, P.S. (1994). *Journal of Animal Science* **72**:2681- 2689.

COFFEY, R.D., CROMWELL, G.L. and MONEGUE, H.J. (1994). *Journal of Animal Science* **72**:2880-2886.

APGAR, G.A., KORNEGAY, E.T., LINDEMANN, M.D. and NOTTER, D.T. (1995). *Journal of Animal Science* **73**:2640-2646.

Pre-and post-weaning growth in relation to creep feed consumption of individual piglets

J.C. Kim*, B.P. Mullan**, H.G. Payne**, J. Callesen***, R.H. Wilson**** and J.R. Pluske*

*School of Veterinary and Biomedical Sciences, Murdoch University, Murdoch, WA 6155; ** Department of Agriculture, South Perth, WA 6151; ***Danish Bacon and Meat Council, Axeltorv 3, DK-1609, Copenhagen, Denmark; **** Wandalup Farms, Mandurah, WA 6210.

Consumption of creep feed pre-weaning is thought to stimulate food intake and growth after weaning (Bruininx *et al.*, 2004). Creep feed intake has traditionally been assessed on the basis of average intake per piglet, which is erroneous because it assumes that all piglets in a litter eat the same amount of feed. Coloured markers can be used in the feed to distinquish between piglets eating different amounts of creep feed by assessing how many times faeces of each piglet is stained with the marker. (Bruininx *et al.*, 2004). In this study we determined whether creep feed intake of individual piglets could be determined qualitatively during lactation using a dye related to post-weaning performance.

Six Large White X Landrace sows and their litters were used. Litter size was equalised to 10-11 piglets per sow within 36 hours of farrowing. At day 12 of lactation, a creep feed containing a dye (Indigo carmine, 5 g/kg of diet) was introduced. The occurrence of dye in the faeces and the weight of all piglets were assessed on day 19, 23, 27 and 31, when piglets were weaned. Post-weaning growth rates were determined on days 34, 38, 45, 59 and 109. Piglets were categorised in lactation as 'good-eaters', 'moderate-eaters' and 'small-eaters' according to the number of times that coloured faeces were observed (4, 2-3 and 0-1, respectively, for 'good', 'moderate' and 'small eaters'). The ANOVA analysis of Statview 5.0 for Windows (SAS Inc.) was used.

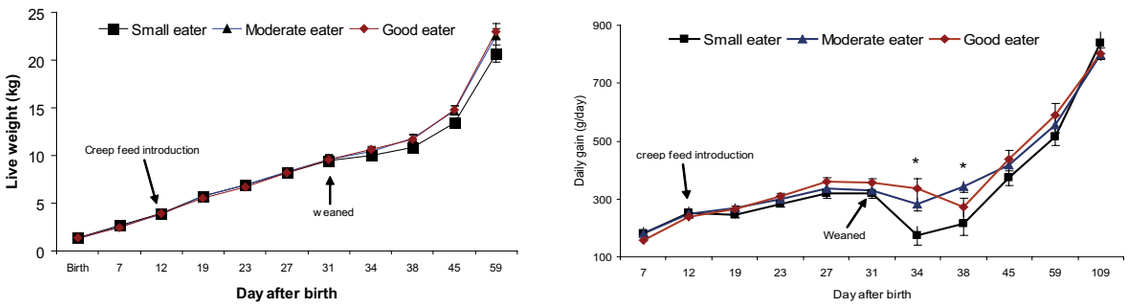


Figure 1. *Effect of creep feed consumption on the pre- and post-weaning performance of pigs.*

Piglets with variable creep feed consumption did not differen in birth weight. Piglets categorised as good-eaters at the end of lactation had numerically lower daily gains in the first 12 days of lactation (238, 248 and 253 g/day for good, moderate and small-eaters, respectively, P>0.05). However, after the introduction of creep feed on day 12, the average daily gain of good-eaters increased to reach 356 g/day on day 31, which was 40 g/day higher than the small-eaters (P<0.05). After weaning on day 31, the growth rate of small-eaters was significantly decreased (P<0.01), most likely due to the fasting of this group. The live weight difference between good-eaters and small-eaters was in excess of 2 kg (P>0.05) up to 4 weeks after weaning. On day 109, however, the differences in weight between three groups had disappeared (63, 62 and 63 kg for good, moderate and small-eaters, respectively). The results suggest, albeit without a statistical significance due to a small number of animals used, that creep feed intake during lactation has no effect on growth rate but piglets not consuming creep feed suffer a growth-check up to four weeks after weaning. These data also suggest that a qualitative technique based on the frequency of faecal staining can be used to distinguish piglets eating different amounts of creep feed.

The Australian Research Council, Danish Bacon and Meat Council and Wandalup Farms are thanked for their support with this work.

References

BRUININX, E.M.A.M., SCHELLINGERHOUT, A.B., BINNENDIJK, G.P., VAN DER PEET-SCHWERING, C.M.C., SCHRAMA, J.W., DEN HARTON, L.A., EVERTS, A and BEYNEN, A.C. (2004). *Animal Science* **78**:67-75.

Achieving production and economic targets on farm: The Premier Pig Program™

W. H. Close*, K. Turnley** and B.P. Mullan***

*Close Consultancy Ltd, Wokingham RG41 2RS, UK. **Alltech Biotechnology Pty Ltd, Dandenong South, Vic. 3175. *** Royston Consulting Australia Pty Ltd, Bassendean, WA 6054.

The Premier Pig Program™ has been developed by Close and Turnley (2004) to provide user-friendly technical information and support to all sectors of the pig industry. The program is presented as a series of interactive workshops and also comprises a manual that provides details on the nutrition, management, housing and healthcare requirements for all classes of pigs.

The Premier Pig Program™ was initially introduced in Australia and New Zealand and has now been developed in many Asia-Pacific countries. At the workshops producers are able to benchmark their levels of productivity against target commercial and intervention levels of performance. This is best illustrated in relation to key aspects of sow productivity from thousands of sows in each country (Table 1). In this way, the major areas where performance is below acceptable levels can be identified and actions suggested.

Table 1. Summary of aspects of sow productivity in different Australasian countries

Country	AUS	NZ	China	Japan	Thailand	Vietnam	Target	Inter-vention
Culling rate (%)	30-60	30-50	10-50	30-50	15-40	10-40	35	>42
Average parity	3-3.5	2.5-3.5	4-6	3-5	3-5	3-4	4	<3 >6
Sow mortality (%)	2-14	2-20	1-10	2-5	2-5	1-15	<5	>5
Farrowing rate (%)	75-90	75-90	50-90	75-95	70-95	60-90	90	<83
Litters/sow/year	2.0-2.5	1.8-2.4	1.9-2.4	2.2-2.4	2.1-2.5	1.9-2.3	2.4	<2.2
Wean-Mating interval (d)	5-10	5-7	5-10	4-10	5-15	3-15	5	>7
Sows mated <7 d (%)	70-95	85-95	75-90	90	75-95	-	90	<85
Empty days/sow/year (d)	25-60	30-60	20-60	11-50	10-60	5-60	15	>20
Piglets born/litter (total)	11-12.5	10-13	10-13	10.5-13	9.5-13	9-12	12.0	<11.0
Piglets born alive	9.7-11.5	9-12	8-12	10-12	8-11	8-11	11.3	<10.0
Mean piglet birth wt (kg)	1.0-1.5	1.4-1.7	1.1-1.6	1.2-1.6	1.1-1.5	1.0-1.5	1.4	<1.1
Pre-wean mortality (%)	7-20	10-20	5-20	3-10	4-15	5-10	10.0	>13.0
Piglets weaned/litter	8-10.5	8.5-11	8-12	9-11	7-10	8-10	10.2	<9.5
Piglets weaned/sow/year	16-26	16-25	17-23	20-25	14-25	16-22	24.5	<21
Age at weaning (d)	14-32	21-32	21-42	19-25	18-28	21-28	25	25
Piglet weaning wt. (kg)	4-10	7-9	5.5-8	5.5-7	5-8	5-7	7	<6
Feed/sow/year (t)	1.1-1.6	1.3-1.5	0.8-1.2	1.0-1.2	0.9-1.1	0.8-1.1	1.10	<1.00
Cost per empty day (local currency)	3.9	3.4	22	500	35	20,000		

The major areas of sow productivity that were below target or intervention levels of performance were similar across countries and were: culling rate, farrowing rate, litters per sow per year and per lifetime, empty or non-productive days, piglets born, born alive and weaned and piglet birth and weaning weight.

The trait most affected was the number of piglets weaned per sow per year. This is a function of the number of piglets reared per litter and the number of litters produced per year. The latter is dependent on lactation length and the period between weaning and mating. Usually, a seven day period is allowed for the sow to return to oestrus and be mated; anything longer than this will unduly increase parity length and hence reduce litters per sow per year. These extra days are termed empty or non-productive days and are expensive (Table 1). These calculations demonstrate the considerable economic loss associated with just one aspect of reduced sow productivity and highlight the usefulness of the Premier Pig Program in establishing such relationships and in suggesting practical solutions to improve performance on farm.

References

CLOSE, W.H. and TURNLEY, K. (2004). In Nutritional Biotechnology in the Feed and Food Industries, pp. 113-119, eds T.P. Lyons and K.A. Jacques (Nottingham University Press, Nottingham).

The use of baseline production data in order to benchmark New Zealand with key international trading partners

A.A. Davidson

New Zealand Pork Industry Board, Wellington, New Zealand.

Benchmarking is the search for industry best practices that will lead to superior performance. By establishing operating targets and productivity programmes based on industry best practices, superior performance can be achieved (Camp 1989).

Internationally compared New Zealand has a very small national sow herd and no natural advantage in the production of pork. To remain viable in the long-term the industry must be able to compete globally. The aim of this research was to establish baseline production data with the intention of benchmarking New Zealand's pork production performance with a number of key international trading partners in order to determine New Zealand's competitiveness.

A set of measurable performance indicators was compiled from Denmark, Canada, United States of America and Australia, and a set of key performance indicators for the pork industry was compiled. A national paper based questionnaire was sent out to the 372 pork producers registered with the New Zealand Pork Industry Board (NZPIB) to ascertain baseline data on selected key performance indicators (Table 1). Responses accounted for 12,836 sows, a response rate representing 28.5 % of the industry. Finally, a national system (known as Porkmark) for collecting data, collating it and, most importantly, disseminating it back to individual producers in order to continue to drive productivity increases, was developed.

Table 1. Key production performance indicators for Australia, Denmark, Canada, the United States of America and New Zealand.

	Australia*	Denmark#	Canada**	USA**	New Zealand
Litters per sow per year	2.17	2.24	2.31	2.20	2.06
Number born alive	10.50	12.10	10.29	9.86	11.02
Born dead (%)	8.80	9.00	-	-	8.17
Pre-weaning mortality (%)	13.50	13.20	12.00	10.00	14.58
Pigs weaned per sow per year	19.67	23.52	20.92	19.17	19.46
Post-weaning mortality (%)	5.10	3.60	6.00	6.80	4.44
Pigs finished per sow per year	17.18	22.70	19.66	17.86	18.59
Finisher herd LW feed conversion	2.62	2.89	2.91	3.09	2.22
Ave live weight at sale (kg)	95.30	102.40	113.00	114.00	86.43

(source: *Australian Pork Limited 2003; #Danske Slagterier 2003; **Knowles 2002)

The number of pigs born alive per litter in New Zealand (11.02 pigs) was only surpassed by Denmark with 12.1 pigs. However, this performance was offset by the average farrowing index (2.06 litters per sow per year). This resulted in the average number of pigs weaned per sow per year (19.46 pigs) being significantly lower than Canada and Denmark, and comparable to Australia and the United States of America. A comparably low post-weaning mortality (4.44 %) resulted in 18.59 pigs being finished per sow year in New Zealand.

The data collected from the survey suggests that New Zealand's overall pork production performance was equal to several of its key trading partners but still a long way short of what would be considered world leading. Improving the farrowing index should be the immediate focus of producers to ensure that New Zealand makes demonstrable steps towards to achieving international competitiveness.

References

AUSTRALIAN PORK LIMITED (2003). 'Pig Stats 2002: Australian pig industry handbook', (Australian Pork Limited: Australia).

CAMP, R.C. (1989). 'Benchmarking: the search for best practices that leads to superior performance', (ASQC Quality Press: Milwaukee, USA).

DANSKE SLAGTERIER (2003). 'Annual Report', (National Committee for Pig Production: Copenhagen, Denmark).

KNOWLES, A. (2002). 'Pig cost of production in selected countries', (British Pig Executive, Meat and Livestock Commission: Milton Keynes, United Kingdom).

7 Reproduction

Symposium – Gilt management for lifetime performance

R.J. Smits

QAF Meat Industries, Corowa NSW 2646.

Introduction

In most pig producing countries the reproductive performance of the national herd as measured by pigs weaned per sow per year has been relatively static. However, the rate at which the sow herd is replaced has been increasing (Hughes and Varley, 2003; Levis, 2005). The concern for the pig industry is the increasing proportion of premature sow culls that are occurring on farms. As a consequence, producers are no longer able to retain as many sows in the herd as they previously did through to parities six, seven or eight. The principle causes for premature culling and sow removal are 1) reproductive failure, 2) low reproductive performance in early parities, 3) sow mortality and 4) structural weakness. The objectives of this symposium are to: identify the costs associated with a high sow turnover; review current recommendations for gilt management and; consider alternative management strategies that may promote sow longevity and hence raise lifetime performance.

The first paper of the symposium introduces the biological aspects associated with sow replacement of the herd based including structural selection, age at first mating and parity performance. This paper reviews published economic models that can be used to estimate how producers can achieve an optimum herd parity structure. The author then speculates on the ideal farm-specific economic model – one that would allow a producer to rectify a breeding herd where the structure is sub-optimal.

The second paper reviews the gilt physiology and the biological processes leading to puberty attainment. Recent research is reported in which the authors investigated the response of lean modern genotypes to boar stimulation. These provide new data for practical boar stimulation regimes. This paper also reviews the impact of gilt nutrition on puberty attainment and the recruitment, growth and development of competent oocytes. Evidence is provided to show that the dynamics of follicle growth and oocyte maturation are influenced by subtle changes in nutrition and the metabolic status of the young gilt. It is suggested that the influence of oocyte quality, or developmental competence, on embryo survival could be as relevant to improving reproductive potential as maximising ovulation rate.

The final paper of the symposium addresses the issue of gilt management recommendations for maximising sow reproductive performance and longevity. The authors compare the latest recommendations with those from a previous APSA symposium in 1989 on pre-mating management of the gilt. Using one of the farms of QAF Meat Industries as a case study, they describe the sequence of events that resulted in a different approach to rearing the gilt before mating. The short- and long-term effects of this approach, which involved housing pre-pubertal gilts in deep litter eco-sheds to increase tissue reserves of fat of genetically lean gilts, are then discussed in terms of sow reproductive performance and longevity in the herd.

Gilt management for lifetime performance and sow longevity have been debated in many forums in the past, including APSA. This symposium brings together the economic justification for maintaining the sow in the herd for longer than is currently achieved and presents new findings that show changes to the management of the modern lean gilt may improve her reproductive potential and longevity.

Biological and economical evaluation of sow longevity

D. G. Levis

University of Nebraska-Lincoln Agricultural Research and Development Center, Nebraska.

Abstract

Several scientific studies provide evidence that pork producers are losing income from culling their sows too young. In fact it appears the average commercial sow is just reaching a positive net present value (NPV) before she is culled. Inconsequence, a large amount of profit potential is not realised because the sow does not remain in the breeding herd to produce litters four, five and six. Various methods have been used to estimate the effect of biological traits and economic factors on the lifetime productivity of a sow. Several scientists have retrospectively analysed farm data to determine the major factors influencing culling and lifetime productivity of sows. Other scientists have designed computer programs to help determine the number of parities a sow must produce before a NPV is reached. One particular program generates a sensitivity table of NPVs to account for changes in production traits, cost of replacement gilts, cost of facilities and other variable operating expenses. Although some computer models are fairly robust to allow the user to change many values, the computer models do not effectively simulate the true situation of all pork production enterprises. A simplified profit model that combines pig production and number of days the female is in the breeding herd might be more valuable to a pork enterprise. Depending on many assumptions, the difference in accumulated profit generated between litter three and litter six ranged from $318 to $440 USD per sow. Although there will always be differences in the optimum culling and replacement rates for different farms, the take home message is to keep the sow highly productive for as long as possible.

Introduction

Replacement gilts play a key role in the biological performance and profitability of sow farms and the financial investment in replacement gilts constitutes a major budget decision. At a minimum, breeding females should be kept in the herd until the investment cost associated with their replacement is recuperated. However, a sow farm needs to generate profit to remain viable and the longer a productive sow is kept in the herd the more opportunity she has to generate pigs to enhance farm profit. In this paper I will briefly discuss biological factors involved with sow longevity and evaluate how these influence the profitability of breeding sows.

Biological aspects

Sow wastage

The main reasons for culling sows from the breeding herd are reproductive problems (34 %); age or high parity (19 %); and locomotive disorders (12 %) (Hughes and Varley, 2003). Although locomotive disorders account for 12 % (range 10-19%) of the reasons for culling, pork producers should make sure they are selecting for appropriate structure of the skeleton. Leg disorders have been reported as the second most important reason for culling sows (Cederberg and Jonsson, 1996). Some people would consider leg disorders to be a welfare problem if the condition causes pain to the sow.

Table 1 indicates the various types of leg weaknesses in swine (Gjein and Larssen, 1995a, 1995b, 1995c; Jorgensen, 2000a, 2000b; Kornegay *et al*., 1990). Buck-kneed forelegs, turned out foreleg, turned out rear leg, upright pasterns on hind leg, stiff locomotion, lameness and tendency to slip are clinical signs of osteochrondrosis and osteoarthrosis (Jorgensen, 2000b). The clinical signs of toe lesions are buck-kneed forelegs, upright pasterns, steep hock joints turned out of hind legs, standing under position of rear legs, stiff movements, swaying hindquarters, goose-stepping rear legs, tendency to slip and lameness (Jorgensen, 2000b). Longevity is negatively associated with buck-kneed forelegs, swaying hindquarters and standing under position of rear legs (Jorgensen, 2000a). Often the lateral or outer toes are larger than the medial or inner toes and more lesions are found on the lateral toes (Kornegay *et al*., 1990). Although the influence of unequal sized toes on gilt soundness has been controversial (Arthur *et al*., 1983; Calabotta *et al*., 1982; Penny *et al*., 1980; Kornegay *et al*., 1990), pork producers should select replacement gilts with large, even-sized toes to enter the development phase. Sows can be at a higher culling risk with a lower leg score during the first four parities (Brandt *et al*., 1999).

Table 1. Various types of structural and locomotion soundness problems in swine.

Buck-kneed front legs	Heal-horn erosion
Upright pasterns	Heal-horn junction crack
Weak pasterns	Median horn crack
Unequal size of toes	White-line horn crack
Toes on front legs pointed out	Longitudinal side-wall crack
Toes on front legs pointed in	Osteochondrosis
Sickle-hocked rear legs	Swaying hindquarters
Standing under position of rear legs	Stiff in front movement
Post legged rear legs	Stiff in rear movement
Cow-hocked rear legs	Goose-stepping rear legs

Although several countries have recording programs for benchmarking biological parameters, pork producers still need to evaluate their own records carefully relating to lameness and locomotor problems of breeding animals. Pork producers need to remember that benchmarking records are only averages. Thus, while lameness may account for 12% on a benchmarking records program, between 20-40% of sows that die or are culled can be traced back to foot and leg problems on some farms.

Parity

The average parity of a sow herd has a large effect on herd productivity (Dijkhuizen *et al.*, 1986). Table 2 indicates the average number of piglets born alive per litter, average number of piglets weaned per litter, farrowing rate, fecundity index (farrowing rate multiplied by number of piglets born alive) and weaning age by parity for a pork enterprise in Nebraska. The average number of piglets born alive steadily increased from parity one (10.3) up to parity four (12.0) and then steadily decreased to 10.9 piglets born alive at parities higher than seven. These data indicate economic losses would occur if sows were culled at an early age. The Nebraskan herd was expanding with the average parity of the herd being 2.51. Dijkhuizen *et al.* (1986) stated that most sows are culled not because they are no longer able to produce but because replacement gilts are expected to yield a higher return. This statement does not hold true for the unpublished data generated in the Nebraskan herd. Fecundity index for parity one was always less when compared with fecundity index of parities 2-7+.

Table 2. Average number of piglets born alive, farrowing rate, adjusted farrowing rate, fecundity index and average weaning age by parity (Levis, unpublished data).

Parity	Number of sows [1]	Average number of piglets born alive per litter	Average number of piglets weaned per litter	Farrowing rate, % [2]	Fecundity index [3]	Average weaning age, days
1	1,701	10.3	9.4	90.0	927	16.6
2	1,854	11.2	10.8	90.1	1,009	16.6
3	1,262	11.7	11.2	87.7	1,026	16.7
4	532	12.0	11.0	92.0	1,104	16.5
5	133	11.7	10.5	88.1	1,031	16.7
6	147	11.0	10.0	88.0	968	15.9
7+	283	10.9	9.7	93.4	1,018	16.4

[1] Data were collected from 1 May 1998 to 30 April 1999. [2] Farrowing rate is the percentage of sows farrowed that were served in a specific period of time. [3] Fecundity index is farrowing rate multiplied by litter size born alive x 100

Rodriquez-Zas *et al.* (2003) analysed records from 32 herds (148,568 sows) in the United States. The average lactation length was not reported. The average number of piglets born alive was more for parity one than parities seven, eight, nine, and 10 ($P<0.05$) (Table 3). Also, litter size born alive peaked at parity three and then declined significantly from parity five through to parity 10 ($P<0.05$). The average litter size born alive did not fall below the parity one value until parity seven. The number of pigs weaned peaked at parity two and then declined from parity three through to parity 10. The authors did not explain the decrease in number of pigs weaned after parity two. For purebred Hampshire sows, Tummaruk *et al.* (2001) found that the number of piglets born alive increased from parity one to parity five ($P<0.05$) (Table 3). The average lactation length was 41.0 $\pm$ 6.2 days (range: 25-59 days). Swedish animal welfare legislation requires a minimum lactation length of 28 days. These reports highlight the importance of maintaining sows longer in the breeding herd.

Table 3. Effect of parity on reproductive performance of sows.

Rodriguez-Zas *et al.* (2003)			Tummaruk *et al.* (2001)				
Parity	Number pigs born alive	Number pigs weaned	Parity	Number sows	Number pigs born alive	Number sows	Farrowing rate (%)
1	10.04 [a]	9.07 [d]	1	1,872	8.1 [a]	1,261	63.7 [a]
2	10.48 [a]	9.50 [a]	2	1,438	8.8 [b]	902	69.5 [b]
3	10.79 [b]	9.35 [b]	3	892	9.5 [c]	671	74.1 [c]
4	10.72 [b]	9.15 [c]	4	587	9.7 [cd]	474	70.0 [bc]
5	10.42 [c]	8.89 [e]	5	382	10.0 [d]	310	72.0 [bc]
6	10.18 [d]	8.64 [f]	6, 7 & 8	421	9.7 [cd]	304	76.3 [c]
7	9.90 [e]	8.41 [g]					
8	9.63 [f]	8.18 [b]					
9	9.43 [g]	8.00 [hi]					
10	9.19 [g]	7.95 [i]					

[a, b, c, d, e, f, g, h, i] Values with different superscripts within column differ, P<0.05

Age at first mating

The optimal age for gilts at first mating so that lifetime reproductive performance and longevity are maximised is probably herd-specific. During the lifetime of a breeding sow, many biological and farm management factors influence reproductive performance and longevity. The main factors relate to genetics, housing, environment and nutritional management practices used during the various phases of production - growing and developing, gestation and lactation. Also important are the marketing situation and the skill of workers at accomplishing a high level of reproductive performance. Because a population of replacement gilts is managed under a common protocol, there is probably a confounding of age, body weight, back fat thickness and oestrous number at puberty. Very few commercial farms collect oestrous data on all their replacement gilts or measure back fat at time of mating. Most farms are organised to breed gilts during a specific age bracket, with the assumption being that if gilts are bred according to age then they will be at their second or third oestrous.

Babot *et al.* (2003) analysed data from a computerised record program in Spain (340 pig farms) to evaluate the effect of age at first mating on lifetime production of 37,698 sows. Lifetime production for number of litters and number of piglets weaned was higher (P<0.001) for gilts mated between 221 and 240 days of age than for gilts mated at less than 210 days of age (Table 4).

Table 4. Effect of age at first mating on the reproductive lifetime performance of sows.

	Class of sows according to their age at the first mating in days						
Item	<210	210-220	221-230	231-240	241-250	251-270	>270
NS [1]	3,733	5,845	5,269	6,836	5,133	6,604	4,278
NH [2]	238	319	310	337	337	324	262
AFM [3]	204.4 [a]	218.2 [b]	227.1 [c]	237.2 [d]	247.1 [e]	262.0 [f]	287.3 [g]
AFF [4]	333.3 [a]	436.1 [b]	351.9 [c]	362.2 [d]	370.6 e	383.1 [f]	405.0 [g]
CA [5]	1038.5 [a]	1083.5 [b]	1108.0 [bc]	1111.4 [bc]	1106.5 [bc]	1127.0 [c]	1132.0 [c]
PC [6]	5.2 [a]	5.4 [ab]	5.6 [b]	5.5 [b]	5.4 [ab]	5.4 [ab]	5.3 [ab]
NBA-L1 [7]	9.2 [h]	9.3 [ij]	9.3 [hi]	9.3 [i]	9.4 [j]	9.4 [j]	9.4 [j]
NBA-L2 [8]	9.1	9.3	9.2	9.2	9.2	9.3	9.2
LTNBA [9]	52.5 [a]	55.4 [abc]	56.5 [bc]	56.3 [bc]	55.0 [ac]	55.6 [bc]	54.4 [c]
LTPW [10]	46.3 [a]	48.7 [ab]	50.3 [b]	49.6 [b]	48.7 [ab]	49.2 [b]	48.1 [ab]

[1] NS is number of sows
[2] NH is number of herds
[3] AFM is average age at first mating, days
[4] AFF is average at first farrowing, days
[5] CA is average age at time of culling, days
[6] PC is average number of parities at time of culling
[a-g] Values with different superscripts within row differ, P<0.001
[h-j] Values with different superscripts within row differ, P<0.05
[7] NBA-L1 is the average number of piglets born alive at first parity
[7] NBA-L1 is the average number of piglets born alive at first parity
[8] NBA-L2 is the average number of piglets born alive at second parity
[9] LTNBA is the average lifetime total sum of all piglets born alive
[10] LTPW is the average lifetime total sum of all piglets weaned

Le Cozler *et al.* (1998) partitioned 976 French commercial herds into three categories according to their average value and standard deviation for age at first farrowing and interval between date of entry and conception. The groups were then classified as farms that used management practices to farrow gilts at either an 'early' (337 days; 8,285 sows), 'usual' (356 days; 19,414 sows) or 'late' (371 days; 7,932 sows) chronological age. Assuming a gestation length of 114 days, the age at mating was 223 (early), 242 (usual) and 257 (late) days. Only Large White x Landrace animals reared indoors were used in the analysis. The cumulative lifetime number of piglets born alive per culled sow was lower ($P<0.001$) in the 'early' farrowing system (51.96 piglets) than the 'usual' farrowing system (53.77 piglets) or the 'late' farrowing system (53.55 piglets). There was no statistically significant difference between the usual and late farrowing systems. When Le Cozler *et al.* (1998) analysed the entire data set on an animal basis (35,631 gilts), the total number of piglets weaned per sow before culling was not different for sows farrowing across a range of less than 330 to 384 days of age. These data provide evidence that the optimal age at first farrowing depends on herd management.

Economical aspects

Without question, the sustainable future of sow farms requires a financial investment in replacement gilts. As described in the symposium papers by Smits *et al.* (2005) and van Wettere *et al.* (2005), many factors are involved in correctly managing replacement gilts to optimise their reproductive performance. Management of gilts and primiparous and multiparous sows is critically important because the current removal rate of sows from a breeding herd is excessively high. Table 5 indicates the replacement rate, culling rate, death rate and average parity of culled sows for farms in Canada and the United States where the computer-based PigCHAMP data share recording program is used (PigCHAMP 2000, 2001, 2002, 2003, 2004). The values presented are the means for the specific trait. The formula used by PigCHAMP to calculate replacement rate is: (females entered/average female inventory) x (365/period length) x 100. The average replacement rate ranged from 49.6-79.5%. The formula used by PigCHAMP to calculate culling rate is: (sows and gilts culled/average female inventory) x (365/period length) x 100. Death rate for sows and gilts includes destroyed animals. The average culling rate ranged from 37-44.6%. Stein *et al.* (1990) reported similar values for annual removal rates in commercial swine breeding herds. The average parity (number of litters farrowed) at time of culling ranged from 3.1-5.2. Due to high replacement and culling rates, a substantial amount of money is spent on replacement gilts. Although it makes common sense to keep a breeding female producing at a high level in the herd as long as possible, the financial impact of culling sows too early is not often determined at the farm level.

Table 5. Replacement rate (RR), culling rate (CR), death rate (DR) and average parity of culled sows (Parity) for farms using PigCHAMP in the United States and Canada during years 2000 through 2004.

	United States data					Canadian data				
Item	2000	2001	2002	2003	2004	2000	2001	2002	2003	2004
No. farms	612	786	nr [5]	199	225	282	455	nr	61	39
RR, % [1]	56.9	57.0	62.9	79.5	51.8	49.6	52.0	58.7	64.6	46.1
CR, % [2]	44.6	39.0	41.6	41.2	43.8	41.1	37.0	39.5	38.1	40.0
DR, % [3]	6.9	6.8	7.8	7.8	7.9	4.7	5.0	6.5	7.3	7.2
Parity [4]	3.1	3.8	3.8	4.0	4.3	4.2	4.6	4.7	5.2	5.0

[1] Replacement rate = (females entered/average female inventory) x (365/period length) x 100
[2] Culling rate = (sows and gilts culled/average female inventory) x (365/period length) x 100
[3] Death rate = (sow and gilt deaths/average female inventory) x (365/period length) x 100
[4] Average parity of culled sows = sum of parity of culled sows/sows and gilts culled
[5] nr indicates data were not reported.

PorkCHOP model

Dijkhuizen *et al.* (1986) developed a computer model to determine the optimum lifespan for individual sows in the herd. The economic replacement model named PorkCHOP was designed to help producers optimise decisions to replace sows of poor productivity (old age and small litters) or with reproductive problems (anoestrus and failure to conceive), sickness, mothering characteristics and lameness. The underlying economic criterion for culling decisions is that a sow of a particular age should be kept in the herd as long as her expected profit for the next parity (marginal profit) was higher than the per parity lifetime average return from a replacement gilt (average profit). The optimum lifespan for each individual sow is based on the average performance in the various parities of sows present in the herd, assuming this was the best estimate for the expected future performance of gilts. The economic optimum lifespan is the last parity with a positive difference between expected and marginal profit of the present sow and average profit of the replacement gilt, excluding the risk of removal. The model calculates the total extra profit to be expected from trying to keep the sow in the herd until the optimum lifespan is reached. Dijkhuizen *et al.* (1989) evaluated whether made by farmers to cull sows correlated with the economically optimum decisions generated by the PorkCHOP model. Sow culling data were analysed from 1,617 culled sows on 12 Dutch farms.

Farmers generally made the correct decision to cull sows directly after weaning due to low productivity (old age, small litters). The economic loss of removing a sow too early because of lameness or leg weakness was 183 Dutch guilders (Dfl) [$141 AUD] per culled sow, removing a sow too early because of non-expression of oestrus was 185 Dfl [$142 AUD] per culled sow and removing a sow too late after not conceiving was 153 Dfl [$118 AUD].

Net present value model

Stalder *et al.* (2000, 2003) used a net present value (NPV) approach to determine the number of parities for which a sow must remain in the herd to recover her investment cost and produce profit. Capital budgeting for replacement gilts takes into consideration the number of parities the gilt will be in the herd and the initial cost of the gilt followed by periods of expenses and income. Tax implications are ignored. The analysis is based on discounted cash flows (DCF) and calculates the NPV and the internal rate of return (IRR).

Discounted cash flow is what someone is willing to invest today in order to receive the anticipated cash flow in future years. Discounted cash flow means converting future earnings into today's money. The reason for putting future dollars in present-day value is because one dollar today is worth more than one dollar tomorrow. This concept is known as discounting. The future cash flows are discounted to express their present values so that the purchase value of the replacement gilt is properly determined. The DCF for an investment is calculated by estimating the cash paid out and the cash received back. The number of times (parities) receipts are expected must also be estimated. Each cash transaction must then be discounted by the opportunity cost of capital over the time between now and when cash will be paid or received. Net present value is the amount of money an investment is worth in today's dollars. Net present value takes into account the amount of the investment, the length of the investment, how long it takes the investment to return a profit and the cost of money (interest and risk).

Pork producers can use NPV analysis when making purchasing decisions or when evaluating breeding animal replacements from sources whose initial cost varies. Additionally, NPV analysis allows producers to compare gilts with different productivity levels, length of service, feed conversions and purchase prices to determine which is the most profitable for their operation.

Net present values greater than zero indicate that an investment will be profitable when all of the above factors are considered. A NPV of less than zero indicates that an investment will not be profitable when all factors are considered. When several alternative investments are considered, the alternative with the highest NPV (greater than zero) is the most profitable.

The internal rate of return (IRR) is the discount rate (or interest rate or rate of inflation) that will cause an investment to have a NPV equal to zero (break-even). The greater the IRR above zero the more profitable the investment. The IRR does not account for interest, but it does account for time, production, expenses, and investment costs. It is also the return received for investing in this gilt. The input information entered by the user into the template for segregated early weaning sow longevity is indicated in Table 6.

Table 6. Items entered into the segregated-early weaning and sow longevity spreadsheet program.

Price of replacement gilt	Adjustment of litter size by parity
Annual discount rate	Average kg of feed per day during gestation
Gilts purchased that do not enter breeding herd	Average kg of feed per day during lactation
Other gilt development cost	Price per tonne of gestation feed
Average number of pigs born alive per litter	Price per tonne of lactation feed
Birth to weaning death loss	Total kg of feed provided gilts during development
Death loss of sows	Total cost of facilities
Weight of gilts at culling	Equity in facilities
Market price of cull gilts	Interest rate on facilities
Market price of segregated-early-weaned pigs	Veterinary and medicine cost
Number of sows in herd	Breeding cost
Litter per sow per year	Waste handling cost
Average parity of farrowed sows	Insurance on facilities
Weight of sows at culling	Labor
Market price of cull sows	Utility cost
Marketing and handling cost	Maintenance and repairs of facilities

The model is designed for a breed-to-wean operation and has lactation length fixed at 19 days. Based on the assumptions entered by the user, the computer generates NPV sensitivity tables. The tables generated include: (1) NPV per gilt by parity (1 to 6) when the price of replacement gilt varies, (2) NPV per gilt by parity when the number of pigs born alive varies, (3) NPV per gilt by parity when weaner pig price varies, and (4) NPV per gilt by parity when discount rate varies. In addition, the computer generates a table that indicates the IRR by parity when price of replacement gilt varies.

The results generated by this template are not surprising. The longer productive gilts remain in the herd, the more profitable the investment. The value of the template is that it places a monetary value on the amount of loss or profit associated with each parity of the breeding female. In addition, the template provides awareness about the number of parities for which a gilt needs to remain in the herd before a break even occurs for the investment. As a brief example of the results that can be generated by the model, the input values indicated in Table 7 were entered into the template. Gilt A did not reach a positive NPV until parity four while Gilt B reached a positive NPV at parity three (Figure 1). If both gilts remain in the herd for six parities, the NPV of Gilt A ($130) is substantially less than for Gilt B ($419). The IRR for this scenario is indicated in Figure 2. Gilt B had a small positive IRR (1.7%) at parity two, while Gilt A did not have a positive IRR until parity four (5.1%).

Table 7. Production values, market value and investment cost used in the net present value analysis example of sow longevity.

Item	Gilt A	Gilt B
Purchase price, USD	$300	$300
Piglets born alive per litter	9.7	11.2
Litter per sow per year	2.1	2.4
Preweaning death loss	15%	10%
Average parity of herd	2.3	3.0
Market price per weaned pig, USD	$30	$30
All other assumptions are the same for Gilt A and Gilt B		

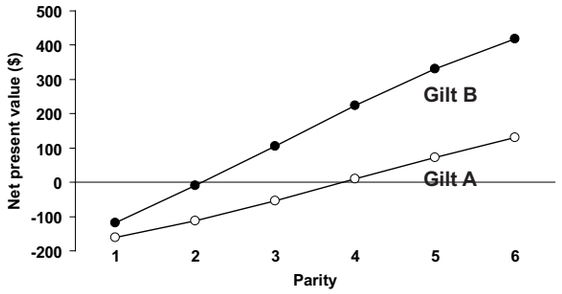


Figure 1. *Net present value of replacement gilt with different assumptions. Assumptions for Gilt A are $300 purchase price, 9.7 piglets born live per litter, 2.1 litters per sow per year, average parity of sow herd is 2.3, $30 market price of segregated-early weaned piglets, and 15% preweaning death loss. Assumptions for Gilt B are $300 purchase price, 11.2 piglets born live per litter, 2.4 litters per sow per year, average parity of sow herd is 3.0, $30 market price of segregated-early weaned piglets, and 10% preweaning death loss.*

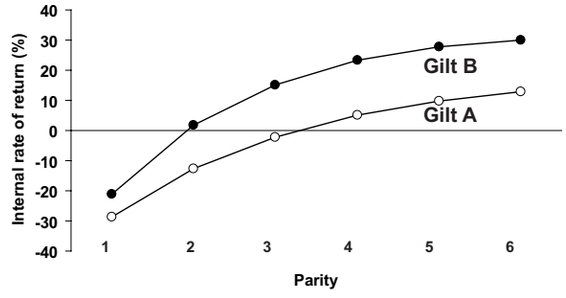


Figure 2. *Internal rate of return for replacement gilt with different assumptions. Assumptions for Gilt A are $300 purchase price, 9.7 piglets born live per litter, 2.1 litters per sow per year, average parity of sow herd is 2.3, $30 market price of segregated-early weaned piglets, and 15% preweaning death loss. Assumptions for Gilt B are $300 purchase price, 11.2 piglets born live per litter, 2.4 litters per sow per year, average parity of sow herd is 3.0, $30 market price of segregated-early weaned piglets, and 10% preweaning death loss.*

The worksheets for gilt replacement can assist pork producers determine whether purchasing replacement gilts at a particular price is a profitable decision. Individual producers can input their current financial and production data to customise the spreadsheet to their particular operation. In this manner a producer can make a more informed decision regarding the purchase of replacement females for their breeding herd. Additionally, a producer can use the spreadsheets to determine if breeding females have been retained in the breeding herd long enough to allow the pork enterprise to recover the gilts' initial investment cost. If not, a producer must concentrate on the management of breeding herd females to improve their productive herd life.

Survival analysis

Brandt *et al.* (1999) evaluated the productive life of a sow by using the Cox-Proportional Hazard Model of survival analysis program ('The Survival Kit'). The authors investigated the influence of the following traits on the productive life of a sow: multiplier farm, litter performance, stage of sow within parity, insemination success, medium price level of weaner pigs, live-weight daily gain, leg quality, shape of ham, and teat score. All traits showed a significant influence on the productive life of a sow, except for the ham and teat score. The traits with the greatest effect were litter performance and insemination success. An increased risk of culling due to a large body size occurred between the third and fifth parity. The leg quality score assigned to the gilts at the time of selection had a significant influence on how long the sows remained in the herd. Gilts that were assessed as having slight or temporary defects of legs had a higher risk of being culled between the start of their productive life and fourth weaning than gilts assigned better leg scores. Factors of least importance were the traits used to select gilts - daily gain, number of teats, and subjective scoring of frame and ham. As could be expected, the pork enterprises culled more sows when the price of replacement gilts was lower and the price of slaughter sows was higher.

Genetics

Rodriguez-Zas *et al.* (2003) examined the records from 148,568 sows in 32 commercial herds from Central Illinois (USA). Longevity and performance data were used simultaneously to compare eight genetic lines. Economic indicators that reflected the economic impact of longevity were expressed in terms of NPV dollars. When considering a $50 net income per litter and discount rate of zero, the difference in NPV between the best and worst longevity line (parity at culling) was $52.39. The difference in NPV between the best two longevity lines was $13.94. The relative value of the line with high longevity decreased with increasing interest rates, because the value of income decreased with increasing time. When considering a $50 net income per litter and a discount rate of 10%, the difference between the best and worst longevity line was $16.37 and the difference between the two best lines was $0.20. This study suggested that the magnitude of the economic improvement attained through the use of sow genetic lines with longer longevity depended on the economic context under which the evaluation was made.

Discussion

High replacement rates, high culling rates, high mortality rates and a decrease in sow longevity are ongoing problems for the pork industry. Although computerised record programs allow pork producers to enter data relating to reasons why females are removed from the inventory of breeding females (culled, death, destroyed, transferred out), the pork industry does not appear to be making progress in lowering culling rate, death rate and excessive replacement rates. At the moment, herd records are only being used to identify the reasons for culling and death of sows, rather than stimulating pork producers to solve herd problems aggressively. What is the true economic cost of an excessive culling rate, death rate and replacement rate? Current recording programs cannot answer this economic question because they only document biological performance. What is the difference in profitability when reproductive performance is evaluated according to lifetime performance versus sow longevity? There is a difference between lifetime performance and sow longevity. Lifetime performance can be defined as any length of time that a breeding-female exists in a herd with a low or high level of reproductive efficiency. Sow longevity can be defined as any breeding female that exists in a herd for long periods of life with either a low or high level of reproductive efficiency. What is the definition for a long period of life of a sow? Most likely, sows that produce 6-10 litters are considered to have had a long and productive life span. Currently, there is no recording program available by which profitability of breeding animals is evaluated according to lifetime performance rather versus sow longevity. Determining the difference in profitability between a sow that produces three litters compared to a sow that produces seven litters is clearly important in how we manage the breeding herd.

The normal benchmarks for sow longevity include average parity of the herd and removal rates. However, each herd needs to place more emphasis on (1) identifying the exact reasons for high culling rates, high mortality rates and high replacement rates and (2) taking appropriate and dedicated action to solve the problem(s). Pork producers

need to evaluate their reason(s) for removing breeding animals from the herd according to parity and the specific stage of the reproductive cycle when removed from the inventory of females. Although emphasis is being placed on best management practices for developing and managing replacement gilts, this single phase of production cycle will not solve the problem with sow longevity or removal rates. Pork producers still need to make sure excellent housing and management procedures are used for all parities and phases of production, especially parities one and two.

Complex computer models are not used by the pork industry to evaluate the profitability of keeping sows in the breeding herd. Although some computer models are fairly robust and allow the user to change many values, the computer models do not effectively simulate the true situation of all pork production enterprises. Within and among swine breeding herds, numerous factors and their interactions impact on costs and returns. A simplified profit model that combines pig production and the number of days the female is in the breeding herd might be more valuable to a pork enterprise. This type of model would imply that various biological effects of an extended weaning-to-breeding interval, number of days from breeding to returning to oestrus, all other reasons for nonproductive days, and changes in litter size by parity are taken into account. The model would essentially have a fixed cost and variable cost each day the female resides in the breeding herd. The date when the gilt is accounted for as a breeding-female would represent the beginning date. The end point would be the date the sow physically leaves the farm. The gilt development and breeding phase would be the interval from the date of entry until conception for production of the first litter. All subsequent reproductive cycles would be the interval from farrowing-to-farrowing. The farrowing-to-farrowing interval would account for length of lactation, non-productive sow days until successful conception for the next litter and length of gestation. The number of days from the last weaning date to when the sow actually leaves the farm are counted as nonproductive days.

The 'baseline' assumptions I used in the model are indicated in Table 8. Several other variable factors could be added to the model to more closely simulate a particular operation. Table 9 indicates additional variables of the model that can be changed to estimate profitability of a sow at a various number of parities based on a daily fixed cost and variable cost. The accumulated profit at parity two through six for the assumptions listed in Tables 8 and 9 is indicated as Scenario 1 in Figure 3. Accumulated profit increased from parity two through six. The difference in accumulated profit for a sow that produced three litters compared to a sow that produced six litters was $360. What effect does increasing the age at first successful mating have on accumulated profit? There will be an increase in housing and feed cost due to increasing the number of days from entry until mating. Scenario 2 in Figure 3 indicates the effect on profit when the age at first successful mating is 250 days and the fixed cost per day for housing is $0.60. Accumulated profit was reduced because there was not an increase in litter size at any parity. If mating of gilts is delayed until 250 days of age and number of pigs weaned is increased (parity one, 10 pigs; parity two, 10 pigs; parity three, 11 pigs; parity four, 11pigs; parity five, 11 pigs; parity six, 10 pigs), there would only be an increase in profit at parities five and six (Scenario 3, Figure 3) compared to Scenario 1. For Scenario 3, the difference in accumulated profit for a sow that produced three litters compared to a sow that produced six litters was $441.

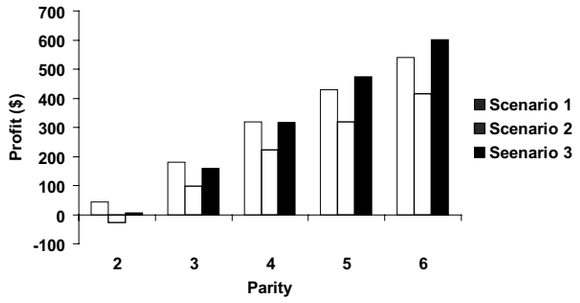


Figure 3. *Accumulated profit at parity 2-6 for three scenarios when using a simplified profit model that combines pig production and number of days the female is in the breeding herd. Scenario 1 is a base-line model. Scenario 2 increased the age at first successful mating without changing reproductive performance. Scenario 3 increased the age at first successful mating and increased reproductive performance.*

There are a number of questions that arise from such an analysis. Will increasing the age at first successful conception of gilts provide a method whereby the sows will produce litters five and six? Will increasing the age at first successful conception produce more weaned pigs during the sow's lifetime? What is the life expectancy of the majority of the sows in the herd when age at first successful conception is increased? Are there cheap alternatives for housing gilts during the period of time from entry to first successful conception that will enhance lifetime reproductive performance? What is the economical impact of checking gilts for pubertal oestrus and only using cyclic gilts at time of entry into the breeding herd? Is lifetime reproductive performance enhanced for gilts that cycle early and mated at the second, third or fourth oestrus? What percentage of early puberty gilts will keep cycling to be mated at an older age?

Table 8. Base line assumptions for the simplified computer model to estimate sow profitability over time.

Item	Value
Age of gilt at entry, days	180
Age at first successful mating, days	220
Average value per pig weaned, USD	$30.90
Semen cost per dose, USD [a]	$5.50
Veterinary and medical costs, USD	
Gilt	$1.56
Parity 1	$2.25
Parity 2+	$2.00
Labour cost per day per sow, USD	$0.18
The building cost per day, USD	$0.50
Value of cull gilt, USD [b]	$116
Purchase price, USD	$250
Cull weight, kg	193
Cull price/per kg, USD	$.6536
Sow death loss	8%

[a] Two doses of semen per service

[b] The following formula was used to calculate the cull value of a gilt: cull weight x cull price x (1 – sow death loss). The depreciated value of the gilt varies according to the number of days in the herd. The gilt was depreciated with a straight-line depreciation formula. The straight-line depreciation method assumes that depreciation is uniform throughout the lifespan of the gilt until removed from the herd.

Table 9. Additional variables used by the simplified computer model to estimate profitability of sows at each parity based on a daily fixed cost and variable cost.

Production phase	Length of lactation, d	Nonproductive days	Length of gestation, d	Total	Accumulated days in herd	Number of pigs weaned
Gilt to Farrow 1		40	115	155	155	
Farrow 1 to 2	21	16	115	152	307	9
Farrow 2 to 3	21	8	115	144	451	10
Farrow 3 to 4	21	7	115	143	594	10
Farrow 4 to 5	21	5	115	141	735	10
Farrow 5 to 6	21	5	115	141	876	9
Farrow 6 to 7	21	30		51	927	9
Total	126	71	690	927		57
Avg kg feed/d						
Gilts		2.49	2.27			
Sows	7.03	2.27	2.27			
Total feed, kg	885.8	260.8	1564.9	2711.6		
Feed cost/kg	.1598	.1268	.1268			
Feed cost, $	141	33	198	373		

There is no doubt that records play a key role in making management decisions, however recording programs do not solve problems. People solve problems. Collecting more records is unlikely to solve the problem of high replacement rates in breeding herds. If death rate is a serious problem on the farm, what is being done to correct the problem? If a many sows are culled after the first litter for not cycling or conceiving at first and second service, what is being done to correct the problem? From a breeding shed manager's point of view, it is easier to cull an animal than to focus on possible ways to prevent culling gilts or parity one and parity two sows.

Conclusion

Currently, the pork industry does not have a uniform method to predict the optimum number of litters a sow should produce for a specific situation. Culling and replacement decisions are usually based on production and economic situations. When making changes to improve reproductive performance and sow longevity there is a financial risk involved. Because of the many factors influencing reproductive performance and sow longevity within and among farms, implementing a way to solve a problem on one farm might not be appropriate for another farm. Pork producers need to concentrate on all aspects influencing reproductive performance in order to optimise lifetime production of the sow. The take home message for a commercial sow farm is to keep the sows producing at a high level for as long as possible.

Management and nutritional factors affecting puberty attainment and first litter size in replacement gilts

W.H.E.J. van Wettere, M. Mitchell*, D.K. Revell** and P.E. Hughes

The University of Adelaide, Roseworthy, SA 5371. * The University of Adelaide, Woodville, SA 5011. **CSIRO Livestock Industries, Wembley, WA 6913.

Abstract

Incidences of gilt reproductive failure remain unacceptably high. Recent evidence reviewed in this paper suggests this problem may be because modern gilt genotypes are very different from their counterparts of 20-30 years ago but are managed much the same. For example, today's gilts are leaner, later maturing and have higher ovulation rates, yet enter the breeding herd at a similar age to their predecessors. In addition, evidence is now surfacing that differences in pre-pubertal nutrition are associated with altered ovarian follicle growth. This affects both ovulation rate and early embryo survival rate (via effects on oocyte quality). It is concluded that improved reproductive performance of gilts will require modification of current management recommendations so that they better suit modern, leaner genotypes.

Introduction

Appropriate management of replacement gilts has been identified as a key determinant of sow lifetime productivity (Aumaitre *et al*., 2000). Combining appropriate nutritional management of the pre-pubertal gilt with effective stimulation of puberty and mating strategies facilitates gilt entry into the breeding herd, maximises subsequent reproductive performance and can potentially increase sow longevity (Evans and O'Doherty, 2001). However, despite selection for increased litter size and the adoption of improved reproductive technologies (eg artificial insemination), reproductive disorders and failure - including the failure to reach puberty; unpredictable timing of the first oestrus; low conception rates; high return rates; and low first litter sizes - remain a common problem associated with replacement gilts (Whittemore, 1996). In fact, about 20% of premature culling of sows from the breeding herd occurs at parity 0, with 65% of these culls attributed to reproductive disorders or failure (Lucia *et al.,* 2000). To address this problem it is necessary to consider whether current gilt management strategies developed for genotypes of 20 to 30 years ago are suitable for today's heavier yet leaner genotypes (Hughes *et al*., 1996; Edwards, 1998; Hughes and Varley, 2003). This paper will focus on some of the strategies used to control the onset of puberty in the gilt, as well as reviewing current knowledge concerning the effects of nutrition on puberty attainment and first litter size.

Puberty attainment

The onset of puberty in the gilt depends on the progressive maturation of the hypothalamus-pituitary-ovarian axis (Dyck, 1988; Pressing e*t al.,* 1992; Slevin and Wiseman, 2003). Growth and development of the reproductive organs begins during the early stages of embryonic development and is associated with sequential changes in gonadotrophin secretion and gradual development of the ovarian follicle population (Elsaesser, 1982; Christenson *et al.,* 1985; Dyck, 1988). Integration of the pituitary-ovarian axis, essential for the attainment of puberty, occurs during the first three to four months of post-natal life following the development of a sufficiently mature ovarian follicle population (Camous *et al.,* 1985; Pressing *et al.,* 1992). This is followed by a 'waiting' phase (Camous *et al.,* 1985), which lasts from about four to five months of age through to the onset of puberty and is characterised by an apparent decrease in hypothalamic sensitivity to the negative feedback effects of gonadal steroids, culminating in a cascade of endocrine events similar to the follicular phase of the oestrous cycle and the first ovulation (Hughes *et al.,* 1990; Slevin and Wiseman, 2003). Female pigs may attain puberty anywhere between 15 and 50 weeks of age and reducing this variation by controlling the onset of puberty is extremely beneficial to the productivity of a breeding herd (Hughes, 1982). In practice this is normally achieved by using boar contact, although exogenous hormone administration does offer an alternative approach.

The boar effect and puberty attainment

Exposing replacement gilts to a mature boar is the most effective method of stimulating precocious puberty attainment and is common practice throughout the pig industry. This topic has been extensively reviewed (see Hughes *et al*., 1990; Hughes, 2003) and we offer here only a brief summary of the key elements of the boar effect.

Although the precise mechanisms responsible for early puberty onset in response to boar stimulation are incompletely understood, it is generally accepted that the gilt responds to the synergistic actions of four main types of cues originating from boars (reviewed by Hughes *et al.,* 1990). In brief, current knowledge suggests that olfactory cues, namely priming pheromones (e.g. 3α-androstenol), present in saliva secreted by the boar's submaxillary salivary glands, act in concert with tactile and possibly auditory and visual stimuli, to alter the pattern of luteinising hormone (LH) secretion in the gilt (Hughes *et al.,* 1990; Pearce and Paterson 1992; Kingsbury and Rawlings, 1993). This actuates an increase in ovarian follicle growth, causing oestrogen concentrations to rise, triggering a cascade of endocrine events equivalent to the follicular phase of the oestrous cycle, and culminating in the onset of oestrus and the first ovulation (Esbenshade *et al.,* 1982; Paterson, 1982; Deligeorgis *et al.,* 1984).

The exhibition of a pubertal response depends on the gilt receiving sufficient boar stimulation and variations in gilt response are attributed primarily to differences in the stimulus value of the boar, the amount of physical gilt-boar interaction that occurs, as well as the frequency at which boar exposure is applied. Hughes *et al.* (1990) reviewed these areas extensively. In summary, it has been known for many years that effective application of the boar effect requires the boar used for stimulation to be at least 9-10 months of age (Kirkwood and Hughes, 1981) and that full physical contact is allowed between the boar and the gilts for at least 15-20 minutes daily from a gilt age of about 23 weeks (Hughes, 1982; Pearce and Hughes, 1987; Paterson *et al.,* 1989; Hughes, 1994).

Table 1. Proportion of gilts attaining puberty in response to daily contact, commencing at 160 days of age, with boars of a low or high sexual motivation* (adapted from Hughes, 1994).

	Treatment		
	No boar contact	Daily contact with a boar of low sexual motivation	Daily contact with a boar of high sexual motivation
Proportion of gilts pubertal by:			
Day 20	0.00	0.19	0.59
Day 40	0.08	0.62	0.81
Day 60	0.35	0.88	0.89
Mean days to puberty **	48	34	19

*based on 3 times 15 minutes tests described by Hemsworth et al. (1978)
**Refers to gilts reaching puberty by 220 days of age

Over the past decade, two studies have shed further light on the boar effect. First, it was demonstrated that individual mature boars (>10 months of age) differ in their ability to stimulate early puberty attainment (Hughes, 1994; Chamberlain and Hughes, 1996 – see Table 1). This effect had been reported in young boars (< 9 - 10 months of age) and was known to reflect their inability to produce and secrete sufficient quantities of primer pheromones (Kirkwood and Hughes, 1981; Hughes *et al.,* 1990). It is likely that differences in the stimulus value of mature boars reflect a similar effect. Second, while it had been evident for several years that maximum gilt response to boar exposure depended on regular reinforcement of the stimulus (Paterson *et al.,* 1989; Philip and Hughes, 1995), recent studies have demonstrated that gilt response is enhanced when boar contact occurs on two or even three occasions each day (Table 2).

Table 2. The effects of frequency of boar contact commencing at 160 days of age on the attainment of puberty in gilts.

		Frequency of daily boar contact			
Source	Proportion pubertal within:	0	1	2	3
Hughes (1994) – study 1	20 days	0.00	0.21	0.86	-
	40 days	0.00	0.50	1.00	-
	60 days	0.00	0.56	1.00	-
	Mean days to puberty	-	26.9	12.9	-
Hughes (1994) – study 2	20 days	0.00	0.31	0.25	0.69
	40 days	0.00	0.62	0.62	0.75
	60 days	0.00	0.75	0.94	0.87
	Mean days to puberty	-	27.8	35.4	17.8

Attainment of early puberty in response to boar exposure relies on the gilt perceiving stimuli from the boar and initiating an appropriate endocrine response (Kingsbury and Rawling, 1993). More specifically, precocious attainment of puberty depends on increased growth of ovarian follicles in response to boar stimuli, and the resulting elevation in plasma oestrogen concentration stimulating a positive feedback effect at the hypothalamus (Paterson, 1982). It is generally accepted that chronological age, as opposed to gilt live weight and body composition, is a more favourable

predictor of gilt response to puberty stimulation (Hughes and Varley, 2003). It has been concluded from early studies that the components of the reproductive mechanism involved in puberty onset were sufficiently integrated by 23 weeks of age to allow a rapid and synchronous pubertal response (Hughes and Cole, 1976; Eastham *et al.,* 1986; Paterson *et al*., 1989). However, although modern gilts are now heavier at 23 weeks of age, they are also leaner and at a lower proportion of their potential mature weight, meaning they are likely to be physiologically less mature than their predecessors of 20 to 30 years ago (for reviews see Whittemore, 1996; Edwards, 1998; Evans and O'Doherty, 2001; Hughes and Varley, 2003).

Improving, or at least maintaining, the efficacy of boar stimulation requires the modification of current recommendations to suit today's later maturing genotypes. Support for this is provided by the recent study of van Wettere *et al.* (2004, unpublished data), where maximal pubertal response to twenty minutes of daily boar contact did not occur until gilts were 26 weeks of age or older, which is about three weeks later than previously reported in the literature. In this study, commencing boar exposure at 26 or 29 weeks of age, as opposed to 23 weeks, resulted in significantly fewer mean days to puberty and a significantly higher proportion of gilts attaining puberty within 10 and 20 days of the start of boar stimulation (Table 3). A faster response to boar stimulation is indicative of continued physiological development of the hypothalamic-pituitary-ovarian axis (Kirkwood and Hughes, 1979; Deligeorgis *et al.,* 1984), which suggests this hormonal axis is not fully developed at 23 weeks of age in current genotypes.

Table 3. Attainment of puberty in gilts in response to 20 minutes of full, daily contact with a vasectomized boar in a detection-mating area when boar exposure commenced at 23, 26 or 29 weeks of age (van Wettere *et al., 2004,* unpublished data).

	Age at start of boar exposure		
	23 weeks	26 weeks	29 weeks
Proportion of gilts pubertal by:			
Day 10	0.24[a]	0.67[b]	0.70[b]
Day 20	0.70[a]	0.81[b]	0.93[c]
Day 35	0.82	0.98	1.00
Mean days to puberty*[1]	18.9 ±1.5 [b]	10.6 ±1.2 [a]	8.3 ± 0.9 [a]

[a, b, c] means, in the same row, with different superscripts are significantly different ($P < 0.05$)
*Gilts that did not attain puberty by day 35 of boar exposure were ascribed a nominal days-to-puberty of 40 days
[1] Interval from initial exposure to a vasectomized boar until exhibition of a standing reflex

Exogenous hormone treatments and puberty attainment

There is a wealth of data demonstrating that administration of exogenous gonadotrophins effectively induces ovulation in prepubertal gilts in the age range 23-30 weeks (see Table 4). However, while gilts will ovulate, this is not always accompanied by behavioural oestrus and about 30% of gilts fail to exhibit normal subsequent oestrous cycles. Hence, these treatments have usually been accompanied by mating/insemination at the induced oestrus, frequently based on a fixed time period after treatment. Interestingly, while gilt response has been known to depend on age and live weight (Britt *et al.,* 1989), a recent study indicates that the provision of boar contact considerably improves the efficacy of exogenous hormone treatment (Smits *et al.,* 2001; Table 5).

Table 4. Typical gilt responses to PG600 treatment.

Source	% Oestrus	% Ovulating	% Cycling
Paterson (1982)	88	100	87
Paterson (1982)	-	97	60
Tilton *et al.* (1995)	70	99	-
Kirkwood (1999)	78	-	67
Knox and Tudor (1999)	69	65	-
Knox *et al.* (2000)	76	86	-

Table 5. The effects of exogenous hormone treatment and boar exposure on the timing of puberty attainment in very young gilts (Smits *et al.,* 2001).

Age at hormone treatment*	18 weeks		21 weeks		24 weeks	
Boar contact	No	Yes	No	Yes	No	Yes
No. gilts mated	88	96	106	107	95	98
Pregnancy (%)	12	26	18	35	35	55

*1,000 IU Folligon at 0 hrs followed by 500 IU Chorulon at +72 hrs and single insemination at +102-104 hrs

Nutrition and puberty attainment

Twenty years ago Kirkwood and Aherne (1985) proposed that minimum threshold levels for live weight and body tissue reserves act in a permissive manner to allow, rather than actively trigger, the onset of puberty. More recent studies suggest that within genotypes, the rate of sexual maturation, and hence puberty attainment, is sensitive to altered nutritional status during the prepubertal period. Differences in live weight and body composition, have been associated with an altered rate of sexual development of gilts. For example, higher rates of fat and protein deposition have been associated with an earlier onset of puberty (Gaughan *et al.,* 1997). Feed restriction during the pre-pubertal period can delay, but not prevent, the attainment of puberty (Beltranena *et al.,* 1991). Similarly, Prunier *et al.* (1993) observed a delayed pubertal response and altered levels of gonadotrophins and metabolic hormones in gilts that were restrictively fed from 80 to 220 days of age.

In our own studies we have been unable to find a significant relationship between gilt response to boar stimulation, across a range of gilt ages, and live weight or P2 bac kfat depth. This supports the view that in the absence of extreme differences in growth characteristics, gilt age is a more reliable predictor of the timing of the pubertal response (van Wettere *et al.,* 2004, unpublished data). Nevertheless, at the level of follicular growth, we have noted that the ovaries of prepubertal gilts fed to reach a target live weight of 100 kg (Heavy) at 23 weeks of age contained a higher proportion of follicles measuring 3 – 6 mm in diameter and a lower proportion of follicles with a diameter of 1 - 2.9 mm compared to gilts fed to reach a target live weight of 70 kg (Light) (Figure 1).

In this same study the concentration of oestradiol in the follicular fluid of Light gilts was lower than in Heavy gilts, suggesting that intrafollicular steroidogenesis was altered in Light gilts, possibly reflecting reduced availability of oestradiol precursors and/or altered expression or activity of key steroidogenic enzymes, (i.e. P450-17α-hydroxylase and/or P450-aromatase). Steroids have an important role in the regulation of ovarian activity. In prepubertal gilts, for example, an association between the steroid content of follicular fluid and the ability of oocytes to reach metaphase 2 *in vitro* has been established (Grupen *et al*., 2003) and higher follicular fluid concentrations of oestradiol have been associated with improved oocyte maturation *in vitro* (Ferguson *et al.,* 2003). Studies involving cattle (Armstrong *et al.,* 2002) and pigs (reviewed by Prunier and Quesnel, 2000) suggest that metabolic effects on follicular steroidogenesis are mediated, at least in part, by changes in the bioavailability of intra-follicular insulin-like growth factors and their binding proteins, which are also involved in the regulation of follicle responsiveness to gonadotrophins (Webb *et al.,* 2004). Consequently, reduced follicle growth and altered intra-follicular steroidogenic activity (van Wettere *et al.,* 2004, unpublished data) could impair the ability of Light gilts to initiate an ovarian response to the rise in LH secretion associated with boar stimulation (Kingsbury and Rawlings, 1993), and may partially explain the delayed attainment in puberty observed in the earlier study of Beltranena *et al.* (1991).

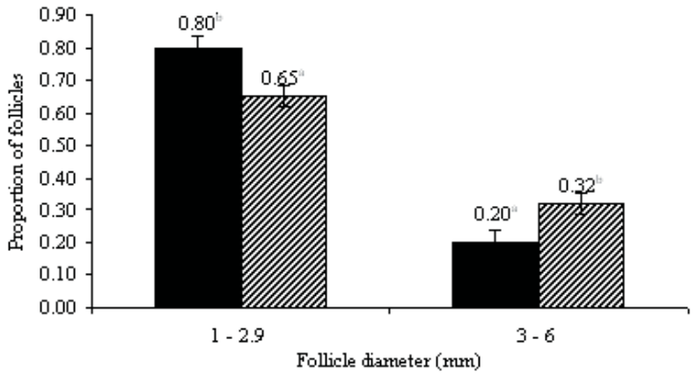


Figure 1. *Average proportion of surface antral follicles with a diameter of 1 - 2.9 mm or 3 – 6 mm to the total number of surface antral follicles greater than 1 mm in diameter on the ovaries of 23 week-old, non-cycling gilts fed to attain a target live weight of 70 kg (Light ■) or 100 kg (Heavy □) at 23 weeks of age.* [ab]*Different superscripts denote significant differences between Light and Heavy gilts.*

The association between growth performance and reproductive maturity is likely to be due to the regulation of reproduction by nutrient intake and/or metabolic status (Rozeboom *et al.,* 1995; Patterson *et al.,* 2002). Booth *et al.* (1994) demonstrated that nutritionally-induced alterations in gilt metabolic status could affect reproductive function without changing weight or P2 back fat. Consequently, identifying the metabolic processes and signals that mediate the effects of feed intake and nutrient supply on reproductive processes will provide greater insight rather than attempting to establish relationships between growth performance and reproductive development.

First litter size

To maximise the size of the litter produced by a gilt it is necessary to ensure that ovulation rate is not limiting (possibly an ovulation rate of 14+). Embryonic and fetal losses must then be minimised during gestation.

Ovulation rate

Traditionally, a strategy of mating gilts for the first time at their second or third oestrus has been widely adopted by the pork industry (Martinat-Botte *et al.,* 1985; Whittemore, 1996). This conservative strategy has been adopted despite the equivocal nature of the literature on this subject. Reports on the benefits of delaying first mating are not only contradictory but also frequently fail to separate the confounding effects of sexual or physiological age (i.e. number of oestrous cycles experienced) from those of chronological age (i.e. days). Consequently, it remains unclear whether advancing sexual age or chronological age has the greater effect on first litter size (Brooks and Smith, 1980; Archibong *et al.,* 1992). Delayed first mating has been associated in some studies with higher ovulation rates (Archibong *et al.,* 1987), improved oocyte quality (Koenig and Stormshak, 1993; Herrick *et al.,* 2003), a decrease in the number of abnormal embryos (Menino *et al.,* 1989; Archibong *et al.,* 1992), and a tendency towards larger first litter sizes (Young *et al.,* 1990a). In contrast, other studies indicate that first litter size and sow productivity over multiple parities are unaffected by oestrus number or age at first mating (Brooks and Smith, 1980; Young *et al.,* 1990a; Young *et al.,* 1990b). Indeed, Aumaitre *et al.* (2000) reported a decrease in annual breeding herd productivity when gilts were mated at their second or third oestrus. In a recent study by us (van Wettere *et al.,* 2004, unpublished data) in which boar exposure commenced at either 23, 26, or 29 weeks of age, and gilts were mated at either their first (pubertal) or second oestrus, neither mating age nor mating oestrus had a significant effect on ovulation rate, embryo number or embryo survival. Furthermore, the results of our study suggest that modern gilts have higher ovulation rates than their counterparts of 20-30 years ago, shedding approximately three more ova at the pubertal oestrus (ovulation rates of older genotypes reported by Paterson and Lindsay, 1980; Brooks and Smith, 1980; Archibong *et al.,* 1992).

The endocrine, intra-follicular and cellular processes involved in the growth of ovarian follicles through to the ovulatory stage have been extensively reviewed (Cardenas and Pope, 2002). In brief, before the onset of puberty and during the luteal phase of the oestrous cycle a proliferating pool of approximately 50 – 100 'recruited' follicles measuring between 1 and 6 mm in diameter are present on the surface of the ovary. Throughout the 5-7 day follicular phase before ovulation, these 'recruited' follicles either become atretic or continue to develop (are 'selected') to form a pool of between 12 and 20 ovulatory follicles. The consequence of this ongoing selection process is that a morphologically and biochemically heterogeneous pre-ovulatory follicle pool is created.

Nutritional effects on follicle growth and ovulation rate appear to be mediated by altered secretion of metabolic hormones, such as insulin and insulin-like growth factor –1 (IGF-1), changes in LH pulsing, and differential expression of growth factors (Cosgrove and Foxcroft, 1996; Cardenas and Pope, 2002). Studies involving pre-pubertal gilts (van Wettere *et al.,* 2004 unpublished data – Figure 1), cycling gilts (Quesnel *et al.,* 2000a) and lactating sows (Quesnel *et al.,* 1998) indicate that altered feed intake affects the growth of 1 – 6 mm follicles, influencing the dynamics of the proliferating pool, and potentially affecting ovulation rate (Almeida *et al.,* 2000). Feed restriction reduces the number of follicles greater than 4-5 mm present on day 19 of the oestrous cycle (Quesnel *et al.,* 2000b; Ferguson *et al.,* 2003) indicating that nutritional effects on ovulation rate reflect alterations in the follicle's final stages of growth. However, studies involving post-pubertal gilts demonstrate that although ovulation rates decrease in response to feed restriction during both the luteal and follicular phases, they are unaffected by reduced feed intake during the luteal phase alone (Prunier and Quesnel, 2000). Consequently, nutritional status during follicle selection appears to be a critical determinant of ovulation rate (Prunier and Quesnel, 2000). Therefore, if nutrient supply is high during the 5-7 day period leading up to ovulation, ovulation rate may not be a limiting factor to first litter size in modern gilts, even if they are mated at the pubertal oestrus.

Embryo losses during gestation

About 30% of viable embryos are lost during the first 30-35 days of gestation and it is generally agreed that the majority of embryos die between days 5 and 18 of pregnancy (Pope *et al.,* 1990; Xie *et al.,* 1990; Geisert and Schmitt, 2002). A number of factors are believed to affect embryo mortality rates before and during implantation (days 5–18 of pregnancy) and these have been well reviewed by Ashworth and Pickard (1998). Essentially, most embryos are lost at this stage because they were either 1). less developed than their littermates before ovulation 2). fertilised later than their littermates or 3). exposed to a sub-optimal uterine environment during their first two weeks of life. Here, we present the most recent information relating to the role of nutrition before and after mating in altering the quality of the oocyte released at ovulation, heterogeneity within the pre-ovulatory follicle pool and the synchrony of development between litter-mate embryos and between the embryo and the uterus. Factors involved in foetal loss in the gilt (between days 30-35 and term) have been recently reviewed elsewhere (Vallet *et al.,* 2002; Ford, 2003).

Heterogeneity of the pre-ovulatory follicle pool and synchrony of embryo development

While growth of the conceptus is programmed through innate developmental cues (Geisert and Yelich, 1997), growth factors and nutrients provided by the uterus are also vital for embryonic development (Vallett *et al.,* 2002). In particular, the timing of changes in uterine secretions is critical for conceptus survival (Soede *et al.,* 1999), as asynchrony between the conceptus and the uterine environment is detrimental for development and survival of embryos.

Naturally occurring variation in development between littermate embryos, combined with altered patterns of uterine secretion in response to nutritional and endocrine signals, are the primary causes of asynchronous development (Vallett *et al.,* 2002). As discussed previously, follicle selection occurs over a protracted period of 5 – 7 days, resulting in the formation of a heterogeneous pre-ovulatory population characterised by differences in follicle diameter, varying concentration of steroids in the follicular fluid, and oocytes in a wide range of meiotic stages (Hunter and Weisak, 1990; Table 6). The actual pattern of pre-ovulatory follicular development and oocyte meiotic maturation within animals is skewed, such that most follicles and oocytes are at a similar stage of development to each other, but there is a small population (ca. 30%) that are less developed. Similarly, time of ovulation is skewed with about 70% of follicles ovulating over a short period of time, with the remaining 30% of oocytes released over a more protracted period (reviewed by Pope *et al*., 1990). This disparity in follicle development and oocyte maturity, has been identified as the principal cause of asynchronous development between litter-mate embryos during the first 12 days of gestation, with later ovulated oocytes becoming the least developed embryos (Hunter and Weisak, 1990; Pope *et al.,* 1990; Xie *et al.,* 1990).

Table 6. Stage of meiosis of oocytes obtained from individual gilts at either 27 or 30 hours after the onset of oestrus (adapted from Pope *et al.,* 1990).

Hours after onset of oestrus	Stage of Meiosis				
	Germinal vesicle	Germinal vesicle breakdown	Metaphase 1	Anaphase 1 to Telophase 1	Metaphase 2
27	3	9			
27	4	1	15		
27			5	11	
27			1	1	9
27			3		16
27			4	2	8
30			2	2	12
30			1	2	9
30			1		13
30			1		16
30			2		11

During days 10 to 12 of gestation, pig blastocysts synthesise and secrete increasing amounts of oestrogen (Pope *et al.,* 1990), stimulating endometrial secretion of proteins and growth factors involved in conceptus elongation and implantation (Geisert and Yelich, 1997). However, uterine secretions that facilitate elongation and implantation of the more developed embryos may not be conducive to the continued development of the least developed embryos, resulting in their elimination (Pusateri *et al.,* 1990). More specifically, less developed blastocysts may not be protected against the embryo-toxic effects of factors such as retinol and uteroferrin that are released into the uterine lumen in response to oestrogen secreted by the conceptus (Vallet *et al.,* 1996; Geisert and Yelich, 1997). Alternatively the attachment of less developed blastocysts may be prevented by changes in uterine surface protein (Soede *et al.,* 1999).

It has been suggested that peri-ovulatory progesterone concentrations mediate nutritional effects on early embryo survival (Jindal *et al.,* 1996 and 1997; Almeida *et al.,* 2000). This is supported by two recent studies. The first demonstrated that a higher plane of feeding (two times maintenance) from day one of gestation delayed the increase in progesterone concentration, and increased embryonic mortality by 20% up to day 28 of pregnancy, compared to gilts receiving 1.5 times maintenance (Jindal *et al.,* 1996). The second study indicated that treating gilts with six intra-muscular injections of progesterone at 12-hourly intervals between 24 and 108 hours after oestrus detection reversed the detrimental effects of higher plane feeding on embryo survival (Jindal *et al.,* 1997). It was concluded that reducing feed intake from day 1 of pregnancy (Day 0 refers to the 24 hour period after the first detection of oestrus) was the most beneficial in terms of increased embryo survival, and that feed intake around the time of ovulation alters the pattern of progesterone secretion due to variations in follicle luteinisation and progesterone secretion. Further, changes in peripheral progesterone concentration may reflect altered metabolic clearance rate. Soede *et al.* (1999) demonstrated a link between feed intake during early pregnancy and both embryo development and uterine protein secretion. In a separate study, progesterone treatment on days two and three of the oestrous cycle or pregnancy resulted in an earlier

rise in the secretion of total protein, uteroferrin and retinol binding protein (Vallett *et al.,* 1998). Together, these studies suggest that changes in progesterone concentration, induced by feeding level or exogenous administration, affect embryo survival via affects on the uterine secretion of proteins, such as uteroferrin and retinol binding protein.

Recent studies of Ashworth *et al.* (1999a and b) indicate that nutrition before mating may in fact have a greater impact on embryo survival than feed intake after mating (Table 7). This supports the earlier finding of Almeida *et al.* (2000) that moderate feed restriction between days 8 and 15 of the oestrous cycle reduced embryo survival in association with lowered peripheral progesterone concentrations. Therefore, in addition to the effects of feeding level around the time of ovulation on the uterine environment and luteal function after ovulation, nutritional effects on the physiological development of recruited and pre-ovulatory follicles may also alter progesterone production and/or secretion (Mao and Foxcroft, 1998), as well as affecting the quality of the oocyte released.

Table 7. Effect of feed intake, before and after mating, on ovulation rate, embryo number and embryo survival on day 12 of gestation in Meishan gilts (adapted from Ashworth *et al.,* 1999a).

Feed intake before mating*	Feed intake after mating*	Ovulation rate	Number of embryos	Embryo survival (%)
Maintenance	Maintenance	19.0	13.8	73.4
	High	19.3	14.5	75.9
High	Maintenance	22.4	22.0	99.0
	High	22.3	20.0	89.6

*Maintenance: 1.15 kg day $^{-1}$, High: 3.5 kg day $^{-1}$; Gilts fed a complete diet containing 20.2% crude protein and 1.2% lysine and supplying 14 MJ DE kg^{-1}; diet before mating fed for entire oestrous cycle before mating; diet after mating commenced on the first day after detection of oestrus.

Oocyte quality

Growth and development of the follicle-oocyte complex before ovulation has a profound effect on an oocyte's ability to develop into a viable embryo (Hunter, 2000). Developmental competence of the oocyte or 'oocyte quality' describes the ability of the oocyte to resume meiosis, maintain a stable metaphase 2, be fertilised, and complete the early stages of embryonic development. Growth of the follicle from the primordial through to the ovulatory stage takes approximately 103 days (Morbeck *et al.,* 1992). During this period, substrates and growth factors of somatic cell origin, obtained either directly from the cumulus cells, or indirectly from the follicular fluid, are vital for the growth and maturation of the oocyte. Altered availability of essential metabolic substrates combined with differential expression of growth factors, affects the maturational state of the oocyte and hence embryogenesis. Further, physiological diversity of follicles within the pre-ovulatory pool results in the release of oocytes that differ in their ability to develop under the same uterine conditions (Hunter, 2000; Eppig, 2001; Hunter *et al.,* 2004). The ovulation of meiotically immature oocytes is responsible for a large portion of the embryo losses that occur between days 5 and 10 of pregnancy (Geisert and Schmitt, 2002).

The effect of nutrition before mating on embryo survival appears to be partially mediated by changes in follicle steroidogenesis and oocyte maturation (Zak *et al.,* 1997b; Ferguson *et al.,* 2003), which influence the developmental competence of the oocytes released at ovulation. This is supported by two studies involving primiparous sows in which the feeding regime during the previous lactation that resulted in decreased embryo survival rates in the subsequent gestation also reduced the ability of oocytes obtained from pre-ovulatory follicles to reach metaphase 2 when matured in vitro (Zak *et al.*, 1997a and b). In the study of Zak *et al.* (1997b) a greater proportion of pre-pubertal oocytes derived from an abattoir were able to reach metaphase 2 when matured in the presence of follicular fluid obtained from sows that were fed ad-libitum between days 21 and 28 of lactation compared to those matured with follicular fluid from sows that were fed restrictively during the same period. Ferguson *et al.* (2003) demonstrated that gilts on a high feeding level (3.5 kg day^{-1}) before ovulation possessed a greater proportion of oocytes in the presumptive ovulatory pool that were able to reach metaphase 2 compared to oocytes obtained from maintenance fed gilts (1.35 kg day^{-1}) (0.88 $\pm$ 2.7 versus 0.68 $\pm$ 6.5, respectively). Concentrations of oestradiol and progesterone were also higher in the follicular fluid of gilts on a high feeding plane (Ferguson *et al.,* 2003). It has been suggested that follicle steroidogenesis is increased in high-fed gilts as a result of increased portal blood flow allowing increased oestradiol metabolism and a subsequent reduction in negative feedback at the hypothalamus (Ferguson *et al.,* 2003). Further, in addition to the indirect effects of nutrition on the pulsing nature of GnRH and LH secretion, there is a growing body of evidence to suggest that nutritionally induced alterations in metabolic hormones have a direct effect on ovarian function. For example, altered activity and expression of insulin-like growth factors 1 and 2 and their binding proteins are believed to alter granulosa cell steroidogenesis as well as oocyte quality (see Prunier and Quesnel, 2000; Hunter *et al.,* 2004). Together, these studies suggest that differences in developmental competence of oocytes induced by nutrition contribute to differences in embryo survival.

It also appears that factors within the follicular fluid are involved in the regulation of meiotic competence of oocytes, and that nutritionally induced alterations in oocyte quality may be mediated, at least in part, by changes in the intra-follicular environment.

Conclusions

Recent findings strongly suggest that sexual maturity, as measured by response to boar exposure, occurs later in modern lean genotypes and that current gilt recommendations (eg Hughes *et al*., 1996) need to be modified accordingly. Puberty is attained more rapidly and with greater synchrony within cohorts of gilts, when first boar exposure commences at 26 – 29 weeks of age, whereas commencing boar exposure at 23 weeks of age results in a delayed and asynchronous pubertal response (see Table 3). Further studies are necessary to establish the optimal duration and frequency of boar exposure required to illicit a maximal response.

Recent results suggest that ovulation rate will be sufficient at the pubertal oestrus to maximise potential first litter size. Equally, delaying first mating until the second oestrus does not appear to be associated with increased embryo survival. Therefore, the choice of when the gilt is to be mated/inseminated appears to be determined by the body condition status that is required to maximise her lifelong performance in the herd and not by any need to delay mating in order to raise ovulation rate to an adequate level, or reduce early embryo mortality. However, the effects of uterine capacity on fetal survival, and hence first litter size are an area in need of further research.

Last, the way gilts are fed clearly exerts a dramatic influence on their reproductive success. Most reports suggest that attainment of puberty is unlikely to be significantly influenced by nutrition, except where the supply of nutrients is severely reduced. However, there are clear indications that subtle changes in nutrition before and during ovulation have a profound effect on potential litter size. High feed intake before mating increases both the number and quality of oocytes ovulated and the ability of fertilised ova to remain viable through the first 3-4 weeks of gestation, whereas low level feeding immediately after ovulation appears to increase the number of embryos that survive.

Gilt management practices – a commercial case study

R.J. Smits*, K. Shaw* and L.J. Johnston**

*QAF Meat Industries, Corowa NSW 2646. **West Central Research and Outreach Centre, University of Minnesota, Morris, MN 56267, USA.

Abstract

Sow replacement levels depend in part on a suitable gilt management system. Recommended gilt management practices have been relatively consistent for the past twenty years but, worldwide, breeding herds are now displaying a trend of poor sow longevity and low lifetime productivity. This paper summarises current gilt management recommendations and describes the reproductive outcomes of an alternative approach for increasing tissue reserves in genetically lean gilts. To increase the pool of cycling gilts on one of our commercial farms we compared the rearing of gilts in eco-sheds with traditional sheds. Housing gilts in deep litter eco-sheds increased live weight and back fat P2 at 27 weeks of age compared to gilts housed conventionally. With eco-shed rearing, the onset of oestrous was improved and the wastage of gilts reduced ($P < 0.001$). When assessed over two parities, sow retention and total piglet production per entered gilt was increased ($P < 0.001$). These results show that improved breeder performance can be achieved by changing housing conditions to alter the phenotype of genetically lean gilts.

Introduction

Pig industry recommendations for managing gilts are well documented and have been fairly consistent since the mid-1980s. However, with the push towards leaner progeny, the breeding sow genotype is now leaner and of a larger size at maturity than the sow genotypes previously studied (Edwards, 1998; Levis, 2002). With poor, or at best static, productivity (Hughes and Varley, 2003; Levis, 2005) and sow replacement rates in excess of 60% in Australia (Australian Pork Limited, 2004), many pig producers have focused again on managing gilts for lifetime performance. At QAF Meats, like other commercial operations, we require gilts and sows to cycle regularly, produce and wean large litters and remain a profitable breeders for as long as possible (*see* Levis, 2005; in this symposium). However, these objectives were not consistently being achieved at one of our commercial farms and this paper briefly summarises current management recommendations and reports on a new way to rear gilts that are genetically lean.

Current recommendations and body composition targets for breeding gilts

Levis (2002) and Hughes *et al.* (2003) reviewed the scientific literature and published recommended gilt management practices and targets. Both publications acknowledge that the genotypes used in many of these experiments were earlier maturing and fatter than the fast-growing, lean genotypes used today. These recommendations are summarised in Table 1 and are compared with conclusions from the 1989 APSA symposium entitled 'Pre-mating management of the gilt' by Paterson (1989), King (1989) and Hemsworth (1989a).

Table 1 is by no means an exhaustive list of recommendations for good gilt preparation. For instance, emphasis is also needed at selection for feet and leg structure and strength; a suitable breeding diet must be offered *ad libitum* for at least two weeks before mating; suitable housing is needed for welfare and climate control and; selection for a functional underline is required.

The proportion of the gilt pool that reached puberty at QAF's piggeries in Corowa began to decline during 2002. This had a significant economic effect due to its impact on reaching mating budgets and hence the volume of weaners produced. The problems of an inadequate supply of cycling gilts continued for several months. Data collected at 29 weeks of age indicated the live weight and particularly P2 back fat of the breeding gilts (120-125 kg and 12 mm) were well below the recommended targets in Table 1. A new approach to rearing gilts so that the number of matings could be increased was required.

Table 1. Current recommendations for management practices and targets (adapted from Levis, 2002; Hughes *et al.,* 2003) compared with recommendations from the 1989 APSA symposium.

Management practice or target	Levis (2002)	Hughes *et al.* (2003)	1989 APSA Hemsworth (1989b)
Boar stimulation			
- Age at start (days)	170-190 d	+160 d	160-170 d
- Frequency (stimulation/day)	1-2	1	1-2
- Contact (full or fence line)	full	full	Full
Group size (gilts/pen)	10-30	4-40	inconclusive
Stocking density (m^2/gilt)	NR	1.5-2.0	min. 2.0
Oestrus at mating	2-3	2-3	2-3
Age at mating (range in days)	220-250 d	200-250 d[1]	190-220
Live weight (kg)			
- 20 weeks of age	NR	min. 85 kg	NR
- mating	NR	min. 130 kg	min. 110 kg
Back fat P2 (mm)			
- 20 weeks of age	min. 13 mm	12-14 mm	inconclusive
- mating	NR	+16 mm	inconclusive

[1]No range given as a recommendation: refers to data that maximises lifetime litter size. NR: No recommendation specified.

Using deep litter eco-sheds to increase body tissue reserves before mating

Pigs reared in deep litter eco-sheds are often fatter than those reared under traditional housing (Connor, 1995; Brumm, 1999; Honeyman *et al.*, 1999; Payne *et al.*, 2000). Slaughter pigs of the QAF genotype often grow faster in eco-sheds and are 1-2 mm fatter at the P2 site than pigs of similar age from conventional production systems. Knowing that QAF's genotype was very lean and fast growing, we hypothesised that we could change sow body condition by altering the housing conditions of the breeder gilts. Our aim was to increase the weight and body fatness of the gilt before mating to levels closer to the recommended targets (Table 1). In a preliminary study, we recorded the live weight and back fat P2 of gilts at 27 weeks of age when reared from 9 to 27 weeks in either conventional housing or deep-litter housing in an eco-shed. Our hypothesis was that housing gilts in an eco-shed environment would increase body weight and fatness of gilts at 27 weeks of age compared with females reared in conventional housing.

Three hundred gilts that had been reared conventionally were housed in grower accommodation (90 per pen; 0.45 m^2/pig) from 9 to 17 weeks of age (initial live weight: 30.4 ± 0.3 kg). From 17 to 27 weeks of age the gilts were housed in finisher accommodation (45 per pen; 0.65 m^2/pig). The conventional accommodation had partially slatted floors and pens were organised in a continuous flow shed with different ages of animals. Three hundred gilts reared in eco-sheds were housed on rice hulls (300 mm deep) with a stocking rate of 1.35 m^2/pig in groups of 100 animals from 9 to 27 weeks of age (initial live weight: 27.8 ± 0.73 kg). Gilts in both treatments were offered the same diet *ad libitum*. Diets in the grower phase (14.0 MJ DE/kg) were phase fed every three weeks with available lysine decreasing from 0.75 g available lysine/MJ DE to 0.62 g available lysine/MJ DE. At 18 weeks of age, all gilts were offered a finisher diet formulated to 13.8 MJ DE/kg and 0.54 g available lysine/MJ DE. Two replicates were carried out consecutively commencing in May and ending in October. At 27 weeks of age, animals were weighed individually and their back fat depth at the P2 site measured by real time ultrasound. The data were analysed by General Linear Model ANOVA using replicate as a random factor. The data showed that we could increase the weight and fatness of breeding gilts by rearing them in a different housing system (Table 2).

Table 2. Live weight and back fat thickness (mean ± SE) of gilts at 27 weeks of age reared either in conventional housing or in an eco-shed filled with a deep litter of rice hulls from nine weeks of age.

	Live weight (kg)	Back fat P2 (mm)
Conventional	120.3 ± 0.73[a]	8.8 ± 0.15[a]
Eco-shed	130.3 ± 0.71[b]	11.1 ± 0.15[b]
P value	0.001	0.001

[ab]Mean values within columns with different superscripts differ significantly (P<0.01).

During 2003, we had the opportunity to rear about half of our breeding gilts from 17-29 weeks of age on an off-site farm using twelve Rheem Canvacon™ roofed eco-sheds (9.1 x 45m) with rice hulls as bedding. We recorded the reproductive performance and the retention in the herd over the first two parities for gilts reared in eco-sheds and compared their performance with gilts reared concurrently in the conventional housing system.

Description of management system for rearing gilts

Gilts were transferred from conventional grower accommodation to the off-site farm at 17 weeks of age and reared as a weekly 'batch' of 300 within an eco-shed that provided 400 m^2 (1.35 m^2/gilt) floor space until 29 weeks of age. Gilts in eco-sheds received no boar contact due to the difficulty of implementing a stimulation program in these facilities with a large group size. Gilts were returned to the piggery where they entered the mating shed and were managed the same as conventionally-reared gilts. Concurrently, gilts housed in the continuous flow, conventional system were housed in groups of 26 gilts per pen in the gilt pool at 1.25 m^2/gilt from 17 to 29 weeks of age. At 24 weeks of age, boars were used to stimulate gilts housed in conventional sheds only. The boar stimulation regime included full boar contact once a day during Monday through to Friday for 20 minutes per day. Gilts in each housing system had ad libitum access to the same diet from 17 weeks of age (13.8 MJ DE/kg and 0.54 g available lysine/MJ DE) until mating. Following arrival to the breeding unit by truck transfer at 29 weeks of age, gilts from each group were stimulated for 10 minutes every day in full contact with mature boars (>9 months) as part of their oestrus detection.

Reproductive performance results to date

Data for the reproductive performance of gilts assigned to the two housing systems were analysed by General Linear Model ANOVA, with month of entry taken as a random factor. There was no significant difference in either gilt litter size or second parity litter size born between the two housing systems (Table 3). Each week a subset of 220 gilts were weighed individually at 29 weeks, with this amounting to about 160 gilts from conventional housing and 60 from the eco-shed housing systems. Gilts housed in eco-sheds were significantly heavier at 29 weeks of age than gilts housed conventionally (143 ± 0.3 kg and 127 ± 0.3 kg, respectively) ($P<0.001$).

Table 3. Litter size born[1] for gilts reared in conventional housing or eco-sheds before mating (19-29 weeks of age) .

	1st litter			2nd litter[2]		
	Litters	Born live	Total born	Litters	Born live	Total born
Conventional	785	9.3 ± 0.11	10.2 ± 0.11	551	11.0 ± 0.13	11.9 ± 13
Eco-shed	1119	9.2 ± 0.09	10.2 ± 0.09	783	11.0 ± 0.11	12.0 ± 0.11
P value		0.871	0.827		0.806	0.388

[1]Litter size comparison analysed by one-way ANOVA using month of entry to the breeding site as a random factor. [2]Litter size resulting from weaned 1st litter sows that were bred skip-a-heat (Clowes *et al.,* 1994)

Differences in the proportion of animals mated and subsequently retained in the herd were compared using Chi-square comparison. Gilt wastage was 9.8% for eco-sheds and 24 % for the conventional system ($P<0.001$; χ = 144.2). Gilts that showed no visible signs of oestrus after 36 weeks of age were culled as anoestrus. Of the reasons given for removal, 105 of 1673 of gilts from eco-sheds (6.3%) were classed as anoestrous compared with 217 of 1375 of gilts sourced from the conventional system (15.8%; $P<0.001$; χ=72.2). There were also differences ($P<0.001$) in the mating age of gilts. Gilts reared in eco-sheds were mated earlier (214.9 ± 0.4 days of age) than gilts reared in the conventional system (223.1 ± 0.4 days; mean ± SE).

Compared to conventional rearing, fewer animals were removed from the unmated gilt pool in the eco-sheds for physical reasons, including death, locomotor problems, body condition and udder damage was reduced ($P<0.001$; χ=32.0). Of the gilts sourced from the eco-sheds, 59 were classed as physical removals (3.5% of entered gilts) compared with 114 gilts reared under conventional housing conditions (8.3% of entered gilts). As a consequence of a lower gilt wastage due to fewer removals for anoestrous and physical causes, there were significantly more gilts mated when they were reared under the eco-shed housing system (Table 4; Figure 1).

Housing gilts in the eco-shed system described in this case study increased the number of gilt matings available for sow replacement in the QAF breeding-herd. The increased proportion of gilts mated was maintained through weaning after parity two (Table 4). From a production viewpoint, this has important implications as it provides additional mature sows for re-breeding. As Levis (2005) discussed earlier in this symposium, there is an economic advantage in retaining older parity sows compared to gilts due to the costs associated with replacement gilts.

Table 4. Females retained from farm entry at 29 weeks of age to weaning after the second litter.

	Gilts	1st Litter sows			2nd litter sows		
	Entered	Mated	Farrowed	Weaned	Mated	Farrowed	Weaned
Conventional	1375	1044	785	767	666	551	529
		(75.9[a])	(57.1[a])	(55.8[a])	(48.4[a])	(40.1[a])	(38.5[a])
Eco-shed	1673	1509	1119	1088	975	783	766
		(90.2[b])	(66.9[b])	(65.0[b])	(58.3[b])	(46.8[b])	(45.8[b])
χ value		113.0	30.9	27.1	29.4	13.9	16.5

[ab]Values within columns with different superscripts differ (P < 0.001). Females retained in the herd as a percentage of gilts entered number in parenthesis.

Table 5 summarises the cumulative number of piglets born over two parities. Gilts were evaluated from gilt entry at 29 weeks of age to the completion of two parities, a period that ranged from 60 to 85 weeks for individual sows. Unmated gilts and/or mated gilts that failed to farrow were given a value of 0. Thus, the data in Table 5 represent females that either produced no piglets; one litter of piglets or; two litters during the study period. The volume of piglets produced per 100 gilts entered was higher (P<0.001) from sows that were housed in eco-sheds as gilts (+18%).

Table 5. Cumulative number of piglets born over two parities from gilts reared either in conventional or eco-shed housing between 19-29 weeks of age.

	Number gilts	Total piglets born alive		Total piglets born live
	entered	1st parity	2nd parity	Per 100 gilts entered[1]
Conventional	1,375	7,358	6,005	972[a]
Eco-shed	1,673	10,246	8,593	1,126[b]
P value				0.001

[1]Litter size comparison analysed by one-way ANOVA using month of entry to the breeding site as a random factor. [ab]Mean values within columns with different superscripts differ (P<0.001).

Although there was a higher retention of gilts at the point of first mating, the rate of retention was similar for both housing systems at each stage of the cycle thereafter (Figure 1). This suggests the effects of eco-shed housing are on short-term rather than long-term physiological responses. Increasing sow longevity involves several management factors including gilt housing and selection; nutrition in terms of adequate energy, protein and minerals and vitamins; feeding levels during gestation and lactation; sow housing conditions; sow health; and genetics. Hughes and Varley (2003) concluded that gilts need to enter the breeding herd with a greater store of fat and protein than they currently do and be fed throughout their breeding life at levels that minimise the depletion of these tissue stores. The notion of designing a nutritional program for the whole reproductive cycle for lifetime performance was also highlighted by Close and Cole (2000). The gilts from both housing systems described in this case study were fed and managed similarly during gestation, lactation and subsequent parities. It is possible that the heavier gilts at mating were unable to retain their extra tissue reserves as they became older. Production systems that result in heavier gilts entering the breeding herd need to adjust for higher maintenance requirements throughout the life of the sow. One such adjustment relates to changes in feeding rates from traditional levels as predicted by various nutrient-partitioning models (Close and Cole, 2000). Careful management of the nutritional requirements of the gilt, as they relate to lifetime productivity, needs further evaluation in commercial practice.

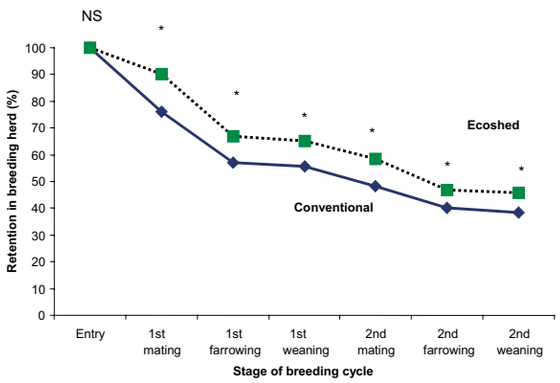


Figure 1. *The decline in sow retention from entry at 29 weeks of age over the first two parities as described in Table 4. NS: Non significant treatment effects. *Treatment differences were significant (P<0.001).*

The performance data we have collected does not allow us to determine why housing gilts in eco-sheds improved onset of oestrus and lowered gilt wastage. There are several factors that may have differed between the two housing systems: air quality; social interaction; the amount of heat loss through floor/bedding; longer duration of transport; activity level and bone and muscle development; feed intake; disease level; and boar stimulation regime. The observed outcome of improved gilt cycling and gilt survival highlights the opportunity to further investigate the mechanisms involved. The housing effect described above is likely to be the culmination of feed intake and utilisation, health and/or social cues on the activation of the hypothalamus-pituitary-ovarian axis as described by van Wettere *et al.* (2005) in this symposium. Carrying out a controlled experimental program would determine the relative effects of each on gilt performance and longevity.

Conclusion

Managing gilts to achieve a longer breeding life is highly desirable. We could not fully implement all recommended practices for gilt development within the constraints of our existing facilities. Consequently, we have adopted a different way of housing gilts before mating to improve the reproductive performance of gilts at QAF, Corowa. Under traditional housing systems, the QAF genotype for breeding gilts is leaner than recommended. Changing to rearing gilts in eco-sheds increased the weight and back fat of gilts entering the mating shed. We have shown that changes to pre-mating gilt management by using deep litter eco-sheds increased the proportion of gilts in oestrus and the total production of piglets from those gilts. However, the housing system before the first mating does not appear to influence sow survival or fertility.

Symposium conclusions

R.J. Smits

QAF Meat Industries, Corowa, NSW 2646.

The management of the gilt and sow for a profitable, high-performing herd requires knowledge of sow biological processes and a degree of economic assessment. There is now ample evidence to show that the sow herd is operating well below potential in terms of lifetime reproductive performance and longevity. This problem is not restricted to Australia or our genetic base, as evidenced by figures from the US and Canada also showing an increase in the rates of sow replacement. In this symposium we reviewed the economic advantages of retaining sows longer within the breeding herd and addressed the topic of managing gilts to improve lifetime performance. In addition, an example of how changing current management practices can affect commercial gilt and sow performance was provided.

Levis (2005) stated that the major goal when managing breeding sows is to keep the breeding female producing at a high level for as long as possible. His paper highlighted how little is known, or acknowledged, about the costs associated with a low-producing breeding sow at the industry and farm level. Examples were provided of computer models that can be used to calculate the costs and financial returns of possible changes or improvements to the way the herd is managed. Levis suggested that the complexity of existing models was a major obstacle to their adoption and that a more simplified profit model, combining reproductive output with the number of days the female is in the breeding herd, might be more valuable to commercial producers. This would allow producers to simulate specific changes in the way their own breeder herds could be managed.

The second paper by van Wettere *et al.* (2005) reviewed the literature on the biological processes involved in puberty attainment and how these processes may have changed with the evolution of a later maturing genotype. Recent experiments by the authors showed that the response of gilts to boar stimulation is maximised at a later gilt age than had previously been reported. These authors suggested that the hormonal axis responsible for recognising and processing boar stimuli may develop later in current lean genotypes than in the earlier maturing fatter genotypes previously investigated. They also reported how nutrition during the pre-pubertal period may affect the rate of sexual development (i.e. the timing of puberty attainment) and the number and quality of oocytes released at ovulation (i.e. ovulation rate and early embryo survival rate). Protein and energy intake may need to be restricted severely to delay puberty attainment. However, the authors also provided new evidence that showed subtle changes in nutritional restriction, or growth interruption during the mid to late oestrous cycle, may affect potential litter size via effects on the population of growing follicles in the ovaries and on the quality of oocytes that are subsequently ovulated.

The final paper by Smits *et al.* (2005) summarised the current recommendations for gilt management. As a case study the authors described a new approach to gilt management that has been adopted at QAF Meat Industries to combat a sustained period of delayed puberty in their commercial gilt herd. The approach, which involved rearing gilts in deep litter eco-sheds, was an effective way to increase the tissue reserves of genetically lean gilts before mating. Specifically, the authors highlighted the improvement in oestrus onset seen with the new approach and a reduction in the level of gilt wastage in the unmated gilt pool. However, it was concluded from the data available so far that rearing gilts in deep litter eco-sheds did not appear to improve sow longevity.

In summary, it is important to provide pig producers with management strategies that are appropriate for today's modern lean genotypes. Equally, pig producers need economic models that determine the cost-benefit of management changes that are designed to increase lifetime production of the sow. The papers presented in this symposium have challenged some of the current gilt management practices and offer some options for improving and economically justifying changes in gilt and sow management.

References

ALMEIDA, F. R. C. L., KIRKWOOD, R. N., AHERNE, F. X. and FOXCROFT, G. R. (2000). Consequences of different patterns of feed intake during the oestrous cycle in gilts on subsequent fertility. *Journal of Animal Science* 78: 1556-1563.

ARCHIBONG, A. E., ENGLAND, D. C. and STORMSHAK, F. (1987). Factors contributing to early embryonic mortality in gilts bred at first estrus. *Journal of Animal Science* **64:** 474-478.

ARCHIBONG, A. E., MAURER, R.R. ENGLAND, D. C. and STORMSHAK, F. (1992). Influence of sexual maturity of donor on in vivo survival of transferred porcine embryos. *Biology of Reproduction* **47**: 1026-1030.

ARMSTRONG, D. G. (2002). Steroidogenesis in bovine granulosa cells: The effect of short-term changes in dietary intake. *Reproduction* **123**: 371-378.

ARTHUR, S.A., KORNEGAY, E.T., THOMAS, H.R., VEIT, H.P. NOTTER, D.R. and BARCZEWSKI, R.A. (1983). Restricted energy intake and elevated calcium and phosphorus intake for gilts during growth. III. Characterization of feet and limbs and soundness scores of sows during three parities. *Journal of Animal Science* **56**:876-886.

ASHWORTH, C. J. and PICKARD, C. J. (1998). Embryo survival and prolificacy. In: J. Wiseman, M. A. Varley and J. P. Chadwick (eds.) Progress in pig science. p 303-326. Nottingham University Press.

ASHWORTH, C. J., ANTIPATIS, C. and BEATTIE. L. (1999a). Effects of pre- and post-mating nutritional status on hepatic function, progesterone concentration, uterine protein secretion and embryo survival in meishan pigs. *Reproduction, Fertility and Development* **11**:67-73.

ASHWORTH, C. J., BEATTIE. L., ANTIPATIS, C.J. and VALLET, J. L. (1999b). Effects of pre- and post-mating feed intake on blastocyst size, secretory function and glucose metabolism in meishan gilts. R*eproduction, Fertility and Development* **11**:323-327.

AUMAITRE, A. L., DOURMAD, J. Y. and DAGORN, J. (2000). Management systems for high productivity of sows in Europe. *Pig News and Information* **21**:89-98.

AUSTRALIAN PORK LIMITED (2004). Australian Pig Check Statistics. www.australianpigcheck.com.au (Australian Pork Limited: Canberra).

BABOT, D., CHAVEZ, E.R. and NOGUERA, J.L. (2003). The effect of age at first mating and herd size on the lifetime productivity of sows. *Animal Research* **52**:49-64.

BELTRANENA, E., AHERNE, F. X., FOXCROFT, G. R. and KIRKWOOD, R. N. (1991). Effects of pre- and postpubertal feeding on production traits at first estrus in gilts. *Journal of Animal Science* **69**:886-893.

BOOTH, P. J., CRAIGON, J. and FOXCROFT, G. R. (1994). Nutritional manipulation of growth and metabolic and reproductive status in prepubertal gilts. *Journal of Animal Science* **72**:2415-2424.

BRANDT, H., VON BREVERN, N. and GLODEK, P. (1999). Factors affecting survival rate of crossbred sows in weaner production. *Livestock Production Science* **57**:127-135.

BRITT, J.H, DAY, B.N., WEBEL, S.K. and BRAUER, M.A. (1989). Induction of fertile estrus in prepuberal gilts by treatment with a combination of pregnant mare's serum gonadotropin and human chorionic gonadotropin. *Journal of Animal Science* **67**:1148-1153.

BROOKS, P. H. and D. A. SMITH. (1980). The effect of mating age on the reproductive performance, food utilization and liveweight change of the female pig. *Livestock Production Science* **7**:67-78.

BRUMM, M. (1999). In 'Pork Industry Handbook'. Iowa State Univiersity, Iowa.

CALABOTTA, D.F., KORNEGAY, E.T., THOMAS, H.R., KNIGHT, J.W., NOTTER, D.R. and VEIT, H.P. (1982). Restricted energy intake and elevated calcium and phosphorus intake for gilts during growth. I. Feedlot performance and foot and leg measurements and scores during growth. *Journal of Animal Science* **54**:565-575.

CAMOUS, S., PRUNIER, A. and PELLETIER, J. (1985). Plasma prolactin, lh, fsh and estrogen excretion patterns in gilts during sexual development. Journal of Animal Science **60**:1308-1317.

CARDENAS, H. and POPE, W.F. (2002). Control of ovulation rate in swine. *Journal of Animal Science* **80**: (E. Suppl. 1): E36-E46.

CEDERBERG, E. and JONSSON, L. (1996). Sow culling in Sweden. *Proceedings of NJF Seminar No. 265 Longevity of sows.* Research Centre Foulum, Denmark, pp7-8.

CHAMBERLAIN, T. S. and HUGHES, P. E. (1996). The influence of mating frequency and nutrition on the stimulus value of boars. *Animal Reproduction Science* **43**:151-160.

CHRISTENSON, R. K., FORD, J. J. and REDMER, D. A. (1985). Maturation of ovarian follicles in the prepubertal gilts. *Journal of Reproduction and Fertility* Supplement No. **33**:21-36.

CLOSE, W.H. and COLE, D.J.A. (2000). 'Sow and Boar Nutrition' (Nottingham University Press: Nottingham).

CLOWES, E.J., AHERNE, F.X. and FOXCROFT, G.R. (1994). *Journal of Animal Science* **72**:283-291.

CONNOR, M.L. (1995). In 'Manitoba Swine Seminar'. Proc. 9. pp. 80-85.

COSGROVE, J. F. and G. R. FOXCROFT. (1996). Nutrition and reproduction in the pig: Ovarian aetology. Animal Reproduction Science **42**:131-141.

DELIGEORGIS, S. G., ENGLISH, P. R., LODGE, G. A. and FOXCROFT, G. R. (1984). Comparison of two methods for evaluating reproductive development in prepubertal gilts. *Animal Production* **38**:283-291.

DIJKHUIZEN, A.A., KRABBENBORG, R.M.M. and HUIRNE, R.B.M. (1989). Sow replacement: A comparison of farmers' actual decisions and model recommendation. *Livestock Production Science* **23**:207-218.

DIJKHUIZEN, A.A., KRABBENBORG, R.M.M., MORROW, M. and MORRIS, R.S. (1990). Sow replacement economics: An interactive model for microcomputers. *The Compendium on Continuing Education for the Practicing Veterinarian* **12**:575-579.

DIJKHUIZEN, A.A., MORRIS, R.S. and MORROW, M. (1986). Economic optimization of culling strategies in swine breeding herds, using the 'PorkCHOP computer program'. *Preventive Veterinary Medicine* **4**:341-353.

DYCK, G. W. 1988. Factors influencing sexual maturation, puberty and reproductive efficiency in the gilt. *Canadian Journal* of *Animal Science* **68:**1-13.

EASTHAM, P. R., DYCK, G. W. and COLE, D. J. A. (1986). The effect of age at stimulation by relocation and first mature boar contact on the attainment of puberty in the gilt. *Animal Reproduction Science* **12:**31-38.

EDWARDS, S. A. 1998. Nutrition of the rearing gilt and sow. In: J. Wiseman, M. A. Varley and J. P. Chadwick (eds.) Progress in pig science. p 361-382. Nottingham University Press.

ELSAESSER, F. (1982). In 'Control of Pig Reproduction' pp 93-116, eds D. J. A. Cole and G. R. Foxcroft. Endocrine control of sexual maturation in the female pig and sexual differentiation of the stimulatory oestrogen feedback mechanism. Butterworth Scientific.

EPPIG, J. J. (2001). Oocyte control of ovarian follicular development and function in mammals. *Reproduction* **122** 829-833.

ESBENSHADE, K. L., PATERSON, A. M., CANTLEY, T. C. and B. N. DAY. 1982. Changes in plasma hormone concentrations associated with the onset of puberty in the gilts. *Journal of Animal Science* **54**:320-324.

EVANS, A. C. O. and J. V. O'DOHERTY. (2001). Endocrine changes and management factors affecting puberty in gilts. *Livestock Production Science* **68:**1-12.

FERGUSON, E. M. et al. (2003). Effect of different nutritional regimes before ovulation on plasma concentrations of metabolic and reproductive hormones and oocyte matruation in gilts. *Reproduction* **126**:61-71.

FORD, S. P. (2003). Placental development, efficiency and embryonic mortality. In: J. Wiseman, M. A. Varley and B. Kemp (eds.) Perspectives in pig science. p 279-292. Nottingham University Press.

GAUGHAN , J. B., CAMERON, R. D. A., DRYDEN, G. M. and YOUNG, B. A. (1997). Effect of body composition at selection on reproductive development in large white gilts. *Journal of Animal Science* **75**:1764-1772.

GEISERT, R. D. and J. V. YELICH. (1997). Regulation of conceptus development and attachment. *Journal of Reproductio*n *and Fertility* **52**:133-149.

GEISERT, R. D. and SCHMITT, R. A. M.(2002). Early embryonic survival in the pig: Can it be improved? J*ournal of Animal Science* 80 (E. Suppl. 1): E54-E65.

GJEIN, H. and LARSSEN, R.B. (1995a). Housing of pregnant sows in loose and confined systems – a field study. 2. Claw lesions: Morphology, prevalence, location and relation to age. *Acta Veterinaria Scandinavica* **36**:433-442.

GJEIN, H. and LARSSEN, R.B. (1995b). Housing of pregnant sows in loose and confined systems – a field study. 3. The impact of housing factors on claw lesions. *Acta Veterinaria Scandinavica* **36**:443-450.

GJEIN, H. and LARSSEN, R.B. (1995c). The effect of claw lesions and claw infections on lameness in loose housing of pregnant sows. *Acta Veterinaria Scandinavica* **36**:451-459.

GRUPEN, C. G. (2003). Relationship between donor animal age, follicular fluid steroid content and oocyte developmental competence in the pig. *Reproduction, Fertility and Development* **15**: 81-87.

HEMSWORTH, P.H. (1989a). In 'Manipulating Pig Production II', pp. 319-322, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).

HEMSWORTH, P.H. (1989b). In 'Manipulating Pig Production II', pp.323-326, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).

HEMSWORTH, P.H. FINDLAY, J.K, and BEILHARZ, R.G. (1978). The importance of physical contact with other pigs during rearing on the sexual behaviour of the male domestic pig. *Animal Productio*n. **27:**201–207.

HERRICK, J. R., BRAD, A. M., KRISHER, R. L. and POPE, W. F.(2003). Intracellular adenosine triphosphate and glutathione concentrations in oocytes from first estrous, multi-estrous, and testosterone-treated gilts. *Animal Reproduction Science* **78:**123-131.

HONEYMAN, M.S., LAY, D.C., JEDLICKA, L., MUGGE, P., KLIEBENSTEIN, J. and DUFFY, M. (1999). Research and demonstration updates., ISU Rhodes Research Farm and PFI on-farm cooperator data. Swine Systems Options for IOWA 1999, Leopold Centre for Sustainable Agriculture. (Iowa State University: Ames).

HUGHES P.E. and VARLEY M.A. (2003). In 'Perspectives in Pig Science' pp. 333-355, eds J. Wiseman, M.A. Varley and B. Kemp. (Nottingham University Press: Nottingham).

HUGHES, P. E. (1982). Factors affecting the natural attainment of puberty in the gilt. In: Control of Pig Reproduction. p 117-138.

HUGHES, P. E. (1994). The influence of boar libido on the efficacy of the boar effect. *Animal Reproduction Science* **35**:111-118.

HUGHES, P. E. (2003). Recent advances in the feeding and management of the swine breeding herd. In: *Proceedings of the 2003 Canadian Society of Animal Science Meeting* Saskatoon, Saskatchewan.

HUGHES, P. E. and COLE, D. J. A. (1976). Reproduction in the gilt: 2. The influence of gilt age at boar introduction on the attainment of puberty. *Animal Production* **23**:89-94.

HUGHES, P. E. and VARLEY, M. A. (2003). Lifetime performance of the sow. In: J. Wiseman, M. A. Varley and B. Kemp (eds.) Perspectives in pig science. p 333-355. Nottingham University Press.

HUGHES, P. E., PEARCE, G. P. and PATERSON, A. M. (1990). Mechanisms mediating the stimulatory effects of the boar on gilt reproduction. *Journal of Reproduction and Fertility* Supplement **40**.323-341.

HUGHES, P.E., PEARCE, G. P. and HEMSWORTH, P. H. (1996). Reproduction. In 'Pig Production', pp 67-88, eds A.C. Dunkin and M. R. Taverner. (Elsevier).

HUGHES, P.E., THACKER, P.A., KIRKWOOD, R.N., McGLONE, J., TILTON, J.E., SINGLETON, W., REICKS, D., YESKE, P., LOULA, T., KIEHNE, R. (2003). CD – Reproductive Management of Pigs. 2nd Edition. (Sus Multimedia Publications: Melbourne).

HUNTER, M. G. (2000). Oocyte maturation and ovum quality in pigs. Reviews of Reproduction **5:** 122-130.

HUNTER, M. G. and WEISAK, T. (1990). Evidence for and implications of follicular heterogeneity. J*ournal of Reproduction and Fertility* Supplement **40**.63-177.

HUNTER, M. G., ROBINSON, R. S., MANN, G. E. and WEBB, R. (2004). Endocrine and paracrine control of follicular development and ovulation rate in farm species. *Animal Reproduction Science* **82-83**: 461-477.

JINDAL, R., COSGROVE, J. F. and FOXCROFT, G. R. (1997). Progesterone mediates nutritionally induced effects on embyronic survival in gilts. *Journal of Animal Scienc*e **75**:1063-1070.

JINDAL, R., COSGROVE, J. F., AHERNE, F. X. and FOXCROFT, G. R. (1996). Effect of nutrition on embryonal mortality in gilts: Association with progesterone. *Journal of Animal Scienc*e **74**: 620-624.

JORGENSEN, B. (2000a). Longevity of breeding sows in relation to leg weakness symptoms at six months of age. *Acta Veterinaria Scandinavica* **41**:105-121.

JORGENSEN, B. (2000b). Osteochondrosis/osteoarthrosis and claw disorders in sows, associated with leg weakness. *Acta Veterinaria Scandinavica* **41**:123-138.

KING, R.H. (1989). In 'Manipulating Pig Production II', pp. 315-318, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).

KINGSBURY, D. L. and RAWLINGS, N. C. (1993). Effect of exposure to a boar on ciculating concentrations of LH, FSH, cortisol and oestradiol in prepubertal gilts. *Journal of Reproduction and Fertility* 98: 245-250.

KIRKWOO, R.N. (1999). Pharmacological intervention in swine reproduction. **7:** 29-35.

KIRKWOOD, R. N. and AHERNE, F. X. (1985). Energy intake, body composition and reproductive performance of the gilt. *Journal of Animal Science* **60**:1518-1529.

KIRKWOOD, R. N. and P. E. HUGHES. (1979). The influence of age at first boar contact on puberty attainment in the gilt. *Animal Production* **29:** 231-239.

KIRKWOOD, R. N. and HUGHES, P. E. (1981). A note on the influence of boar age on its ability to advance puberty in the gilt. *Animal Production* **32**:211-213.

KNOX, R. V. and TUDOR, K. W. (1999). Influence of norgestomet in combination with gonadotropins on induction of estrus and ovulation in prepubertal gilts. *Journal of Animal Science.* **77:**1348-1352.

KNOX, R.V., K TUDOR,W., RODRIGUEZ- ZAS, S.L., ROBB, J.A. (2000). Effect of subcutaneous vs intramuscular administration of P.G. 600 on estrual and ovulatory responses of prepubertal gilts. *J. Anim Sci* **78**: 1732-1737.

KOENIG, J. L. F. and STORMSHAK, F. (1993). Cytogenetic evaluation of ova from pubertal and third-estrous gilts. *Biology of Reproduction* **49**:1158-1162.

KORNEGAY, E.T., BRYANT, K.L. and NOTTER, D.R. (1990). Toe lesion development in gilts and sows housed in confinement as influenced by toe size and toe location. *Applied Agricultural Research* **5**:327-334.

LE COZLER, Y., DAGORN, J., LINDBERG, J.E. AUMAITRE, A. and DOURMAD, J.Y. (1998). Effect of age at first farrowing and herd management on long-term productivity of sows. *Livestock Production Science* **53**:135-142.

LEVIS, D.G. (2002). Pork Information. Ohio State University website. http://porkinfo.osu.edu/Word%20Documents/giltdevDL.doc

LEVIS, D.G. (2005). Biological and economical evaluation of sow longevity. In 'Manipulating Pig Production X', p.169. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

LUCIA JR, T., DIAL, G. D. and MARSH, W. E. (2000). Lifetime reproductive performance in female pigs having distinct reasons for removal. *Livestock Production Systems* **63**: 213-222.

MAO, J. and FOXCROFT ,G. R. (1998). Progesterone therapy during early pregnancy and embryonal survival in primiparous weaned sows. *Journal of Animal Science* **76**:1922-1928.

MARTIN, G. B., RODGER, J. and BLACHE, D. (2004). Nutritional and environmental effects on reproduction in small ruminants. *Reproduction, Fertility and Development* **16**:491-501.

MARTINAT-BOTTE, F., BARITEAU, F., BADOUARD, B. and TERQUI ,M. (1985). Control of pig reproduction in a breeding programme. *Journal of Reproduction and Fertility Supplement* 33: 211-228.

MENINO, A. R., ARCHIBONG, A.E., LI, J-R, STORMSHAK, F. and ENGLAND, D. C. (1989). Comparison of in vitro development of embryos collected from the same gilts at first and third estrus. *Journal of Animal Science* **67**:1387-1393.

MORBECK, D. E., ESBENSHADE, K. L., FLOWERS, W. L. and BRITT, J. H. (1992). Kinetics of follicle growth in the prepubertal gilts. *Biology of Reproduction* **47**:485-491.

PATERSON, A. M. (1982). The controlled induction of puberty. In: Control of Pig Reproduction, Nottingham, United Kingdom. p 139-160.

PATERSON, A. M. and LINDSAY, D. R. (1980). Induction of puberty in gilts 1. Effects of rearing conditions on reproductive performance and response to mature boars after early puberty stimulation. *Animal Production 31: 291-297*.

PATERSON, A. M., HUGHES, P. E. and PEARCE, G. P. (1989). The effect of season, frequency and duration of contact with boar on the attainment of puberty in gilts. *Animal Reproduction Science* **21**:115-124.

PATERSON, A.M. (1989). In 'Manipulating Pig Production II', pp. 310-314, eds J.L. Barnett and D.P. Hennessy. (Australasian Pig Science Association: Werribee).

PATTERSON, J. L., BALL, R. O., WILLIS, H. J., AHERNE, F. X. and FOXCROFT, G. R. (2002). The effect of lean growth rate on puberty attainment in gilts. *Journal of Animal Science* **80**:1299-1310.

PAYNE, H., MULLAN, B.P. and TREZONA, M. (2000). Review of Alternative housing systems for pigs. A final report for the Pig Research and Development Corporation. Pig Research and Development Corporation: Canberra).

PEARCE, G. P. and PATERSON, A. M. (1992). Physical contact with the boar is required for maximum stimulation of puberty in the gilt because it allows transfer of boar pheromones and not because it induces cortisol release. *Animal Reproduction Science* **27**:209-224.

PEARCE, G. P. and P.E. HUGHES. (1987). The influence of boar-component stimuli on puberty attainment in the gilt. *Animal Production* **44**:293-302.

PENNY, R.H.C., CAMERON, R.D.A., JOHNSON, S., KENYON, P.J., SMITH, H.A., BELL, A.W.P., COLE, J.P.L. and TAYLOR, J. (1980). Footrot in pigs: Observations on the clinical disease. *Veterinary Record* **107**:350-351.

PHILIP, G. and HUGHES, P. E. (1995). The effects of contact frequency and season on the efficacy of the boar effect. *Animal Reproduction Science* 40: 143-150.

PigCHAMP. (2000). PigCHAMP DataSHARE. Available at http://www.pigchamp.com/2000Datashare.htm. Accessed April 19, 2005.

PigCHAMP. (2001). PigCHAMP DataSHARE. Available at http://www.pigchamp.com/2001Datashare.htm. Accessed April 19, 2005.

PigCHAMP. (2002). PigCHAMP DataSHARE. Available at http://www.pigchamp.com/2002Datashare.htm. Accessed April 19, 2005.

PigCHAMP. (2003). PigCHAMP DataSHARE. Available at http://www.pigchamp.com/2003Annual Datashare.htm. Accessed April 19, 2005.

PigCHAMP. (2004). PigCHAMP DataSHARE. Available at http://www.pigchamp.com/20044th Datashare.htm. Accessed May 11, 2005.

POPE, W. F., XIE, S., D. BROERMANN, M. and K. P. NEPHEW. (1990). Causes and consequences of early embryonic diversity in pigs. *Journal of Reproduction and Fertility*. Supplement **40:** 251-260.

PRESSING, A., G. D. DIAL, K. L. ESBENSHADE, and C. M. STOUD. (1992). Hourly administration of gnrh to prepubertal gilts: Endocrine and ovulatory responses from 70 to 190 days of age. *Journal of Animal Science* **70**: 232-242.

PRUNIER, A. and QUESNEL, H. (2000). Influence of the nutritional status on ovarian development in female pigs. *Animal Reproduction Science* **60-61**: 185-197.

PRUNIER, A., MARTIN, C., MOUNIER, A. M. and BONNEAU, M. 1993. Metabolic and endocrine changes associated with undernutrition in the peripubertal gilt. *Journal of Animal Science* **71:** 1887-1894.

PUSATERI, A. M., ROTHSCHILD, M. F., WARNER, C. M. and FORD, S. P. (1990). Changes in morphology, cell number, cell size and cellular estrogen content of individual littermate pig conceptuses on days 9 and 13 of gestation. *Journal of Animal Science* **68**: 3727-3735.

QUESNEL, H., PASQUIER, A., MOUNIER, A. M. and PRUNIER, A. (1998). Influence of feed restriction during lactation on gonadotropic hormones and ovarian development in primiparous sows. *Journal of Animal Science* **76**: 856-863.

QUESNEL, H., PASQUIER, A., MOUNIER, A. M. and PRUNIER, A. (2000a). Feed restriction in cycling gilts: Gonadotrophin-independant effects on follicular growth. *Reproduction*, *Nutrition and Development* **40:** 405-414.

QUESNEL, H., PASQUIER A., JAN, N. and PRUNIER, A. (2000b). Influence of insulin treatment and feed restriction on follicular development in cyclic gilts. Animal Reproduction Science **64:** 77-87.

RODRIGUEZ-ZAS, S.L., SOUTHEY, B.R., KNOX, R.V., CONNOR, J.R., LOWE, J.F. and ROSKAMP, B.J. (2003). Bioeconomic evaluation of sow longevity and profitability. *Journal of Animal Science* **81**:2915-2922.

ROZEBOOM, D. W., PETTIGREW, J. E., MOSER, R. L., CORNELIUS, S. G. and KANDELGY, S. M. E. 1995. Body composition of gilts at puberty. *Journal of Animal Science* **73:** 2524-2531.

SLEVIN, J. and WISEMAN, J. (2003). Physiological development in the gilts. In 'Perspectives in Pig Science', pp 293-332, eds J. Wiseman, M.A. Varley and B. Kemp (Nottingham University Press).

SMITS, R.J., LUXFORD, B.G., MORLEY, W.C., HUGHES, P.E. and KIRKWOOD, R.N. (2001). Boar exposure increases conception rate in induced 'once-bred' pre-pubertal gilts. In 'Manipulating Pig Production VIII' pp 194, eds P.D.Cranwell, (Australasian Pig Science Association, Werribee).

SMITS, R.J., SHAW, K. and JOHNSTON, L.J. (2005). Gilt management practices – A commercial case study. In 'Manipulating Pig Production X', p.168. ed J. E. Paterson (Australasian Pig Science Association, Werribee).

SOEDE, N. M., VAN DER LENDE, T. and HAZELEGER, W. (1999). Uterine luminal proteins and estrogens in gilts on a normal nutritional plane during the estrous cycle and on a normal or high nutritional plane during early pregnancy. *Theriogenology* **52**: 741-756.

STALDER, K.J., LACY, R.C., CROSS, T.L. and CONATSER, G.E. (2003). Financial impact of average parity of culled females in a breed-to-wean swine operation using replacement gilt net present value analysis. *Journal of Swine Health and Production* **11**:69-74.

STALDER, K.J., LACY, R.C., CROSS, T.L., CONATSER, G.E. and DARROCH, C.S. (2000). Net present value analysis of sow longevity and the economic sensitivity of net present value to changes in production, market price, feed cost, and replacement gilt costs in a farrow-to-finish operation. *The Professional Animal Scientist* **16**:33-40.

STEIN, T.E., DIJKHUIZEN, A., D'ALLAIRE, S. and MORRIS, R.S. (1990). Sow culling and mortality in commercial swine breeding herds. *Preventive Veterinary Medicine* **9**:85-94.

TILTON, S.L., BATES, R.O. and PRATHER, R.S. (1995). Evaluation of response to hormonal therapy in prepubertal gilts of different genetic lines. *Journal of Animal Science* **73**:3062-3068.

TUMMARUK, P., LUNDEHEIM, N., EINARSSON, S. and DALIN, A.-M. (2001). Reproductive performance of purebred Hampshire sows in Sweden. *Livestock Production Science* **68**:67-77.

VALLET, J. L., LEYMASTER, K. A. and CHRISTENSON, R. K. (2002). The influence of uterine function on embryonic and fetal survival. *Journal of Animal Science* **80**:(E. Suppl. 2): E115-E125.

VALLET, J. L., CHRISTENSON, R. K. and MCGUIRE, W. J. (1996). Association between uteroferrin, retinol-binding protein and transferring within the uterine and conceptus compartments during pregnancy in swine. *Biology of Reproduction* **55**:1172-1178.

VALLET, J. L., CHRISTENSON, R. K., TROUT, W. E. and KLEMCKE, H. G. (1998). Conceptus, progesterone, and breed effects on uterine protein secretion in swine. *Journal of Animal Science* **76**:2657-2670.

VAN WETTERE, W.H.E.J., MITCHELL, M., REVELL, D.R. and HUGHES, P.E. (2005). Management and nutritional factors affecting puberty attainment and first litter size in replacement gilts. In 'Manipulating Pig Production X', p.198. ed J. E. Paterson (Australasian Pig Science Association, Werribee).

WEBB, R., GARNSWORTHY, P. C., GONG, J. G. and ARMSTRONG, D. G. (2004). Control of follicular growth: Local interaction and nutritional influences. *Journal of Animal Science* **82** (E. Suppl.): E63-E74.

WHITTEMORE, C. T. (1996). Nutrition reproduction interactions in primiparous sows. *Livestock Production Science* **46:**65-83.

XIE, S., D. M. BROERMANN, K. P. NEPHEW, M. D. BISHOP, and W. F. POPE. (1990). Relationship between oocyte maturation and fertilization on zygotic diversity in swine. *Journal of Animal Science* **68**:2027-2033.

YANG, H., PETTIGREW, J.E., JOHNSTON, L.E., SHURSHON, G.C., WHEATON, J.E., WHITE, M.E., KOKETSU, Y., SOWER, A.F. and RATHMACHER, J.J. (2000). Effects of dietary lysine intake during lactation on blood metabolites, hormones and reproductive performance in primiparous sows. *Journal of Animal Science* **78**:1001-1009.

YOUNG, B. A., KING, G. J., WALTON, J. S., MCMILLAN, I. and KLEVORICK, M. (1990a). Age, weight, back fat and time of mating effects on performance of gilts. *Canadian Journal of Animal Science* **70:**469-481.

YOUNG, I. G., KING, G. J., WALTON, J. MCMILLAN, S., I. and KLEVORICK, M. (1990b). Reproductive performance over four parities of gilts stimulated to early estrus and mated at first, second or third observed estrus. *Canadian Journal of Animal Science* **70**:483-492.

ZAK, L. J., COSGROVE, J. F., AHERNE, F. X. and FOXCROFT, G. R. (1997a). Pattern of feed intake and associated metabolic and endocrine changes differentially affect postweaning fertility in primiparous lactating sows. *Journal of Animal Science* **75**:208-216.

ZAK, L. J., Xu, X., HARDIN, R. T. and FOXCROFT, G. R. (1997b). Impact of different patterns of feed intake during lactation in the primiparous sow on follicular development and oocyte maturation. *Journal of Reproduction and Fertility* **110**:99-106.

Live weight gain affects gilt reproductive performance

W.H.E.J. van Wettere*, M. Mitchell**, D.K. Revell* and P.E. Hughes***

*University of Adelaide, Roseworthy Campus, SA 5371. **University of Adelaide, SA 5011. ***Pig and Poultry Production Institute, Roseworthy, SA 5371.

The physiological maturity of the hypothalamic-pituitary-ovarian axis controls the time that gilts attain puberty in response to boar exposure and also the number of oocytes shed during the pubertal oestrus. This study was designed to test two hypotheses. First, that reproductive potential is affected by a low growth rate before puberty and second that the timing of this low growth is important to fecundity. This was achieved by studying the effects of four growth curves on the timing of the pubertal response to boar contact as well as ovulation rate at the pubertal oestrus. At 10 weeks of age and 29.4 ± 0.63 kg live weight (LW), 48 Large White/Landrace crossbred gilts were allocated randomly to one of two treatment groups; one group (n=24) was fed to a target of 100 kg (heavy) at 23 weeks of age, while the other group was fed to reach 80 kg (Light). By 23 weeks, Heavy gilts had significantly higher ($P<0.01$) LW and P2 back fat than Light gilts: 100.7 ± 1.7 kg versus 77.7 ± 1.3 kg, and 10.3 ± 0.37 mm versus 7.3 ± 0.25 mm, respectively. At 23 weeks of age, half of the gilts in each group (n=12) were fed to gain LW at 1.0 kg per day (high) until they attained puberty, while the remaining half (n=12) were fed to gain LW at 0.5 kg per day (low). At 25 weeks of age, Heavy gilts still had significantly higher ($P<0.01$) LW than Light gilts (111.0 ± 1.9 kg versus 88.0 ± 1.5 kg), and gilts with a High LW gain from 23 weeks onwards were significantly heavier ($P<0.01$) than gilts with a Low LW gain (103.2 ± 3.2 kg versus 96.4 ± 2.6 kg). The same diet was used across all treatment and daily feed intake adjusted weekly to achieve the described target LW and LW gains. Boar contact commenced at 25 weeks of age and consisted of 15 minutes of full contact with a mature boar twice each day in a detection mating area. Gilts were artificially inseminated at the pubertal oestrus with the reproductive tracts collected post slaughter at 10.2 ± 0.2 days after first detection of oestrus. The number of corpora lutea was counted and animals recorded as pregnant based on the presence of embryos in the uterine flushing. A two-way analysis of variance was used to examine treatment effects on puberty attainment and ovulation rate. There was no interaction between LW at 23 weeks of age and LW gain from 23-25 weeks of age on any of the traits reported here. Main effects only are presented in Table 1.

Table 1. Effects of pre-pubertal growth curve on puberty attainment and pubertal ovulation rate (mean ± se).

	Target live weight at 23 weeks of age		Target live weight gain from 23 weeks of age to puberty	
	Light	Heavy	Low	High
Mean days to puberty*	23.7^{b} ± 2.46	16.3^{a} ± 2.06	21.0 ± 2.43	18.8 ± 2.33
LW at puberty (kg)	96.7^{a} ± 2.6	118.4^{b} ± 2.7	102.0^{a} ± 2.9	113.1^{b} ± 4.2
LW gain from 23 weeks to puberty (kg/day)	0.76 ±0.10	0.71 ± 0.08	0.42^{a} ± 0.04	1.00^{b} ± 0.03
Ovulation rate (number of corpora lutea)	13.0 ± 1.03	13.7 ± 0.76	11.7^{a} ± 0.71	15.0^{b} ± 0.76

[ab] means in same row, within main effect, are significantly different ($P < 0.05$); * Gilts not attaining puberty by day 28 of boar exposure were ascribed a nominal days-to-puberty of 33 days.

Heavy gilts reached puberty 7.4 days earlier than Light gilts, irrespective of their growth rate from 23 weeks of age (Table 1). By day 28 of boar exposure, 75% of Heavy gilts compared to 43% of Light gilts were pubertal. LW gain from 23 weeks of age to puberty did not affect the pubertal response. Ovulation rate was unaffected by LW at 23 weeks of age, but was nearly 30% higher in gilts with a High LW gain from 23 weeks of age to puberty, compared to gilts with a Low LW gain. At slaughter, pregnancy rate was 83% and 70% for Heavy and Light gilts, and 80% and 77% for gilts with a High or a Low 23 week to puberty LW gain. The data indicate that differences in pre-pubertal LW gain alter the rate of sexual maturation in the gilt. It is suggested that the enhanced pubertal response of Heavy gilts is indicative of a more mature hypothalamic-pituitary-ovarian axis. It is noteworthy that High (or flush) feeding of Light gilts only restored ovulation rates to those of gilts that were high fed throughout the study. In conclusion, this study has shown that early growth rate (10-23 weeks of age) influences gilt response to boar stimulation, while nutrition immediately before the pubertal oestrus influences ovulation rate.

The effect of growth characteristics on the ovarian follicle population of pre-pubertal gilts

W.H.E.J. van Wettere*, M. Mitchell**, S. Schulz**, D.K. Revell* and P.E. Hughes***

*Discipline of Animal Science, University of Adelaide, Roseworthy Campus, SA 5371. **University of Adelaide, Adelaide, SA 5011. ***Pig and Poultry Production Institute, Roseworthy SA 5371.

The number and size of the follicles present on the ovaries of pre-pubertal gilts are thought to affect the time that puberty is attained in response to boar contact as well as the ovulation rate achieved during this first oestrus. Although a relationship between feed intake during lactation and both ovarian development and subsequent litter size has been demonstrated in primiparous sows, information regarding the influence of growth rate and nutrition on ovarian development in pre-pubertal gilts is limited (Prunier and Quesnel, 2000). This study determined the effect of live weight gain of pre-pubertal, non-cycling gilts on the size and dynamics of the antral follicle pool at 25 weeks of age. Sixty-four Large White/Landrace crossbred gilts were used in two blocks (n=8 gilts per treatment/block). At 10 weeks of age and 26.2 ± 0.35 kg live weight (LW), gilts were allocated randomly to one of two treatment groups; one group (n=16/block) was fed to a target of 100 kg (Heavy) at 23 weeks of age, while the other group was fed to reach 80 kg (Light). Thus, by 23 weeks of age Heavy gilts had significantly higher ($P<0.01$) LW and P2 back fat than Light gilts: 103.7 ± 1.3 kg versus 76.0 ± 1.3 kg, and 10.4 ± 0.3 mm versus 7.2 ± 0.2 mm, respectively. At 23 weeks of age, half of the gilts in each group (n=8) were fed to gain LW at 1.0 kg per day (High), whilst the remaining half (n=8) were fed to gain LW at 0.5 kg per day (Low). At 25 weeks of age, Heavy gilts still had significantly higher ($P<0.01$) LW than Light gilts (113.9 ± 1.7kg versus 89.1 ± 1.7kg) and gilts with a High LW gain from 23 weeks onwards were significantly heavier ($P<0.01$) than gilts with a Low LW gain (106.4 ± 2.9 kg versus 96.6 ± 2.6 kg). The same diet was used across all treatments and daily feed intake adjusted weekly to achieve the described target LW and LW gains. At 25 weeks of age, ovaries were collected following slaughter from non-stimulated gilts and surface follicles counted according to the following size classes: small (1-2.9 mm); medium (3-6 mm); and large (>6 mm). A two-way analysis of variance was used to examine treatment effects on gilt LW, LW gain, ovarian weight and the number of follicles within specific size categories. There was no interaction between LW at 23 weeks of age and LW gain from 23-25 weeks of age on any of the traits reported here. Main effects only are presented in Table 1.

Table 1. Effect of pre- and peri-pubertal live weight gain on ovarian weight and the distribution of follicles (Mean ± S.E) within specific size categories on the ovaries of 25 week old, non-cycling gilts.

	Target live weight at 23 weeks of age		Target live weight gain from 23-25 weeks of age	
	Light	Heavy	Low	High
Ovarian weight (g)	$7.1^{c} \pm 0.3$	$8.2^{d} \pm 0.4$	7.3 ± 0.3	8.0± 0.4
Number of small surface follicles	77.2 ± 6.8	75.6 ± 8.1	$92.8^{d} \pm 8.4$	$59.5^{c} \pm 5.2$
Number of medium surface follicles	$24.5^{a} \pm 2.6$	$32.5^{b} \pm 2.9$	$22.7^{c} \pm 2.7$	$34.3^{d} \pm 2.6$

[ab] Means in same row, within main effect, are significantly different ($P < 0.05$); [cd] Means in same row, within main effect, are significantly different ($P < 0.01$)

All gilts were considered pre-pubertal at time of ovary collection due to the absence of corpora lutea. The number of Large follicles was unaffected by treatment ($P>0.05$). The ovaries of gilts with a High LW gain from 23–25 weeks of age possessed significantly more ($P<0.01$) medium sized follicles than gilts with a Low LW gain (Table 1). The results show that short-term changes in feed intake (e.g. 23–25 weeks of age) significantly affect the distribution of follicles within the described size classes, presumably reflecting the influence of metabolic hormones on circulating gonadotrophin levels as well as follicle responsiveness to gonadotrophins. Interestingly, irrespective of 23–25 week LW gain, Light gilts possessed fewer medium sized follicles than Heavy gilts, which may be indicative of a less mature hypothalamic-pituitary-ovarian axis. In conclusion, pre-pubertal LW gain altered the dynamics of the proliferating follicle pool, which may have implications for gilt responsiveness to boar contact as well as pubertal ovulation rate.

References

PRUNIER, A. and QUESNEL, H. (2000). *Animal Reproduction Science* 60-61: 185-197.

8 Housing systems, nutrition and feed additives

A review - group housing for gestating sows – strategies for a productive and welfare friendly system

R. S. Morrison

QAF Meat Industries, Corowa, NSW 2646.

Abstract

Housing sows in a group environment for at least part of their gestation will be a requirement in the New Zealand pork industry from 2015. Group housing may also be a requirement in the Australian pork industry in the future, depending on the outcomes of the current review of the *National Model Code of Practice for the Welfare of Animals - Pigs*. Group housing enables the sow to perform locomotory behaviours and have social contact with other sows that may improve sow welfare. However, there are also associated risks when sows are housed in groups, such as increased aggression between sows that may impact negatively on animal welfare and reproductive performance. It is critical that piggery managers and stockpeople have an understanding of the social management of sows and the relationships between social behaviour and the physical pen environment and breeding and feeding management of the group-housed pregnant sow – as all these factors can affect the success of the group housing system. There are still many issues left unresolved regarding the optimum space and social requirements of sows housed within group systems. Further research to fine tune the management of group housing systems will need to be done within the next five years to ensure that pork producers are provided with strategies for implementing successful housing systems.

Introduction

Housing sows in gestation stalls has been highlighted as a welfare concern by sections of the Australian and international community and is currently one of the most controversial issue in pork production (Barnett *et al*., 2001). The importance that pork producers give to issues associated with the housing of gestating sows is changing rapidly. In New Zealand, the updated *Animal Welfare (Pigs) Code of Welfare 2005* released by the Minister of Agriculture will limit the optional use of dry sow stalls to a maximum of four weeks after mating, as from January 2015. In Australia, the *National Model Code of Practice for the Welfare of Animals - Pigs* is under review. As a result the housing of pregnant sows is currently a topic of much discussion within the Australasian pork industry, the research community, government representatives, veterinarians, retailers and animal liberation groups. There have also been changes to legislation in the European Union (EU) with gestation stalls being banned between four weeks after mating and one week before farrowing, from January 2013 (Council Directive 2001/88/EC; EU, 2001; Ministeries van Justitie en van LNV, 1998, 2003). There are countries within the EU that are more stringent in the use of gestation stalls and have banned the use of gestation stalls entirely. Regardless of the outcome from the Australian review, there is definitely a shift, in New Zealand and the EU at least, towards group housing of gestating sows for a significant part of their gestation period.

Individual sow housing during gestation was originally introduced to control feed intake of individual sows (i.e. reduce variation in feed intake) and protect the gestating sow during feeding from aggressive pen-mates, which can result in reduced embryo loss (Barnett *et al*., 2001). However there are disadvantages with this housing system because gestation stalls restrict sow movement, prohibit locomotory behaviours and do not allow direct social contact between sows. Barnett *et al*. (2001) suggested that about 62% of pregnant sows in Australia are housed in individual stalls at some stage of their gestation. The Australasian pork industry is reviewing group housing for gestating sows and is assessing the impacts of group housing system on sow welfare, reproduction and sustainability of the industry.

There is enormous variation in the design and management of group housing systems for gestating sows. In consequence, few statements can be made that are applicable to all group-housing systems for gestating sows (Gonyou, 2003a,b). In traditional group-housing systems, sows are housed in a relatively confined system. These conventional systems are indoors and have an automated ventilation system, fully- or partially-slatted floors and liquid manure handling systems. Pens usually house 5-50 sows with a floor space allowance of 1.4 –2 m^2 per sow. Other alternative group housing options include deep litter, large group housing and outdoor 'free-range' systems. Deep litter systems are naturally ventilated, have a floor base of deep litter (e.g. rice hulls or straw), consist of larger group sizes (ranging from 15-200 sows per pen) and greater space allowance of about 2.5 – 3.5 m^2 per sow.

The scientific literature is extremely controversial regarding the effects of housing systems on sow welfare and reproductive performance. For every research publication that confers negative impacts of group housing on sow welfare and reproductive performance, there is a dissenting paper suggesting the opposite.

Furthermore, many scientific papers reporting a welfare assessment of sows in a variety of gestation sow housing systems have not used measures of sow welfare that are rigorous and often do not incorporate behavioural, physiological, health and fitness responses to assess the biological functioning of the animal (Barnett *et al*., 2001; Barnett and Hemsworth, 2003).

In more recent times, the most comprehensive reviews of studies comparing sow welfare and reproductive performance across a range of housing systems have been carried out by Barnett *et al.* (2001) and McGlone *et al.* (2004). McGlone *et al.* (2004) carried out an extensive meta-analysis across 35 studies done between 1970 and 2002 to determine whether reproductive performance and welfare differed between group housing and gestation stalls. The authors showed there was a trend for sows in groups to have lower farrowing rates and this negative impact on farrowing rate was exacerbated in dynamic groups of sows where sows were mixed throughout gestation. There was no apparent significant differences between the reproductive parameters of piglets born alive, number of stillborn piglets, total piglets born and average piglet birth weight between the two housing systems (Table 1).

Table 1. Summary of reproductive performance of sows in group housing systems and stalls (from McGlone *et al*., 2004).

	Group housing	Stall housing	P value
Farrowing rate (%)	75.9 ± 2.9	83.3 ± 2.3	0.09
Piglets born alive per litter	9.9 ± 0.27	9.9 ± 0.27	0.87
Stillborn pigs per litter	0.73 ± 0.08	0.58 ± 0.09	0.26
Total pigs born per litter	10.8 ± 0.32	10.5 ± 0.36	0.53
Piglet birth weight (kg)	1.46 ± 0.03	1.43 ± 0.03	0.42

The authors concluded that within the confinement of their analyses, there was no evidence indicating there were negative effects on sow welfare and reproductive performance between well-managed stall and group housing systems. The authors did however emphasise the need for further comparisons of the effects of gestation housing on other welfare parameters such as sow longevity, injury and lameness. Research outcomes and the fact that a percentage of pork producers are already using group housing for a significant component of gestation indicate that group housing is a viable option. For example, Paterson *et al.* (1997) estimated that 62% of sows in Australia are housed in gestation stalls for a restricted time during gestation.

To ensure success of group housing systems for gestating sow it is necessary to have an understanding of the social environment (i.e. social management of sows and relationships between social behaviour and the physical pen environment) and breeding and feeding management of the pregnant sow. Therefore the aim of this literature review is to provide an overview of the current state of knowledge of these factors in a variety of group housing systems currently used in the Australasian pork industry.

The social environment of groups of gestating sows

An understanding of the social behaviour and organisation of sows living in a group is essential when attempting to achieve optimum welfare and reproductive performance in any pork production system. Animals behave in ways to maximise their individual fitness, which will ultimately enable their genes to spread (Manning and Dawkins, 1992). Group living enables animals to maximise their chance of survival, as animals living in a group can detect food and predators more quickly. However, animals within a group have to compete with each other for available resources, such as mates, food and shelter. Significant population dynamics occur within social groups as animals shift balances between these costs and benefits, which ultimately helps to regulate the size of the group and population density (Stricklin and Mench, 1987).

The social structure of the wild pig (*Sus scrofa)* is the matriarchal herd, which consists of up to four females with their offspring. The mature boars are not permanently associated with the herd and often form 'bachelor' groups or remain alone (Signoret *et al*., 1975). Stolba and Wood-Gush (1989) observed this social structure in intensively-reared domestic pigs living under natural conditions. Domestication has modified this free-ranging foraging pig to a more docile animal (*Sus scrofa domestica*), which is raised under intensive farming conditions (Signoret *et al*., 1975). The social behaviour of group-housed gestating sows has been described by Jensen (1980, 1982) and can be categorised into social tactile interactions, agonistic behaviours, sexual behaviour and social grooming. Social tactile interactions are important in maintaining social organisation. Pigs are often described as 'contact' animals and usually have tactile contact with other pigs when resting (Hafez, 1975). Social tactile interactions such as nose-to-body, nose-to-nose and anal nosing have been described in group-housed gestating sows by Jensen (1980, 1982). Jensen (1980) concluded that these behaviours are involved in individual animal recognition through olfactory and tactile cues and these behaviours

are important when groups of animals need to establish effective competitive and co-operative social relationships (Stricklin and Mench, 1987).

Agonistic behaviours are a series of behaviours that occur in response to a conflict - and include offence, defence, submissive or escape components. The behaviours may include contacts such as biting or pushing or non-contacts such as body postures or gestures. Aggressive behaviour is a component of agonistic behaviour and includes contact and threatening behaviours (Petherick and Blackshaw, 1987). Aggressive behaviour is often used interchangeably with agonistic behaviour and in the strict definition aggressive behaviour refers to the attack and actual fighting (Hart, 1985; Fraser and Broom, 1998). Parallel pressing, head-to-head knocks and levering are defined as aggressive behaviours, which have been reported by McBride *et al.* (1964), Beilharz and Cox (1967), Hafez and Signoret (1969), Signoret *et al.* (1975), Jensen (1980) and Fraser and Broom (1998). Head-to-head knocks have also been found as a component of play behaviour by van Putten and Dammers (1976). Agonistic behaviour has also been described by Hafez and Signoret (1969) and the authors considered agonistic behaviours to be part of the establishment of the social hierarchy. Social grooming rarely occurs among pigs, however this behaviour has been observed by Krosniunas (1979) and Stolba and Wood-Gush (1989). Pigs in a semi-natural environment formed small subgroups of the female with her offspring or other females and performed social grooming. It was concluded that this behaviour may be involved in maintaining social bonds within the social organisation. Pigs will rub against pen fixtures or upright objects and this can be considered part of grooming behaviour (Signoret *et al.*, 1975).

Domestic pigs organise and maintain a dominance hierarchy throughout their life. The function of the dominance hierarchy is to reduce aggression within the social group when resources are limited. A frequently used definition is 'priority of access to an approach to resources or away from an avoidance situation' (Stricklin and Mench, 1987). The social organisation of pigs usually reflects a dominant and subordinate relationship (Hart, 1985). Dominance-submission relationships occur when there is a consistent relationship as the result of agonistic reactions between two pigs (Puppe, 1996). Two types of social organisation have been described in the domestic pig; the teat order within a litter of piglets and the dominance hierarchy, which is established after weaning (Signoret *et al.*, 1975).

The teat order develops soon after birth and remains relatively stable to weaning, which enables efficient milk intake. Piglets will compete for the more productive anterior teats of the sow (McBride, 1963). If the teat order and dominance order were the same, stable dominance hierarchy may occur throughout life if the pigs were kept together (Ewbank, 1976). However, pigs are generally mixed in commercial pork production systems, particularly before and after weaning. This mixing enables pigs of similar body weight and sex to be housed together so that nutritional demands can be met. The current scientific literature on social hierarchies in pigs suggests that the dominance hierarchy is established during periods of high levels of agonistic behaviour immediately after mixing.

The structure of the dominance hierarchy is usually linear especially in smaller groups at low stocking densities (Ewbank, 1969, 1976). In an established linear hierarchy the animal occupying the top rank will take precedence in a competitive situation (e.g. feeding) without fighting any animals in the group. McBride *et al.* (1964) described three types of behaviour involved in the creation of the dominance hierarchy, especially in confined areas. The behaviours included (i) intra-specific aggressiveness, (ii) submission signals and (iii) an acceptance of submission. When two previously unacquainted pigs are placed in a competitive situation for resources, they will fight and eventually one will win and the other will lose. This process is repeated on subsequent occasions, and eventually the habitual loser responds to a mere threat of attack with submissive behaviour (Signoret *et al.*, 1975).

Meese and Ewbank (1972) suggested that a linear social hierarchy may not be as stable as previously assumed, and that pigs may alter position in the hierarchy frequently, especially in the middle or lower ranks of the hierarchy. Nevertheless, rarely does the most dominant pig change position (Meese and Ewbank, 1972). Once the social hierarchy is developed in a group of sows, the position in the social hierarchy may also impact on the welfare and reproductive performance of sows in group housing systems. The initial perception may be that lower ranking subordinate sows may have reduced welfare and reproductive performance in a group systems, however some authors have documented that socially-intermediate sows may be more at risk. Csermely and Nicosia (1991) showed that subordinate sows performed more stereotypic behaviour, were more restless, and had more interrupted suckling bouts then more dominant sows post farrowing. Mendl *et al.* (1992) and Nicholson *et al.* (1993) also showed that sows in the middle ranks of the hierarchy had reduced biological fitness, were susceptible to chronic stress, and had reduced reproductive performance compared to dominant and lower ranking submissive sows.

The length of time that is required for sows to establish social hierarchy (and therefore reduced aggression) can vary. Groups of sows can become stable after three to ten days (Van Putten and Van de Burgway, 1990), however other studies have shown that it can take up to eight weeks to establish stable social hierarchy (Arey and Edwards, 1998a). Factors that may be responsible for these inconsistencies in hierachial development include pen space allowances, group size and dynamics, feeding system and pen shape (Barnett *et al.,* 2001).

For a group of animals to establish effective competitive and cooperative social relationships it is necessary for the animals within the group to promptly identify and communicate with each other (Stricklin and Mench, 1987). A common estimate of the total number of group members that can be recognised by an individual is 20 to 30 in pigs (Fraser and Broom, 1998). Cues of social recognition are essential for the survival of individuals and ultimately the species because these cues relate to reproduction, maternal behaviour, protection and learning (Ewing *et al.*, 1999). The scientific literature is still somewhat unclear on how the mechanism of individual animal recognition operates in pigs, although it is evident that different cues of social recognition exist (Fraser and Broom, 1998). It appears that when pigs identify individuals within a group, auditory, olfactory, tactile and visual cues are important (Hart, 1985; Stricklin and Mench, 1987). Research has shown the importance of auditory cues in the organisation of social behaviour. The auditory range in pigs is from approximately 55Hz to 40 kHz and the pigs are most sensitive from 500Hz to 16kHz (Ewing *et al.*, 1999). More than 20 different auditory cues have been identified in the pig during social encounters such as resting, play, feeding, maternal, agonistic and sexual behaviours (Ewing *et al.*, 1999). Common auditory signals are grunts, barks, squeals and screams. Longer grunts are expressed in response to a familiar sound, and shorter length grunts are typical in excited pigs. As the pigs become more excited, the grunting frequency increases. Squeals and screams are associated with high levels of fear and barks are expressed when the pig is surprised or expressing dominance behaviour (Houpt, 1991).

Sows have well-developed hearing, which requires movement of the head to locate the sound, as the ears are short and immovable (Signoret *et al.*, 1975). McBride *et al.* (1964) have suggested that auditory cues may be used as a submission signal and pigs may be able to recognise sounds from specific individuals. Sows have highly developed olfactory capability (Ewing *et al.*, 1999), which is important for social recognition and feeding behaviour. McBride (1963) has suggested that piglets may show marking behaviour which assists is locating their position in the teat order. Boars determine mating receptivity of females using the vomeronasal organ as well as observing sexual behaviour (Ewing *et al.*, 1999). Vision is well developed in pigs as they have cones (duplex retina) and rods in the eyes which gives rise to high sensitivity and high acuity, and suggests the capability for colour vision (Ewing *et al.*, 1999). Ewbank (1974) studied the importance of vision and olfactory cues in the formation of social hierarchy. When opaque lenses were fitted over the pigs' eyes, the pigs formed a stable social hierarchy, however when the whole head was covered social hierarchies were not formed, which suggests that olfactory cues are involved in social recognition. Individual recognition is also assisted by vision, since hierarchies were also formed when eyes were uncovered.

Relationships between the social environment and the physical pen environment

The social behaviour of the sow can be influenced by the physical group pen environment where she is accommodated. Factors such as group size, pen space allowance and pen shape all impact on the social dynamics of the group. Other aspects of the sow's environment such as the housing of a boar with sows post mixing, the use of dynamic vs. static groups of sows and the addition of straw enrichment may influence the social dynamics of the group. An important outcome of group housing systems is to reduce aggression in group housed sows, as this behaviour, and the physiological stress response induced in early pregnancy can affect reproduction (embryo implantation and development) (Varley and Stedman, 1994) and welfare of the sow (injuries and chronic stress responses if aggression continues). Unfortunately there are few recommendations in the scientific literature for reducing aggression in group housed sows and this area requires further research.

Group size

The scientific literature is unclear on what are the recommended group sizes of gestating sows to ensure maximum welfare and reproductive performance. The majority of the research in this area has been conducted in growing pigs and poultry, and due to differences in social behaviour between classes of pigs and species, it is difficult to extrapolate these data to groups of pregnant sows. Barnett *et al.* (1984, 1986) showed that it was less stressful for gilts when they were housed in groups of four to eight, compared to in pairs, however there is little data in the literature on the impacts of larger group sizes on welfare and reproductive performance. Conventional thinking has been that smaller groups of pigs are able to form stable dominance hierarchies. It was thought that pigs kept in larger groups would have an unstable dominance hierarchy, as individual animal recognition may decline, and in turn outbreaks of aggressive behaviour. Little is known about the maximum number of pigs that are able to form a stable dominance hierarchy. Craig and Guhl (1969) studied hens in flocks of 100 to 400 birds, and hens in the larger groups tended to remain in particular areas of the shed and appeared to be 'territorial' in areas where they spent most of their time. The authors suggested that social recognition would be easier for the hens if they remained in a particular area of the shed, and associated with the same individuals daily. It would appear beneficial for animals to remain in smaller groups within a larger group, as excessive aggressive behaviour would be prevented (Craig, 1981).

Recent experiments carried out with laying hens and broiler chickens have shown that aggressive behaviour declines as group size increases (Pagel and Dawkins, 1997; Hughes *et al.*, 1997; Estevez, 1998; Nicol *et al.*, 1999). These authors have several suggestions to explain this finding. For example, that the greater availability of resources, such as total free space, availability of feeding places and preferred lying areas, may eliminate the need for a dominance hierarchy, which functions to control aggression when resources are limited. Furthermore, the animals may be more socially tolerant in large groups or animals may abandon all attempts to establish social hierarchies in large groups. However, other experiments conducted on laying hens (Bilcik and Keeling, 2000) have shown that most feather pecking activity and also the most aggressive attacks occurred in the largest group of hens. Fraser and Rushen (1987) reviewed the literature on aggression in pigs and stated that increases in group size and a reduction in floor space are associated with a higher incidence of aggression. However, the majority of experiments on pigs have studied group sizes of 60 or less. Morrison *et al.* (2003a) showed that groups of 200 growing pigs had a higher level of general activity and social tactile interactions with each other, which may have explained the increase in aggressive behaviours, which is similar to those results of Randolph *et al.* (1981) and Hsia (1984) and Spoolder *et al.* (1999). Spoolder *et al.* (1999) concluded that in larger groups individuals are more likely to encounter other pigs that they do not recognize or with which they have to reconfirm their relative rankings. Al-Rawi and Craig (1975) hypothesised that group size is positively correlated with a failure to resolve rank disputes without aggression, as the animals cannot recognise each other in large groups. This may result in an increase in social stress and may ultimately affect welfare and performance (Spoolder *et al.*, 1999).

The dominance hierarchy in pigs and the influence of group size on this behaviour has received considerable attention from researchers, but most of the research has been conducted on poultry or growing pigs in group sizes of 30 or less. It is unknown whether a dominance hierarchy exists in large groups of pigs, and whether the cues of social recognition are still functional to enable dominance hierarchies to be formed. Ewbank (1969) suggested that the maximum number of pigs kept in a pen should be the same as the number of individuals that can organise themselves into a behaviourally-stable group. The impact of large group sizes of sows on aggressive behaviour, the dominance hierarchy and in turn welfare and reproductive performance of gestating sows is unknown and requires further scientific investigation.

Pen space allowance

There are limited studies that give conclusive results on the recommended pen space allowance for group-housed gestating sows, therefore this area requires further research. The current recommended pen space for gestating sows ranges from 1.4-2m^2 /sow (Cale, 1979; New Zealand Code, 2005). Hemsworth *et al.* (1986b) and Barnett *et al.* (1992) and have clearly showed that there are negative consequences for sow welfare and reproductive performance when pen space is reduced to 1m^2 or less.

Weng *et al.* (1998) investigated sow aggression and incidence of injuries in groups of six sows with a pen space (2.0, 2.4, 3.6 and 4.8 m^2) and individual feeding stalls. This experiment showed that sow aggression and incidence of injuries reduces as pen space increased 2.4 to 3.6m^2. The authors emphasised that these results cannot be generalised to other group housing systems as there are system differences in terms of group sizes, feeding systems, management of social structure etc. Furthermore, the sows in that experiment had access to straw bedding which may have influenced the results.

The layout of the pen may influence aggression between sows in group housing systems. Modifications, including hides, barriers and partial stalls may reduce aggression in group housing systems (Petherick, 1987; Nehrig, 1981; McGlone and Curtis, 1985), however the success of these modifications may be dependent on the feeding system used. Barnett *et al.* (1993) showed that aggression between sows was less in rectangular pens with a pen space allowance of 1.4 m^2 per pig compared with square pens of a similar stocking density. In that experiment, there appeared to be no benefit of increasing the pen space allowance to 3.4 m^2 per pig.

The scientific literature is deficient in information on the interactions between group size and pen space allowance. For example, when sows are kept in larger groups there is additional 'free space' in the pen that may allow the opportunity for reduced pen space allowance in these groups. This area requires further investigation.

Housing boars with sows

Anecdotal evidence suggests that housing boars (either entire or vasectomised) with sows at the point of mixing in group systems reduces aggression between sows. This is common industry practice, however the scientific literature is deficient in information on how this management practice influences aggression. It is thought that the presence of boars (i.e. pheremones, visual stimulation, tactile contact) encourages the sows to redirect their aggressive behaviour towards the boar and/or the boar pheremones may quieten the sows. Androsterone present in the saliva of

boars may also have a sedative effect on the sows (McGlone *et al.*, 1986; McGlone and Morrow, 1988). Grandin and Bruning (1991) showed that the presence of mature boars significantly reduced fighting in newly mixed finisher pigs. Research investigating the use of boar to reduce aggression between group housed sows is in progress.

Dynamic versus static groups

The way in which groups of sows are mixed can influence the amount of aggression between sows. Dynamic group housing of sows give flexibility in terms of the number of sow mated each week, however they are difficult to mange since sows are being introduced to the group continuously and the group contains sows at all stages of gestation. Static group systems of sows are preferred i.e. managing the group as a batch i.e. sows bred, farrowed and weaned at the same time since dynamic grouping of sows i.e. continuously adding and removing sows leads to problems with aggression throughout gestation and may lead to poor reproductive performance and injury (Bokma and Kersjes, 1988; O'Connell *et al.*, 2002). Furthermore, returning sows at weaning to the same social group of the previous gestation, may ameliorate problems with aggression as sows may recognise sows form the previous gestation. Arey and Jamieson (1997,1998a,1998b) investigated the effect of time between removal and regrouping of sows on social recognition and showed that sows could recognise each other after being housed individually for six weeks, and that latency to aggress was reduced at mixing as sows became more familiar. However, static group systems are not feasible on some pork production enterprises therefore further research is required to investigate strategies for reducing aggression in dynamic groups. Durrell *et al.* (2003) showed that pre-mixing small groups of sows before their introduction into a larger dynamic group may reduce aggression between the total group. Harold Gonyou and the research team at the Prairie Swine Centre, Elstow are currently studying electronic sow feeder systems with groups of static and dynamic groups and are carrying out intensive observations on sow social behaviour and injuries (Gonyou, pers. comm.).

Replacement gilts should be introduced to the pen before the group of weaned sows. This management of gilts may give them pre-exposure to auditory and olfactory stimulation in their new pen (Kennedy and Broom, 1996). Industry evidence has shown that three to four days is sufficient time for the gilts to become accustomed with their surrounds, and may allow them to be less prone to aggression by sows when mixed.

Straw enrichment

The effect of straw on reducing sow aggression in groups of sows is equivocal, with some authors reporting benefits of supplementary feeding of straw (Durrel *et al.*, 1997) and straw bedding on reducing aggression (Meyer *et al.*, 1984), while Whittaker *et al.* (1999) reported sows bedded on straw may exhibit increased aggression. The addition of straw to group housing systems may have a two-fold effect in improving overall welfare and performance of the sow – feeding additional fibre (i.e. straw) may reduce boredom and hunger (promoting satiety), enrich the sows environment and encourages foraging (Fraser, 1975: Fraser *et al.*, 1991). This redirection of behaviour towards the straw may reduce aggression, antisocial and stereotypic behaviour (Beattie *et al.*, 1996; 2000a, 2000b), and thus may improve reproduction loss and poor welfare caused by aggression. The majority of studies in the literature compare the addition of straw to the sow's environment to a barren concrete floor/slatted system. Therefore, depending on the feeding system, pen space allowance, frequency of addition of straw material, this has varying effects on aggression. In some studies aggression is reduced with the addition of straw, however in other studies aggression has increased since sows are extremely competitive for the straw (since straw was only supplied to their environment in small amounts at daily intervals) (Anderson and Boe, 1999; Whittaker *et al.*, 1999). From the scientific literature it appears that *ad libitum* forage material should be provided to ensure sows do not fight over the limited resource. However, manure management becomes a significant problem when forage material is provided. Blockages and other disruptions of the flow in liquid effluent systems arise when bulky material enters the system. Other alternative group systems have been developed in Europe such as the 'Straw-flow' system, whereby gestating sows are provided with *ad libitum* straw in racks at the front of the pen and after pulling the straw from the racks it is either eaten or pushed towards the back of the sloping pen, from where a scraper systems removes the soiled bedding with the manure (i.e. liquid effluent systems are eliminated, and the solid material is physically removed). Obviously this incorporation of this housing design required major pen modifications of traditional housing system. Morrison and Buckingham (2004) showed that aggression and injuries were reduced at mixing in a 'straw-flow system' (21.02 *vs.* 5.08 aggressive bouts/sow/hour in the traditional group system and straw flow system, respectively) and furthermore injuries were reduced in the straw flow system at mixing. There were no significant differences in aggression and injuries at 110 days of gestation, which indicated that the groups of sows (nine sows/pen) were socially stable at that time. Unfortunately these changes in aggression did not confer improvements in farrowing rate or reproductive performance. However, in that experiment, sows were confirmed pregnant after five weeks of gestation stall housing, so there may be advantages in terms of reproductive performance of providing straw if sows are mixed earlier during gestation during the sensitive time of embryo implantation.

The review by Barnett *et al.* (2001) mentioned numerous other options that may be useful in reducing aggression between sows such as using masking odours and mood altering drugs, grouping sows after dark, providing *ad libitum* feed to sows, however the authors concluded that these methods may postpone aggression rather then reducing it.

Breeding management

Time of mixing in relation to mating

There are numerous options available to the pork producer in terms of when to group sows in relation to breeding. Sows can be grouped from weaning, immediately after breeding or five to six weeks into gestation, after pregnancy testing has been conducted. The sow is particularly vulnerable to injuries immediately in the post weaning period, since her body condition may be reduced from lactation period, her vulva may be sensitive as she returns to oestrus and if natural mating is being performed she is prone to injuries from the boar. Therefore, management strategies should be implemented in the post weaning period to ensure that the welfare of the sow is not at risk. Strategies may include individual housing and other methods to reduce aggression if group housed. Since this is a time for the sow to recoup body reserves lost during lactation the feeding regime should ensure that the sow has unrestricted access to feed.

A simple knowledge of the reproductive physiology of the female pig is required to understand the impacts that stressors imposed early on in gestation can have on reproductive performance. Once puberty is attained at 160 – 200 days of age the female pig will show signs of oestrus approximately every 21 days, which lasts for approximately 50-60 hours, during which time ovulation occurs, where on average 10-25 ova will be released. During this time the sow is either mated or artificially inseminated and fertilisation occurs. After conception the ova develop into embryos and they implant to the uterine wall. This process of embryo implantation begins 12 –13 days post conception and is usually complete by about week four of gestation. The gestation period for the female pigs is 110-119 days (Hughes and Varley, 1980). The general consensus in the scientific literature is that most embryonic death occurs within the first 30 days of gestation (Figure 1). The first 30 days of gestation is a sensitive time when a range of environmental, social, genetic, nutritional, hormonal and biochemical factors are interacting with each other, all having significant influences on conception rate and ultimate litter size (Ashworth and Pickard, 1998; Monogastric Research Centre, 2001). Group housing may exacerbate these issues since environmental and nutritional factors may be more difficult to control compared to individual stall housing.

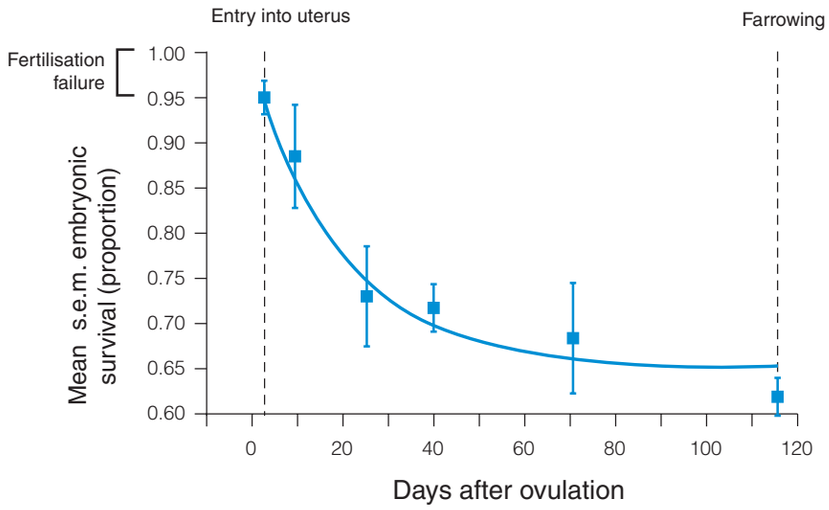


Figure 1. *Estimates of pre-natal mortality throughout gestation (from Ashworth and Pickard, 1998).*

Therefore, the safest option in terms of embryo survival is housing sows in gestation stalls for at least five weeks after mating to ensure that stressors associated with mixing (i.e. aggression, change in environment etc.) do not impact on embryo implantation (Barnett *et al.*, 2001; Harmon *et al.*, 2004). This time in gestation stalls also encompasses the time period for ultrasonic detection of pregnancy, which is feasible after day 30 of gestation. The Monogastric Research Centre (2001) reviewed several experiments that compared the reproductive performance of sows housed immediately post breeding compared to about five weeks after mating. These authors concluded that

there was sufficient evidence that grouping of sows earlier in gestation resulted in reduced litter size. The conception rates were similar between housing treatments in the research conducted by Fisker (1995) and Nielsen *et al.* (1997). The data from Schmidt *et al.* (1995) was the exception to the trend and showed that reproductive performance can be achieved if sows are mixed post mating. Grouping sows earlier in gestation involves much tighter management during this critical time of embryo implantation. From an industry perspective there are many producers that have had success with grouping sows at weaning, immediately post mating or within seven days of mating. Further research into the factors causing reproductive failure, management techniques to overcome problems and the most suitable time for grouping sows within the first week post mating needs to be conducted.

Table 2. Effect of housing system after mating on reproductive performance in sows (from Monogastric Research Centre, 2001).

	Conception rate		Litter size	
	Grouped after mating	Grouped approx. 5 weeks after mating	Grouped after mating	Grouped approx. 5 weeks after mating
Agribiz Engineering (1999)	-	-	10.8[a]	11.6[b]
Fisker (1995)	83.6	83.7	11.8c[c]	12.4[d]
Nielsen *et al.* (1997)	84	86	12.1	12.6
Schmidt *et al.* (1995)	78[a]	66[b]	9.8	9.2

Within rows, means with different letters (a, b) and (c, d) are significantly different at P<0.05 and P<0.001, respectively.

Heat detection in group housing systems is possible by bringing the boars either into the pen, or into an adjacent pen each day. Another option is to run the sows past the boar pen after feeding or whilst the sows are in feeding stalls (Gonyou, 2001, 2003).

Feeding management

Feeding systems in any group housing system should be designed to minimise competition for feed, avoid aggression and provide ease of management for the stockperson.

Floor feeding

Floor or ground feeding of sows (by either hand feeding or mechanical 'dump' feeders) is the simplest and cheapest feeding system available to producers, however this system results in a very competitive situation and can create social and nutritional stress for sows, especially those subordinate sows in the pen. Most industry success in using ground feeding has been with small groups of sows, in a static social grouping with sows of similar size and nutritional requirements (Gonyou, 2001, 2003).

Trickle feeding

Trickle feeding systems are designed as a slow release of feed during the feeding period to keep the attention of the more dominant sow at the feeding trough whilst allowing more timid sows the opportunity to eat at their own pace. Trickle feeding is currently being used by some producers to feed group-housed sows, however it appears to have most success in smaller groups of sows.

Individual feeding stalls

Providing individual sow feeding stalls either within the sow's home pen or in an external feeding area seems to be the most successful in both small and large herds of sows under Australian conditions (Barnett *et al.,* 1997; Morrison and Smits, 2005). Andersen *et al.* (1999) investigated the use of body and shoulder feeding partitions compared to no partitions between sows and found that there was less aggression using full body partitions compared to shoulder or nil partitions between sows. On wet feed however, aggression did not differ between shoulder and body partitions and the time spent feeding was similar. The authors concluded that simple feeding arrangements may be used using shoulder partition in wet feeding, however in dry feeding systems body partitions are recommended. There is a cost to the labour component of this feeding system as the sows are moved and usually locked into the feeding stall.

Electronic sow feeders (ESF)

The use of ESF's that can allocate a restricted ration to individuals allows the advantages of individual feeding provided by stalls and the advantages of group housing. Large group sizes reduce the capital cost of the feeder per sow, making these systems a commercial option. Electronic feeders have been in use overseas for many years and newer designs of ESF's and a better understanding of pen design to facilitate these feeding systems have been made (Broom *et al.*, 1995; Gonyou, 2001, 2003). Studies have shown a higher incidence of vulva biting in groups with ESF systems (Olsson *et al.*, 1992; Anil *et al.*, 2003) as the sows queue for access to the feeding stations. Other studies have shown that dynamic grouping of sows is feasible with ESF system, in groups of 15-70 sows, with low to moderate aggression, however these sow also had access to straw enrichment (Broom *et al.*, 1995; Gjein and Larssen, 1995; Hodgkiss *et al.*, 1998). Further research needs to be conducted on pen design and management of ESF systems, especially in larger groups of sows, more advanced ESF systems which reduce aggression within the feeding area and other low capital cost feeding systems which are feasible for group housing systems. Currently ESF systems are capable of providing accurate feed levels depending on the condition of the sow, changing the amount of feed required at a certain point in gestation, sorting and identification of certain sows.

Gonyou (2001) summarised the impacts on the sow of the five main gestation feeding systems (Table 3). This summary uses the concept of the 'Five Freedoms' for animal welfare which do not include physiological and biological measures of the welfare homeostatic approach. Obviously if freedom of movement is found to be fundamental to welfare of the sow then gestation stalls would be moved lower down the scale. It appears that ESF systems may be a viable option for feeding group housed sows, especially in large group situations.

Table 3. Ranking of gestation feeding systems for each of the five freedoms for animal welfare (higher scores are desirable) (from Gonyou, 2001).

	Floor feeding	Trickle feeding	Feeding stalls	Gestation stalls	Electronic sow feeders
Freedom from malnutrition	1	2	3	4.5	4.5
Freedom from discomfort	3.5	3.5	3.5	1	3.5
Freedom from pain and injury	1	2	3.5	5	3.5
Freedom from movement	3.5	3.5	3.5	1	3.5
Freedom from fear	1	2	3	5	4
Overall	10	13	16.5	16.5	19

Alternative housing options for group housing of sows

Deep litter, large group housing systems

Housing systems based on deep litter and large groups have been developed as an alternative to traditional group systems and are now an integral component of the Australian pig industry. It is estimated that progeny of about 30% of the Australian sow herd are housed in systems using deep litter and large groups for a significant part of the growing period (Payne *et al.*, 2000a, 2000b; Morrison *et al.*, 2003a, 2003b). Deep litter systems for gestating sows have been adopted by some producers in Australia, as low capital cost system. The first deep litter, large group housing system for pigs (The Ishigami System®) was developed in Japan in 1970. This system consisted of a polyvinyl-covered tunnel house with sawdust deep litter (Gadd, 1993). In 1985 straw-based deep litter systems were developed in Canada. The BioTech® deep litter system consisted of tubular steel framing with a polyviny cover, timber side barriers, earthen floors, and a concrete feeding pad at one end of the shed. The earthen floor was deep bedded and was cleaned out after each group of pigs (Connor *et al.*, 1997). Deep litter systems, similar in structure to the original systems developed in Canada (Clearspan® and EcoShelter®) have been developed in the Australian pig industry over the past ten years (Payne *et al.*, 2000a, 2000b). Deep litter systems are cheaper to establish**,** are perceived as being more 'welfare-friendly' for sows (offer the sows more room for locomotory and social behaviours) and are more sustainable environmentally as liquid effluent systems are not necessary. Furthermore, the housing of sows in large groups offers more total space to individual sows, which results in the sows having a greater degree of choice over their own microenvironment than sows in smaller groups in traditional group housing (Spoolder *et al.*, 1999).

Bedding management is a critical component of deep litter group housing systems as poor quality or insufficient bedding can lead to a wet and boggy environment, which can lead to sow health problems and leg injury (Harmon *et al.*, 2004). Pork producers should only consider deep litter, group housing an option if they have a consistent supply of good quality bedding throughout the year. Rice hulls and straw are commonly used in Australia as bedding materials. Morrison and Smits (2005) showed that approximately 100 kg of bedding (rice hulls or straw) is required per sow gestation in deep litter systems (assuming that the sows are in the deep litter housing system for 14 weeks), and this

amount of bedding was sufficient to maintain a dry area for all of the sows to lie on and a wet dunging area. The labour requirement to add bedding to the pens of sows was similar in terms of time required to add bedding over the whole gestation, however the straw bedding pens required straw bales to be added weekly, whereas the majority of the rice hull bedding was added at the start of gestation.

There is evidence that the consumption of deep-bedding (i.e. straw or rice hull bedding) litter ingestion of high fibre diets such as straw from deep-bedding may increase litter size and weaning weight (Ewan *et al.*, 1996). Morrison and Smits (2005) showed that bedding treatment (i.e. straw *vs.* rice hulls) had no effect on total number of pigs born alive, number of piglets born, number of pigs weaned or average piglet weight at weaning (Table 4).

Table 4. Mean reproductive performance and feed intake during lactation from mixed parity sows housed in pens of rice hull or straw deep bedding.

Treatment	No. sows mated	No. sows farrowed (%FR)	Number born alive	Total number born	Number weaned	Av. piglet weight at weaning (kg)	Av. daily lactation feed intake of sows (kg)
Rice hull	248	185(74.6)	10.7	11.6	9.0	7.8	6.5
Straw	230	182(79.1)	10.3	11.2	9.0	7.8	6.4
SEM	-	2.61	0.23	0.20	0.18	0.14	0.11
P value	-	0.241	0.428	0.390	0.908	0.980	0.714

% FR farrowing rate calculated as number of failed pregnancies/sows mated. Failed pregnancies resulted from abortion, returning to cycle, death of sow, abortion and culling.

There are also reports in the scientific literature that the consumption of high fibre diets during gestation result in higher lactation feed intake as a result of increased capacity for gut fill (Matte *et al.*, 1994; Farmer *et al.*, 1996). Morrison and Smits (2005) showed that deep litter straw bedding did not influence average daily feed intake during lactation. All other reported studies have been conducted on sows in conventional concrete/slatted systems with access to free-choice straw or have had the fibrous diet ingredients added directly to the diet. Furthermore, there is an enormous range of fibre types that have been studied (i.e. alfalfa meal, oat hulls, sugar beet pulp, soy bean hulls etc.). The study by Morrison and Smits (2005) is unique because sows in the 'control' housing treatment still had access to bedding material in the form of rice hulls and perhaps consumed this fibre source as well. On the other hand, free access to straw may not be suitable for changes in gut physiology and other fibre sources may be more suitable in causing these changes.

Morrison and Smits (2005) also showed that the most practical pen design for a deep litter system (eco-shelter) is a flat floor, with a minimal litter depth of 200 mm. Different pen configurations were tested in that experiment which included concrete laying platforms, which were thought to give the sows a cooler option for controlling their thermal comfort over summer. However, regardless of the season, sows preferred to lie on the deep litter bedding. This preference to lie on the deep litter bedding did not influence reproductive performance of the sows (i.e. increased temperature surrounding the sow from the bedding did not influence reproduction). In Australian conditions, the management of the environment is essential for sow comfort. The provision of misting spray-cooling systems along with natural ventilation should be provided to ensure sow comfort under Australian environmental conditions. Thermal comfort during winter is provided through the deep litter bedding, with straw and rice hull bedding having similar thermal properties (Morrison and Smits, 2005).

Morrison and Smits (2005) showed that both rice hull and straw bedding are suitable for housing gestating sows in deep litter systems and that there may be some additional benefits of adding straw (or perhaps some other substrate) at the point of mixing to reduce aggression in rice hull, deep litter systems. It was difficult to compare the results from the current experiment to those published in the scientific literature in regards to the influence of straw on aggression. The current study is unique in the sense that we compared two deep-bedded treatments, both of which provide an avenue for the sow to conduct exploratory rooting behaviours (i.e. an enriched environment). The rice hulls still may act as a mitigator in reducing aggression between sows, especially at mixing. Furthermore, the deep litter environment of increased pen space allowance and group size makes it difficult to compare studies conducted in more traditional, concrete environments. A decision on which bedding type is more appropriate is obviously dependent on availability, cost, quality of the bedding and labour availability. Furthermore, bedding increases the solid manure and requires different type of manure management compared to liquid effluent systems (Harmon *et al.*, 2004).

Few rigorous comparisons have been conducted between the various modifications of deep litter systems or between deep litter and conventional systems for gestating sows. Deep litter group housing systems are perceived as being welfare friendly, however there is limited data in the scientific literature on the welfare aspects of housing sows

in these systems. It is difficult to compare data from experiments assessing welfare and performance of sows in deep litter, group-housing systems because a variety of deep litter systems have been used. Systems vary in the type and amount of bedding, the number of sows per group, floor space allowance per pig, method of providing feed and water and the construction materials used to build the deep litter system.

Karlen *et al.* (2003) studied the relative welfare implications of the housing gestating sows in conventional gestation stalls or in large groups on deep litter. The results of that experiment indicated a number of housing treatment effects on sow welfare and reproductive performance. Early in gestation, there was evidence that sows in the deep litter system were under greater challenges in adapting to housing treatment than sows in conventional stalls. Sows in the deep litter treatment had higher salivary cortisol concentrations in Week 1 of gestation but not in Week 9 and had poorer farrowing rates (66% *vs.* 77% in deep litter and stalls respectively). In that experiment, correlations within the deep litter housed sows between behaviour, stress physiology and reproductive performance suggest that stressors, including social stressors, early post-mating may have be associated with reduced litter size. In contrast, late in gestation sows in gestation stalls were under greater challenges to adapt to housing treatment than sows in the deep litter system based on changes in the major cells of the immune system. Sows in stalls had higher neutrophil concentrations and percentages, lower lymphocyte percentages and higher neutrophils:lymphocyte ratios than sows in deep litter systems. Sows in the deep litter system had reduced feet and leg problems, better immune status and improved body condition, but reproductive performance was poorer.

A further experiment is currently being conducted to examine of the behaviour and physiology of sows, especially in the first five weeks post-insemination, to examine the events that may have a negative impact on the welfare and reproduction of sows in deep litter systems. The literature on rodents indicates that progesterone together with oestrogens, as occurs in gestation particularly after 16 days post-mating in the sow, markedly reduces aggression (de Jonge *et al.*, 1986; Kohlert and Meisel, 2001; Davis and Marler, 2003). These hormone levels are maintained through the majority of pregnancy. Therefore placing sows into group housing later in gestation (i.e. five weeks post mating) may reduce the level of aggression between sows seen around the time of mixing. Such an understanding may provide opportunities to improve the welfare and reproductive performance of sows in these large groups on deep litter and in conventional group housing.

Deep litter group housing systems are a viable option for pork producers who are looking for an alternative to gestation stalls or traditional group housing. As with all housing systems there are advantages and disadvantages of deep litter, group housing systems in terms of welfare and reproductive performance. Current research assessing the welfare of sows in deep litter systems needs to be completed before conclusions can be made on the welfare of sows in deep litter systems. Preliminary research to date suggests that there may be welfare implications at mixing in deep litter systems (through change in environment, aggression between sows etc.), however over time these welfare concerns may decrease and the advantages of improved body condition, reduced leg and feet problems and better immunological status of sows may be an advantage in terms of welfare. Pork producers should only consider this housing option for their gestating sows if they have a good consistent source of bedding, have a feeding system which is suitable for the group of sows and are aware of the need to understand the social dynamics within a group of sows. Further research needs to be conducted to assess ways of improving reproductive performance of sows housed in deep litter systems.

Pasture based group housing systems

Pasture based housing systems offer producers lower capital costs, but are extremely dependent on good stockperson skills, suitable climate and site selection. It is estimated that 5% of sows in Australia and 28% sows in New Zealand are housed outdoors (Barnett *et al.*, 2001). Recommendations for the locality for pasture based systems are relatively flat land, low rainfall (750 mm per year), although higher rainfall is suitable if there is adequate drainage in the soil. The management of sows in these more extensive systems is not dissimilar to that already discussed in this paper, however issue such as pen space requirement to reduce aggression are probably not applicable.

The importance of the stockperson in group housing systems

Regardless of the housing system used to accommodate the sows, the capacity and willingness of stockpeople to manage livestock and identify sick and injured sows will influence the welfare and reproductive performance of sows. Extensive research has shown that the behaviour of the stockpeople, especially behaviours that evoke a fear response in pigs, influence the welfare and reproductive performance of pigs (Hemsworth *et al.*, 1989;1986a). Furthermore, stockperson characteristics such as technical skills and knowledge, job satisfaction, job motivation and commitment will also influence the success of group housing systems (see Hemsworth and Coleman, 1998). From a facility design and management perspective, gestation stalls provide the unique opportunity to medicate and treat individual sows as

needed, while protecting them from other sows. Group housing systems should incorporate a separate pen or method of restraining individual sows for short periods of time to treat individual sows.

Conclusion

In conclusion, the move towards group housing of sows for a significant part of their gestation is on the horizon for many pork producers. Group housing is a viable option, and is already incorporated in many pork production enterprises, however further research needs to be conducted to assess the spatial requirements for gestating sows (i.e. what are the levels of pen space and group size that are amenable to good welfare and reproductive performance) and further management strategies to reduce aggression between group housed sows to ensure that maximum welfare and reproductive performance can be attained.

Acknowledgements

I would like to thank Australian Pork Limited and QAF Meat Industries Pty Ltd for providing me with the opportunity to continue investigating strategies for understanding and implementing management regimes to ensure the success of group housing for sows. I am also grateful for the continued collaboration with the Department of Primary Industries, Animal Welfare Centre, Werribee, especially my relationship with Dr Greg Cronin and Professor Paul Hemsworth who have been instrumental in the development of my career and are always willing for open discussion. Finally, I would like to thank pork producers in Australia and those I had the opportunity to work with during my employment as the Alternative Swine Production Systems Scientist at the University of Minnesota, USA for teaching me strategies for managing group housing gestating sows. There are many producers in the industry successfully using group-housing systems and many research ideas have been developed from working with these pork producers.

References

AGRIBIZ ENGINEERING. (1999). Welfare implications and recommendations for outdoor sows. Report to the Pig Research and Development Corporation, Canberra.

AL-RAWI, B. and CRAIG, J.V. (1975). Agonistic behaviour of caged chickens related to group size and area per bird. *Applied Animal Ethology* **2**: 69-80.

ANDERSON, I.L. and BOE, K.E. (1999). Straw bedding or concrete floor for loose-housed pregnant sows: consequences for aggression, production and physical health. *Acta Agriculturae Scandinavica Section: Animal Science* **49**:190-195.

ANIL, L., BHEND, K.M.G., BAIDOO, S.K., MORRISON, R.S. and DEEN, J.D. (2003). Comparison of injuries in sows housed in gestation stalls versus group pens with electronic sow feeders. *Journal American Veterinary Medicine Association* **223** (9):1334-1338.

AREY, D.S. and EDWARDS, S.A. (1998a). Factors influencing aggression between sows after mixing and the consequences for welfare and production. *Livestock Production Science* **56**:61-70.

AREY, D.S. and JAMIESON, W.G. (1998b). Social recognition in group-housed sows after removal and re-grouping. Pr*oceedings of the British Society of Animal Science,* British Society of Animal Science, Scarborough, United Kingdom, pp. 39.

AREY, D.S. and JAMIESON, W.G. (1997). Time course for the formation of a stable social hierarchy in group-housed sows. *Proceedings of the British Society of Animal Science*, British Society of Animal Science, Scarborough, United Kingdom, pp. 108.

ASHWORTH, C.J. and PICKARD, A.R. (1998). Embryo survival and prolificacy. In "Progress in Pig Science", pp. 303-325, eds. J. Wiseman, M.A. Varley, J.P. Chadwick. (Nottingham University Press: United Kingdom).

BARNETT, J.L. and HEMSWORTH, P.H. (2003). Science and its application in assessing the welfare of laying hens in the egg industry. *Australian Veterinary Journal* **81**:615-623.

BARNETT, J.L., HEMSWORTH, P.H., CRONIN, G.M., JONGMAN, E.C. and HUTSON, G.D. (2001). A review of the welfare issues for sows and piglets in relation to housing. *Australian Journal of Agricultural Research* **52**:1-28.

BARNETT, J.L. (1997). Modifying the design of group pens with individual feeding places affect the welfare of pigs. In 'Livestock Environment V' pp. 613-618, eds. R.W. Bottcher and S.J. Hoff (American Society of Agricultural Engineers: Michigan).

BARNETT, J.L., HEMSWORTH, P.H., CRONIN, G.M., NEWMAN, E.A., McCALLUM, T.H. and CHILTON, D. (1992). Effects of pen size, partial stalls and method of feeding on welfare-related behavioural and physiological responses of group-housed pigs. *Applied Animal Behaviour Science* **34**:207-220.

BARNETT, J.L., CRONIN, G.M., McCALLUM, T.H. and NEWMAN, E.A. (1993). Effects of pen size/shape and design on aggression when grouping unfamiliar adult pigs. *Applied Animal Behaviour Science* **36**:111-122.

BARNETT, J.L., HEMSWORTH, P.H., WINFIELD, C.G. and HANSEN, C. (1986). Effects of social environment on welfare status and sexual behaviour of female pigs. I. Effects of group size. *Applied Animal Behaviour Science* **16**:249-257.

BARNETT, J.L, CRONIN, G.M., WINFIELD, C.G. and DEWAR, A.M. (1984). The welfare of adult pigs: the effects of five housing treatments on the behaviour, plasma corticosteroids and injuries. *Applied Animal Behaviour Science* **12**:209-232.

BEATTIE, V.E., O'CONNELL, N.E., KILPATRICK, D.J. and MOSS, B.W. (2000a). Influence of environmental enrichment on welfare-related behavioural and physiological parameters in growing pigs. *Animal Science* **70**:443-450.

BEATTIE, V.E., O'CONNELL, N.E. and MOSS, B.W. (2000b). Influence of environmental enrichment on the behaviour, performance and meat quality of domestic pigs. *Livestock Production Science* **65**: 71-79.

BEATTIE, V.E., WALKER, N. and SNEDDON, I.A (1996). An investigation of the effect of environmental enrichment and space allowance on the behaviour and production of growing pigs. *Applied Animal Behaviour Science* **48**: 151-158.

BEILHARZ, R.G. and COX, D.F. (1967). Social dominance in swine. *Animal Behaviour* **15**: 117-122.

BILCIK, B. and KEELING, L.J. (2000). Relationship between feather pecking and ground pecking in laying hens and the effect of group size. *Applied Animal Behaviour Science* **68**(1):55-66.

BOKMA, S. and KERSJES, G.J.K. (1988). The introduction of pregnant sows in an established group. Proceeding of the International Congress of Applied Ethology of Farm Animals, Skara, Sweden, pp. 166-169.

BROOM, D.M., MENDL, M.T. and ZANELLA, A.J. (1995). A comparison of the welfare of sows in different housing conditions. *Animal Science* **61**:369-385.

CALE, W.H. (1979). Housing of breeding stock. In 'Australian Pig Manual', pp. 119-126, eds. J.A.A. Gardner and A.C. Dunkin (Australian Pig Industry Research Committee: Barton, ACT).

CONNOR, M.L., FULAWAKA, D.L., and ONISCHUK, L. (1997). Alternative low-cost group housing for pregnant sows. In 'Livestock Environment V' (vol 1). Proceedings of the Fifth International Symposium ASAE. St. Joseph, MI.

CRAIG, J.V. (1981). Domestic Animal Behaviour-causes and implications for animal care and management. New Jersy, United States of America: Prentice-Hall Inc.

CRAIG, J.V. and GUHL, A.M. (1969). Territorial behaviour and social interactions of pullets kept in large flocks. *Poultry Science* **48**:1622-1628.

CSERMELY, D. and NICOSIA, E. (1991). Maternal behaviour in sows of different social rank. *Journal of Ethology* **9**:83-89.

DAVIS, E.S. and MARLER, C.A. (2003). The progesterone challenge: steroid hormone changes following a simulated territorial intrusion in female Peromyscus californicus. *Hormones and Behaviour* **44**:185-198.

DE JONG, F.H., EERLAND, E.M. and VAN DE POLL, N.E. (1986). Sex specific interactions between aggressive and sexual behaviour in the rat: effects of testosterone and progesterone. *Hormones and Behaviour* **20**:432-444.

DURREL, J., SNEDDON, I.A. and BEATTIE, V.E. (1997). Effects of enrichment and floor type on behaviour of loose-housed dry sows. *Animal Welfare* **6**:297-308.

DURRELL, J.L., BEATTIE, V.E., SNEDDON, I.A. and KILPATRICK, D. (2003). Pre-mixing as a technique for facilitating subgroup formation and reducing sow aggression in large dynamic groups. *Applied Animal Behaviour Science* **84**:89-99.

ESTEVEZ, I. (1998). Broiler chickens: a tolerant social system? *Etologia* **5**:19-29.

EWAN, R.C., CRENSHAW, T.D., CROMWELL, G.L., EASTER, R.A., NELSSEN, J.L., MILLER, E.R., PETTIGREW, J.E. and VEUM, T.L. (1996). Effect of addition of fibre to gestation diets on reproductive performance of sows. *Journal of Animal Science* **74** (Suppl. 1), 190.

EWBANK, R. (1976). Social hierarchy in suckling pigs and fattening pigs: a review. *Livestock Production Science* **3**:363-372.

EWBANK, R. (1974). Individual recognition and the dominance hierarchy in the domestic pig. The role of sight. *Animal Behaviour* **22**:473-480.

EWBANK, R. (1969). Social behaviour and intensive animal production. *The Veterinary Record* **85**:183-186.

EWING, S.A., LAY D.C. and D VON BORELL, E. (1999). 'Farm Animal Well-Being' (Prentice Hall: New Jersey).

EU (2001). Council Directive 2001/88/EC of 23 October 2001 amending Directive 91/630/EEC laying down minimum standards for the protection of pigs. *Official Journal of the European Communities* **L 316**: 1-4 and 36-38.

FARMER, C., ROBERT, S. and MATTE, J.J. (1996). Lactation performance of sows fed a bulky diet during gestation and receiving growth hormone-releasing factor during lactation. *Journal of Animal Science* **74**:1298-1306.

FISKER, B.N. (1995). Strategy for introduction of group-fed gestating sows to loose housing systems. Danish Applied Research Scheme. Danske Slagterier, Copenhagen, Denmark.

FRASER, D. (1975). The effect of straw on the behaviour of sows in tether stalls. *Animal Production* **21**:59-68.

FRASER, A.F. and BROOM, D.M. (1998). 'Farm Animal Behaviour and Welfare'(Bailliere Tindall: London).

FRASER, D. and RUSHEN, J. (1987). Aggressive behaviour. In "Farm Animal Behaviour", pp. 285-305, ed. E.O Price (Saunders Publishing Company: Philadelphia).

FRASER, D., PHILLIPS, P., THOMPSON, B.K. and TENNESSEN, T. (1991). Effect of straw on the behaviour of growing pigs. *Applied Animal Behaviour Science.* **30**:307-318.

GADD, J. 1993. Tunnel housing of pigs. In Livestock Environment IV. Fourth International symposium. American Society of Agricultural Engineers, Michigan pp. 1040-1048.

GJEIN, H. and LARSSEN, R.B. (1995). Housing of pregnant sows in loose and confined systems-a field study 1. vulva and body lesions, culling reasons and production results. *Acta veterinaria scandinavia* **36** (2):185-200.

GONYOU, H. (2001). Group housing of sows: the potential for the future. *Proceedings of the Focus on the future conference, Red Deer*, Alberta, Canada, pp. 29-35.

GONYOU, H. (2003). Group housing: alternatives systems, alternative management. *Advances in Pork Production* **14**:101-107.

GRANDIN, T. and BRUNING, J. (1991). Boar presence reduces fighting in mixed slaughter-weight pigs. *Applied Animal Behaviour Science* **33**:273-276.

HAFEZ, E.S.E. and SIGNORET, J.P. (1969). The behaviour of swine. In 'The Behaviour of Domestic Animals' ed. E.S.E. Hafez (Bailliere, Tindall and Cassell: London).

HAFEZ, E.S.E. (1975). 'The Behaviour of Domestic Animals'. (The Williams and Wilkins Company: Baltimore).

HARMON, J.D., HONEYMAN, M.S., KLIEBENSTEIN, J.B., RICHARD, T. and ZULOVICH, J.M. (2004). Hoop barns for gestating swine. MidWest Plan Service. Revised edition. Iowa State University, Iowa.

HART, B.L. (1985). 'The Behaviour of Domestic Animals' (W.H.Freeman and Company: New York, USA).

HEMSWORTH, P.H., BARNETT, J.L and HANSEN, C. (1986a). The influence of handling by humans on the behaviour, growth and corticosteroids of male and female pigs. *Applied Animal Behaviour Scienc.* **15**:303-314.

HEMSWORTH. P.H.,BARNETT, J.L., HANSEN, C and WINFIELD, C.G. (1986b). Effects of social environment on welfare status and sexual behaviour of female pigs. II. Effects of space allowance. *Applied Animal Behaviour Science* **16**:259-267.

HEMSWORTH, P.H., BARNETT, J.L, COLEMAN, G.J. and HANSEN, C. (1989). A study of the relationships between the attitudinal and behavioural profiles of stockpersons and the level of fear of humans and reproductive performance of commercial pigs. *Applied Animal Behaviour Science* **23**:301-314.

HEMSWORTH, P.H. and COLEMAN, G.J. (1998). 'Human-Livestock Interactions. The Stockperson and the Productivity and Welfare of intensively Farmed Animals' (CABI Publising: Oxon).

HODGKISS, N.J., EDDISON, J.C, BROOKS, P.H and BUGG, P. (1998). Assessment of the injuries sustained by pregnant sows housed in groups using electronic feeders. *The Veterinary Record* **143**:604-607.

HOUPT, K.A. (1991). 'Domestic Animal Behavior' (Iowa State University Press: Ames, Iowa).

HSIA, L.C. (1984). The effect of group size and feeding space on social and feeding behaviour of pigs. *Proceedings of the International Congress on Applied Ethology in Farm Animals*, Kiel, Germany:

HUGHES. P.E. and VARLEY, M.A. (1980). 'Reproduction in the pig' (Butterworths: London).

HUGHES, B.O., CARMICHAEL, N.L., WALKER, A.W. and GRIGOR, P.N (1997). Low incidence of aggression in large flocks of laying hens. *Applied Animal Behaviour Science* **154**:215-234.

JENSEN, P. (1980). An ethogram of social interaction patterns in group-housed dry sows. *Applied Animal Ethology*. **6**: 341-350.

JENSEN, P. (1982). An analysis of agonistic interaction patterns in group-housed dry-sows-aggressive regulation through an avoidance order. *Applied Animal Ethology* **9**:47.

KARLEN, G.A., HEMSWORTH, P.H., GONYOU, H.W., FABREGA, E., STROM, D. and SMITS, R.J. (2005). The welfare of gestating sows in conventional stalls and large groups on deep litter. Report to Australian Pork Limited. Project number 1825. In press.

KENNEDY, M.J. and BROOM, D.M. (1996). Factors modulating aggression received by pigs mixed individually into groups. *Proceedings of the 30th International Congress of the International Society for Applied Ethology*, Guelph, Canada, pp. 52.

KOHLERT, J.G. and MEISEL, R.L. (2001). Inhibition of aggression by progesterone and its metabolites in female syrian hamsters. *Aggressive Behaviour* **27**:372-381.

KROSNIUNAS, E.H. (1979). Social facilitation and foraging behaviour of the feral pig (*Sus scrofa*) on Santa-Cruz Island, California. M.A. Thesis. Zoology Department, University of California, Davis.

MANNING, A. and DAWKINS, M.S. (1992). 'An Introduction to Animal Behaviour' (The Bath Press: Avon, Great Britian).

MATTE, J.J., ROBERT, S., GIRARD, C.L., FARMER, C. and MARTINEAU, G.P. (1994). Effect of bulky diets based on wheat bran or oat hulls on reproductive performance of sows during their first two parities. *Journal of Animal Science* **72**:1754-1760.

MENDL, M., ZANELLA, A.J. and BROOM, D.M. (1992). Physiological and reproductive correlates of behavioural strategies in female domestic pigs. *Animal Behaviour* **44**:1107-1121.

MEYER, B., HAGELSO, A.M., HANSEN, L.L. and JEPPERSEN, L.L. (1984). Effect of environment and rank order on agnostic behaviour in pigs. *Proceedings of the International Congress on Applied Ethology in Farm Animals*, Kiel, Germany, pp. 162-165.

MCBRIDE, G. (1963). The teat order and communication in young pigs. *Animal Behaviour* **11**:53-56.

MCBRIDE, G., JAMES, J.W. and HODGENS, N. (1964). Social behaviour of domestic animals. IV Growing pigs. *Animal Production* **6**:129-139.

MCGLONE. J.J, VON BORELL, H., DEEN, J, JOHNSON, A.K., LEVIS, D.G., MEUNIER-SALAUN, M., MORROW, J. REEVES, D. SALAK-JOHNSON, J.L and SUNBERG, P.L. (2004). Review: Compilation of the scientific literature comparing housing systems for gestating sows and gilts using measures of physiology, behaviour, performance, and health. *The Professional Animal Scientist* **20**:105-117.

MCGLONE, J.J. and CURTIS, S.E. (1985). Behaviour and performance of weanling pigs in pens equipped with hide areas. *Journal of Animal Science* **60**:20-24.

MCGLONE, J.J. and MORROW, J.C. (1988). Reduction in pig antagonistic behaviour by andosterone. *Journal of Animal Science* **66**:880-884.

MCGLONE, J.J., STANSBURY, W.F. and TRIBBLE, L.F. (1986). Aerosolized 5∝-androst-16-in-3-one reduced agonistic behaviour and temporarily improved growth performance of growing pigs. *Journal of Animal Science* **63**:679-684.

MEESE, G. B. and EWBANK, R. (1972). A note on instability of the dominance hierarchy and variations in level of aggression within groups of fattening pigs. *Animal Production* **14**: 359-362.

MINISTRIES VAN JUSTITIE EN VAN LNV (1998). Varkensbesluit. Staatsblad van het Koninkrijk der Nederlanden **473**:1-8.

MINISTRIES VAN JUSTITIE EN VAN LNV (2003). Varkensbesluit. Staatsblad van het Koninkrijk der Nederlanden **184:**1-6.

MONOGASTRIC RESEARCH CENTRE. (2001). Effects of dry sow stall use for a limited period after mating: a literature review for the New Zealand Pork Industry. Institute of Food, Nutrition and Human Health, Massey University, Palmerston North. pp 1-10.

MORRISON, R.S., HEMSWORTH, P.H., CAMPBELL, R.G. and CRONIN, G.M. (2003a). The social and feeding behaviour of growing pigs in deep litter, group housing systems. *Applied Animal Behaviour Science* **82**:173-188.

MORRISON, R.S, HEMSWORTH, P.H., CAMPBELL, R.G. and CRONIN, G.M. (2003b). The effect of restricting pen space and feeder availability on the behaviour and growth performance of entire male growing pigs in a deep litter, group housing system. *Applied Animal Behaviour Science* **83**:163-176.

MORRISON, R.S and BUCKINGHAM, J. (2004). The welfare and performance of gestating sows in a straw flow pen compared to conventional group pen. Report to QAF Meat Industries Ltd., Corowa, Australia.

MORRISON, R.S. and SMITS, R.J. (2005). A quantitative comparison of litter system for gestating sow ecosystems. Report to Australian Pork Limited.

NEHRING, A. (1981). One answer to the confinement pig problem. *British Veterinary Journal* **2**:256-259.

NICHOLSON, R.I., McGLONE, J.J. and REID, L.N. (1993). Quantification of stress in sows: comparison of individual housing versus social penning. *Journal of Animal Science* **71** (Suppl**.** 1): 112.

NICOL, C.J., KNOWLES, N.G., PARKMAN, T.G. and WILKINS, L.J. (1999). Differential effects of increased stocking density, mediated by increased flock size, on feather pecking and aggression in laying hens. *Applied Animal Behaviour Science* **65**:137-152.

NIELSEN, N.P. FISKER, B.N., HANSEN, L.U. and RUBY, V. (1997). Strategies for mixing sows in small static groups. Danish Applied Pig Research Scheme. Danske Alagterier, Copenhagen, Denmark.

O'CONNELL, N.E., BEATTIE, V.E. and MOSS, B.W. (2002). Influence of social status on the welfare of sows in static and dynamic groups. *Animal Welfare* **12**:239-249.

OLSSON, A., SVENDSEN, J., ANDERSSON, M., RANTZER, D. and LENKENS, P. (1992). Group housing systems for sows. I. Electronic dry sow feeding on Swedish farms. An evaluation of the use of the system in practice. *Swedish Journal of Agricultural Research* **22**:153.

PAGEL, M. and DAWKINS, M.S. (1997). Peck orders and group sizes in laying hens: 'future contracts' for non aggression. *Behavioural Processes* **40**:13-25.

PATERSON, R., POINTON, A. and CARGILL, C. (1997). Sow wastage in the Australian pig herd-degree, cost and prevention. Report to the Pig Research and Development Corporation, Canberra.

PAYNE, H., MULLAN, B., and TREZONA, M. (2000a). Review of alternative housing systems for pigs-PART A. Report to Pig Research and Development Corporation, Canberra.

PAYNE, H., NICHOLLS, R. and DAVIS, M. (2000b). Straw-based group housing for sows. PRDC Project DAW 38/1252 (Pig Research and Development Corporation, Canberra).

PETHERICK, J.C. and BLACKSHAW, J.K. (1987). A review of the factors influencing the aggressive and agonistic behaviour of the domestic pig. *Australian Journal of Experimental Agriculture* **27**:605-611.

PETHERICK, J.C, BODERO, D.A.V. and BLACKSHAW, J.K. (1987) The use of partial barriers along the feed trough in group housing systems for non lactating sows. *Farm Building and Engineering* **4**:32-36.

PUPPE, B. (1996). Social dominance and rank relationships in domestic pigs: a critical review. *Berl Munch Tierarztl Wochenschr.* **109(11-12)**: 457-464.

RANDOLPH, J.H., CROMWELL, G.L., STAHLY, T.S. and KRATZER, D.D. (1981). Effects of group size and space allowance on performance and behaviour of swine. *Journal of Animal Science* **53**(4): 922-927.

SCHMIDT, W.E., STEVENSON, J.S. and DAVIS, D.L. (1985). Reproductive traits of sows penned individually or in groups until 35 days after breeding. *Journal of Animal Science* **60**: 755-759.

SIGNORET, J.P., BALDWIN, B.A., FRASER, D. and HAFEZ, E.S.E. (1975). The Behaviour of Swine. In 'The Behaviour of Domestic Animals'. ed E.S.E. Hafez. (Bailliere, Tindall and Cassell: London).

SPOOLDER, H.A.M., EDWARDS, S.A. and CORNING, S. (1999). Effects of group size and feeder space allowance on welfare in finishing pigs. *Animal Science* **69**:481-489.

STOLBA, A. and WOOD-GUSH, D.G.M. (1989). The behaviour of pigs in a semi-natural environment. *Animal Production* **48**:419-425.

STRICKLIN, W.R. and MENCH, J.A. (1987). Social Organisation. In 'Farm Animal Behaviour', pp. 307-322, ed. E.O. Price (Maple-Vail Book Manufacturing Group. York, PA).

VAN PUTTEN, G. and VAN DE BURGWAL, J.A. (1990). Vulva biting in group-housed sows: a preliminary report. *Applied Animal Behaviour Science* **26**:181-186.

VAN PUTTEN, G. and DAMMERS, J. (1976). A comparative study of the well-being of piglets reared conventionally and in cages. *Applied Animal Ethology* **2**:339-356.

VARLEY, M. and STEDMAN, R. (1994). Stress and reproduction. In 'Principles of Pig Science', pp. 277-296, eds D.J.A. Cole, J. Wiseman and M.A.Varley (Nottinghamn University Press: Nottingham).

WENG, R.C, EDWARDS, S.A. and ENGLISH, P.R. (1998). Behaviour, social interactions and lesion scores of group-housed sows in relation to floor space allowance. *Applied Animal Behaviour Science* **59**:307-316.

WHITTAKER, X., SPOOLDER, H.A.M., EDWARDS, S.A., LAWRENCE A.B. and CORNING, S. (1999). Effects of straw bedding and high fibre diets on the behaviour of floor fed and group-housed sows. *Applied Animal Behaviour Science* **63**:25-39.

Stall dimensions for sows

J.L. Barnett and G.M. Cronin

Department of Primary Industries, Werribee, Vic. 3030.

Housing sows in stalls is one of the most controversial welfare issues in pig production. In this factorial experiment we examined two factors of stall dimensions - width (0.6 m and 0.75 m; centre-to-centre measurements) and length (2.0 m, 2.2 m and 2.4 m; free space). Both treatments had stall divisions of horizontal bars. A tether treatment provided a negative control. Each treatment used four multiparous sows in a bank of similar sized stalls (two adjacent experimental sows in the centre, flanked by non-experimental sows). The experimental unit was a pair of central sows and treatments were located at random in the shed and the position of treatments was randomised between each of four replicates. Sows were placed in treatments within one week of mating. Behaviour measurements were recorded on video after seven weeks. Physiological measurements were made following a recovery period of four days, in sows catheterised under full surgical anaesthesia after eight weeks. Profiles of total and free cortisol concentrations and the cortisol response one hour after an IM injection of 50 IU of synthetic adrenocorticotrophic hormone (ACTH) and immune status (percent increase in ear margin thickness 24 hours after an injection of 500 μg leucoagglutin {a red kidney bean extract} in 500 μL saline) were measured. Data were analysed for treatment effects by analysis of variance using Genstat 5 with replicates in time used as a blocking effect.

Significant treatment effects and their P values are presented in Table 1 while data for kneeling are presented within the text. In the wider stalls, sows stood for a longer proportion of time and lay for a shorter proportion of time and descended to kneeling later after feeding. Sows stood for less time in the 2.0 m than the 2.2 m long stall. The frequency of kneeling was higher in tethered sows during two hours of observations in the afternoon (mean values were 1.9 vs. 0.9LSD=0.44; P<0.05). The maximum angle that pigs could turn their heads was higher in wider stalls and tethers and highest in the 2.0 m long stalls. Total and/or free cortisol concentrations and the response to ACTH were lower in the narrower and tether stalls and lowest in the 2.2 m long stalls. The immune response was lower in tethers and the wider stalls and highest in 2.2 m long stalls.

Table 1. Treatment effects on behaviour and physiology variables (mean values).

Factor →	Width (m)		Length (m)			Stall	Tether stall	$lsd_{max)}$ (P=0.05)
Variable Treatment→	0.6	0.75	2.0	2.2	2.4			
Standing (%)[1]	73.7^{a}	86.4^{b}	75.8	81.0	82.8	79.9	81.2	14.48
Lying (%)[1]	23.9^{a}	12.8^{b}	24.0	14.8	16.2	18.3	18.6	14.90
Latency to kneel[2] (min)	86.5^{a}	101.4^{b}	88.8	95.7	97.3	93.9	93.6	17.90
Turn angle[3] (°)	38^{p}	50^{q}	48^{b}	42^{a}	41^{a}	44^{y}	36^{x}	7.5
Maximum angle[3] (°)	47^{x}	59^{y}	61^{y}	50^{x}	49^{x}	53^{b}	44^{a}	12.2
Total[4]	18.4^{p}	25.5^{q}	22.5	20.8	22.6	22.0^{x}	36.6^{y}	6.09
Free[4]	2.3^{x}	3.0^{y}	2.7^{b}	2.3^{a}	2.9^{b}	2.6^{x}	3.5^{y}	0.48
Post-ACTH[4,5]	2.80^{a}	3.02^{b}	2.95^{q}	2.76^{p}	3.02^{q}	2.91^{p}	3.13^{q}	0.183
Cell mediated immunity[6]	108.4^{b}	91.2^{a}	91.1^{pqa}	119.1^{qb}	89.1^{pa}	99.8^{b}	81.7^{a}	19.56

[1]% of time in a 2 h session from time of feeding; [2]after feeding, on the way to lying; [3]angle that a sow turned her head relative to a line parallel to a stall's side panel; [4]cortisol concentrations (nMol); [5]% increase (log_{10} value); [6]% increase in skin thickness; within factors, means with different superscripts differ ^{ab}P<0.05, ^{pq}P<0.01 and ^{xy}P<0.001.

The findings for stall width from this experiment contrast with recommendations based on allometric measurements for a sow to lie without her udder protruding into the adjacent stall (0.72 m; McGlone *et al.*, 2004). In the present experiment welfare was better in a 0.6 m than a 0.75 m wide stall, in which there was a chronic stress response indicated by the sows' lower total and free cortisol concentrations, a reduced response to ACTH and an increased immune response. The data for stall length are not as clear; free cortisol concentrations and the response to ACTH were lower in the 2.2 m long stalls, but the immune response was increased. The data suggest stalls should be 0.6 m wide and possibly 2.2 m long to minimise stress and its consequences.

Supported in part by Australian Pork Limited.

References

MCGLONE, J.J., VINES, B., RUDINE, A.C. and DUBOIS, P. (2004). *Journal of Animal Science* **82:**2421-2427.

Is a stall length of 2.7 metres too long for sows?

G.M. Cronin, S.S. Borg and J.L. Barnett

Department of Primary Industries, Werribee, Vic. 3030.

The need to provide industry and the community with science-based recommendations on the housing and husbandry of breeding sows during gestation is an important issue in the welfare debate regarding the use of sow stalls. Conventional sow stalls (2.0 m long and 0.6 m wide) are considered too small for modern sows (McGlone *et al*., 2004) but if stalls are lengthened will there also be welfare implications? In this experiment we investigated whether increasing the sow stall length to 2.7 m, by using the aisle space behind the stalls, would affect sow behaviour and welfare more than conventional stalls. The stalls used had horizontal bars in the side panels. There were six replicates and 48 sows per treatment. The experimental unit was the cohort of eight sows per treatment per replicate, situated in adjacent stalls within the same dry sow shed. On the day of their second artificial insemination, commercial crossbred sows (Large White Landrace) were allotted to treatments at random within parity number strata and placed in the gestation stall treatments. Parity number ranged from two to eight, although most sows (92.7%) were in the range of parity two to five. Sows were fed once daily at about 0715 h and were provided with water *ad libitum* in their trough about 1.5 h after feeding. Measurements were taken over two days at four and nine weeks in treatment. Sow behaviour was recorded over two hours using the instantaneous scan sampling technique with a three-minute interval between observations, by an observer who walked quietly along the aisle in front of the sows. Observations commenced when all experimental sows had received their meal. Saliva was collected from four sows per treatment in each replicate between 1300 h and 1500 h, when the sows were 'at rest', using Salivettes® (Sarstedt, Germany). Salivettes were placed immediately on ice and saliva was later extracted by centrifugation and stored frozen at –22º C until assayed. Data were analysed for treatment effects using one-way analysis of variance blocked on replicate, or the Chi-squared test for proportional data, in Genstat 7.2 (Rothamsted Experimental Station).

The main posture recorded for sows during the two hours following feeding was standing, which accounted for about 80% of the observations. There were no differences ($P>0.05$) due to length of stall on time spent standing, lying or kneeling at both four and nine weeks. Sitting posture, which was recorded at a relatively low frequency, occurred more frequently in 2.0 m than 2.7 m stalls at four weeks (1.8% *vs.* 0.3% of observations; $P<0.05$, sed=0.54) but not at nine weeks (2.0% *vs.* 0.4%; $P>0.05$). In addition, the proportion of sows recorded to sit during the two-hour observations at four weeks was greater ($P<0.01$) in the 2.0 m than 2.7 m stalls (22.1% *vs.* 7.1% of sows; $\chi^2_1=7.56$). The difference was not significant at nine weeks (21.4% *vs.* 11.4% of sows; $\chi^2_1=1.3$, $P>0.05$). Although sows in the 2.0 m stall spent less time eating their meal at four weeks than sows in the 2.7 m stalls (15.1 *vs.* 16.4% of observation time; $P<0.05$, sed=0.53), there were no differences in time spent licking the trough, floor or bars or in aggression or resting behaviour at either four or nine weeks. Sows in the 2.0 m stalls had lower ($P<0.05$) free cortisol concentrations at both week four ($\log_{10}$ transformed values were 0.632 and 0.815, respectively; sed=0.0567) and week nine (0.560 and 0.724, respectively; sed=0.0447) than sows in the 2.7 m stalls.

There was a potential for improved sow health from a lower risk of urino-genital tract infections in the 2.7 m stalls due to both fewer sows sitting and less time spent sitting (Bertschinger, 1999). Nevertheless, the incidence of this behaviour was low and the risk of infection depends on shed hygiene but this was not measured in the current experiment. However, elevated saliva cortisol concentrations of sows in the 2.7 m stalls suggests these animals were more stressed. The higher cortisol concentrations of pigs in the 2.7 m stalls agree with Barnett and Cronin (2005), in which sows were more stressed in 2.4 m stalls than in 2.0 m stalls. Interpreting these data, particularly the behaviour data, in terms of welfare is difficult and suggests we need to be cautious when making decisions about how much stalls should be lengthened. In addition, the results highlight our lack of understanding of the subtle consequences of changing housing design on sow behaviour, particularly on social interactions between adjacent sows in stalls and their associated physiological responses.

Supported in part by Australian Pork Limited.

References

BARNETT, J.L. and CRONIN, G.M. (2005). In 'Manipulating Pig Production X', p.218. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

BERTSCHINGER, H.U. (1999). In 'Diseases of Swine', 8th Edition, pp. 464-468; Eds. B.E. Straw, S. D'Allaire, W.L. Mengeling and D.J. Taylor; Iowa State University Press, Ames Iowa.

McGLONE, J.J., VINES, B., RUDINE, A.C. AND DUBOIS, P. (2004). *Journal of Animal Science* **82:** 2421-2427.

Australian Pork

Behaviour and stress physiology of gestating sows in conventional stalls and in large groups on deep litter

G.A. Karlen, P.H. Hemsworth and H.W. Gonyou*

Department of Primary Industries, Vic. 3030. *Prairie Swine Centre, Canada.

Confining breeding sows and gilts is a controversial issue in livestock production (Barnett *et al.*, 2001). Individual housing in stalls restricts the opportunity of sows to exercise and also limits the ability of animals to interact socially and with their environment. However, group housing raises welfare issues concerning space and aggression. The hypothesis tested in this experiment was that sow welfare and reproductive performance would be similar for sows housed in conventional stalls (stalls) or in large groups on deep litter ('eco-sheds') during gestation. About 85 recently mated sows (Landrace x Large White) were allocated to each treatment each week over eight weeks. Sows in eco-sheds were mixed one to four days after mating. Measurements were done on 18 focal sows, selected at random within parity strata (six sows in each of parities 1, 2 and 3+) in each of the eight replicate groups in each treatment. Incidence of aggression within the eco-shed treatment was observed during weeks one and nine of gestation. Saliva samples to assess cortisol concentrations were collected in weeks one and nine of gestation and blood samples to assess haematology were collected at week 15 of gestation. Data were analysed by analysis of variance (using group means values) while proportional data were analysed using the Chi-square test.

There was a strong tendency for higher saliva cortisol concentrations (P=0.06) in the eco-shed treatment at week one, suggesting greater stress during this time - which may be related to the increased aggression and a higher rate of returns to oestrus in this treatment (P<0.01). Sows in the stall treatment had a higher percentage of neutrophils (P<0.05) and a lower percentage of lymphocytes (P<0.05) than sows in the eco-shed treatment at week 15, suggesting higher stress in stalled sows during week 15 of gestation. There was a strong tendency for a higher reproductive failure (P=0.06) in the eco-shed treatment. While sows housed in stalls had a higher farrowing rate (P<0.001), they weaned fewer piglets per litter (P<0.01). However, sows in the stall treatment tended to wean more piglets per 100 mated sows (P=0.07).

Table 1: Comparison of stress physiology and reproductive parameters in groups of sows housed for 15 weeks of gestation in either Stalls or Ecosheds (N=8 per treatment).

Measurement	Stalls	Eco-sheds	s.ed.	χ^2_1 value
Cortisol concentrations at week 1 of gestation (nM)	4.03	6.29	1.037	
Cortisol concentrations at week 9 of gestation (nM)	3.81	4.02	0.304	
Aggression at week 1 (bouts per sow)		1.4		
Aggression at week 9 (bouts per sow)		0.6		
Neutrophils at week 15 of gestation (%)	46.0[b]	41.0[a]	2.090	
Lymphocytes at week 15 of gestation (%)	41.6[a]	46.5[b]	1.833	
Return to oestrus (%)	7.4[a]	13.2[b]		4.659
Reproductive failure (%)	14.5[a]	27.3[b]		3.865
Farrowing rate (%)	76.8[d]	66.0[c]		26.218
Piglets weaned per sow	8.3[a]	9.0[b]	0.161	
Piglets weaned per 100 mated sows	601.1	562.2	0.454	

Within rows, values with different superscripts differ ([a, b] P<0.05; [c,d] P<0.001)

The hypothesis tested in this experiment was not supported. The results suggested that sows in the stall treatment were less stressed during early gestation and more stressed later in gestation than sows in the eco-shed treatment during the same time periods. Increased stress during early gestation for the sows in the eco-shed treatment, particularly before implantation of embryos, may have been responsible for the higher rate of returns to oestrus recorded for the group-housed sows. However, the group-housed sows that remained pregnant had larger litters at weaning. In conclusion, this experiment highlights the importance of reducing stress in early gestation to improve reproductive performance of sows housed in groups.

Funded by DPI Victoria and APL. Supported in part by QAF Meat Industries Pty. Ltd.

BARNETT, J. L., HEMSWORTH P. H, CRONIN G. M., JONGMAN E. C. AND HUTSON G. D. (2001). *Australian Journal of Agricultural Research* **52:**1-28.

Australian Pork

Pigs reared in deep litter have an altered pattern of growth and tissue deposition

D. Suster*, D.J. Henman**, D.J. Cadogan*** and F.R. Dunshea*

*Department of Primary Industries, Vic. 3030 **QAF Meat Industries, NSW 2646. ***Feedworks, Eagle Farm, Qld. 4009.

Deep litter systems offer the advantages of a low capital cost of production, perception of a welfare-friendly system and reduced pollution load. However, such systems may also increase feed consumption, reduce feed use efficiency and increase fatness. These problems may cause altered feeding patterns and animal stress during the finisher period as space per pig diminishes. This study was carried out to identify any negative effects of deep litter systems on carcass composition and to determine at what growth stage these problems might occur. Three successive replicates of 360 Large White × Landrace pigs (180 males and 180 females) weaned at an average of 21 days into two housing systems were used. Within each replicate 120 pigs of each sex were weaned into a deep litter (rice hulls and straw) with the remaining 60 pigs of each sex weaned into groups of 20 in conventional pens with solid concrete flooring. All pigs were located in the same airspace with a stocking density of 0.65 m^2/pig. However, for the first three weeks conventionally housed animals were reared in raised weaner cots (within their allocated pen) with supplemental heat lamps (0.13 m^2/pig). All animals had *ad libitum* access to pelleted commercial diets at all times. Growth rate and feed intake were monitored. Dual energy X-ray absorptiometry (Suster *et al*., 2003) was used to measure body composition in focus pigs (12 per deep litter pen and four per conventional pen) allocated randomly at various time points. Data were analysed by ANOVA with pen as the experimental unit.

Table 1. Effect of deep litter housing and sex on body composition of pigs from 26 to 152 days of age.

Housing (H)	Deep Litter		Conventional			Significance		
Sex(S)	Male	Female	Male	Female	s.e.d.[a]	H	S	H × S
Empty body lean (kg)								
Day 26	6.94	6.98	6.87	6.78	0.24	0.41	0.88	0.71
Day 68	20.2	21.9	19.7	20.4	0.59	0.014	0.005	0.25
Day 110	42.9	43.1	42.4	41.8	1.11	0.27	0.78	0.60
Day 152	66.8	63.2	67.8	64.1	1.48	0.38	<0.001	0.94
Empty body fat (kg)								
Day 26	0.53	0.54	0.57	0.51	0.044	0.91	0.45	0.28
Day 68	2.46	2.62	2.14	2.48	0.14	0.020	0.012	0.36
Day 110	7.43	9.85	7.39	8.30	0.44	0.013	<0.001	0.017
Day 152	14.7	18.5	15.4	18.9	0.97	0.40	<0.001	0.87

[a]s.e.d for interaction between sex and treatment.

In the first three weeks after weaning, conventionally housed pigs ate more (199 *vs.* 170 g/d, P=0.009) and grew 35 g/day faster than deep litter pigs (189 *vs.* 154 g/d, P=0.035). However, feed intake was higher in pigs housed in the deep litter system between 58 and 85 days of age (1466 *vs.* 1360, P=0.005). Over the final growth phase, between 114 and 149 days of age, feed intake was higher in pigs that were conventionally housed (2726 *vs.* 2551 g/d, P=0.016). The lean tissue content of pigs housed under deep litter conditions was higher than those housed in the conventional system at day 68, but from then on the differences in lean tissue disappeared (Table 1). During the grower period (day 68-110), the fat content of animals reared in deep litter was higher than in those reared conventionally, however this difference disappeared at day 152. Females contained more fat than males from 68 days age onwards and contained 3.7 kg more fat and were 3.6 kg less lean at the end of the experiment. An interaction (P=0.017) was observed at day 110 where females had a lower lipid content when reared in the conventional system, an effect that was not observed in males. Hot carcass weight and back fat were not affected by either housing system or sex. Dressing percentage was not affected by housing, however females dressed out at a higher percentage then males (78.8 *vs.* 76.7%, P<0.001), which was expected due to reproductive organs. These data suggest that under the same relatively high stocking density there was little difference in the final carcass composition and overall growth performance of pigs housed under either conventional deep litter conditions. However, there were quite clear differences in the pattern of growth and tissue deposition with the deep litter pigs initially eating more and growing faster before slowing down.

References

SUSTER, D., LEURY, B.J., OSTROWSKA, E., BUTLER, K.L., KERTON, D.J., WARK, J.D. and DUNSHEA, F.R. (2003). *Livestock Production Science* **84**:231-242.

Acetyl-CoA carboxylase activity and belly fat of gilts housed conventionally or on deep litter

M. Trezona, D.W. Pethick*, J.R. Pluske*, B.P. Mullan, F.R. Dunshea** and D.N. D'Souza

Department of Agriculture, South Perth, WA 6983. * Murdoch University, Murdoch, WA 6150. **Department of Primary industries, Werribee, Vic. 3030.

Lipid accretion, or the storage of fat, is characterised by the formation of triglycerides, primarily from free fatty acids (FFA) and glycerol-3-phosphate. In pigs, FFA formation occurs when glucose is metabolised via glycolysis and the citrate cleavage pathway to form acetyl-CoA in the cytoplasm where it can act as a substrate for acetyl-CoA carboxylase, the rate-limiting step of lipogenesis (Dunshea and D'Souza 2003). Trezona *et al.* (2005) reported that pigs housed conventionally had more belly fat than pigs housed on deep litter. In this study, we hypothesised that acetyl-CoA carboxylase activity in the belly fat of pigs housed conventionally would be higher than in deep litter pigs.

One hundred and fifty two female pigs were stratified by weight at three weeks of age and allocated to housing treatments, conventional (C) or deep litter (DL). Pigs housed conventionally were housed within an insulated building in groups of ten in eight commercial weaner and grower-finisher pens. In the deep litter housing system, 72 pigs were housed as one group in a deep-litter shelter that was bedded with straw. Pigs were phase-fed the same commercial cereal-based diets. Pigs were allocated a predetermined slaughter date based on age. Eight pigs from each housing treatment were slaughtered at a commercial abattoir at: 7, 13, 24 and 35 weeks of age. Belly fat samples were collected into liquid nitrogen from the hot carcass and were stored at -80^{0}C until assayed for acetyl-CoA carboxylase activity (Harris *et al.,* 1993), which was expressed relative to the supernatant protein. Data were analysed by two-way ANOVA using Genstat v6.

Table 1. Activity of acetyl-CoA carboxylase in belly fat (expressed as nmol of radioactive bicarbonate incorporated per min per mg protein).

			C*	DL	lsd[1]	P-value
		Treatment	4.04[a]	1.93[b]	1.65	0.014
Age (weeks)	7	Treatment*Age	9.68[a]	4.13[b]	2.35	0.051
	13		5.11[a]	2.01[b]		
	24		0.92	1.10		
	35		0.46	0.50		

[1]lsd: least significant difference; [a,b] rows with different superscripts are significantly different (P≤ 0.05). *See text for treatment explanation.

Within each age there were no treatment differences in live weight, carcass weight or P2 back fat (P>0.05). There was a significant effect of age on acetyl-CoA carboxylase activity. As age increased acetyl-CoA carboxylase activity decreased (P<0.001). For 7, 13, 24 and 35 weeks the activity was 6.90, 3.56, 1.01, 0.48 nmol of radioactive bicarbonate incorporated/min/mg protein respectively. There was also an overall effect of treatment where acetyl-CoA carboxylase activity was higher in the belly fat of pigs housed conventionally (P=0.014). The interaction between treatment and age on enzyme activity was significant at 7 and 13 weeks of age (P=0.051). These results show that in young pigs the activity of acetyl-CoA carboxylase in belly fat is more than two-fold higher than in pigs housed conventionally. The higher level of enzyme activity indicates that lipogenesis would be occurring at a higher rate in the belly of young, conventionally housed pigs than pigs of the same age housed on deep litter. This difference in lipogenesis would result in greater fat deposition in the belly during the early growing phase and the higher level of fat is likely to remain significant through to finishing and slaughter. Results reported by Trezona *et al.* (2005) support these findings, where belly fat was higher in the carcasses of 24-week-old pigs housed conventionally than in pigs raised on deep litter. This would suggest that the activity of acetyl-CoA carboxylase in the belly fat of young pigs is a good indicator of belly fat deposition and possibly belly composition at slaughter.

References

DUNSHEA, F.R. and D'SOUZA, D.N. (2003). In 'Manipulating Pig Production IX' pp.127-150, ed. J.E. Paterson. (Australasian Pig Science Association: Werribee).

HARRIS, D.M., DUNSHEA, F.R., BAUMAN, D.E., BOYD, R.D., WANG, S.-Y., JOHNSON, P.A. and CLARKE, S.D. (1993). *Journal of Animal Science* **71**:3293-3300.

TREZONA, M., MULLAN, B.P., PLUSKE, J.R., PETHICK, D.W. and D'SOUZA, D.N. (2005). In 'Manipulating Pig Production X' p. 274. ed J.E. Paterson (Australasian Pig Science Association: Werribee).

Modelling weaner performance in deep litter systems with climate control via internal kennels

B.K. Vaschina, G. Taylor* and G. Roese*

CSIRO Livestock Industries, Armidale, NSW 2350. *Department of Primary Industries, Calala, NSW 2340.

Variations in shed temperature and wind speed are the major cause of poor performance when weaners are housed on deep litter. Hyun *et al.* (1998) subjected pigs to combinations of temperature, stocking rate and social group stressors and found that multiple concurrent stressors depressed growth performance in pigs by 31%. As the number of stressors increased, the gain to feed ratio decreased linearly. Similarly, Quiniou *et al.* (2001) demonstrated the negative impact of cold exposure on group-housed animals.

The aim of this study was to use Auspig™ (1989) computer simulations to predict the performance of weaners subjected to varying wind speeds and temperatures. The data used to parameterise the simulation were obtained from a piggery on the New England tablelands of northern NSW. Pigs weaned at 14, 21 and 28 days were exposed to temperature ranges from -9^{0}C in winter to 36^{0}C in summer. Wind speeds ranged from 2.3 m/s to 3.1 m/s. The temperatures and wind speeds were obtained from the Bureau of Meteorology weather station located near the piggery and are representative of climatic conditions experienced within the open nature of the buildings at the piggery. Table 1 presents the average temperature and wind speed data used in the simulation.

Table 1. Climate data used for Auspig™ simulation weaner study.

	Summer Max	Summer Min	Winter Max	Winter Min
Temperatures (^{0}C)	36.20	4.40	22.80	-9.30
	Summer	Autumn	Winter	Spring
Wind Speed (m/s)	2.67	2.31	2.83	3.13

In the model, temperature and wind speed were controlled within the deep litter shed using an internal kennel arrangement with a steel roof covered with about 60 cm of straw. The weaner pigs were free to move between the kennel and the main shed area. To simulate the heat retained by the building/kennel structure, the temperature variation was reduced to predicted minimums of 20^{0}C in summer (thermo-neutral) and 10^{0}C in winter and the predicted wind speed was halved to about 1.4 m/s. This resulted in a predicted improved average daily gain (ADG) from one day after weaning to four weeks after weaning (Table 2).

Table 2. Effect of implementing temperature and wind speed controls on average daily gain (ADG) for weaned pigs from weaning to four weeks after weaning.

Age (days)	Sex	Wean weight (kg)	ADG g/day (Standard deep litter shed with no kennels)	ADG g/day (inclusion of internal kennels)
14	Female	4.0	329	410
14	Male	4.0	336	426
21	Female	5.5	394	456
21	Male	5.5	414	471
28	Female	7.5	451	468
28	Male	7.5	472	484

In this model, providing kennels within the main deep litter shed enabled higher temperatures to be maintained and reduced wind speed. This resulted in the predicted average daily growth of pigs weaned at 14, 21 and 28 days, being 25%, 14.7% and 3.1% higher in deep litter sheds with internal kennels.

References

CSIRO Livestock Industries, (1989). 'Auspig A Computer Decision Support System'.

HYUN, Y., ELLIS, M., RISKOWSKI, G. and JOHNSON, R.W. (1998). *Journal of Animal Science* **76:** 721 -727.

QUINIOU, N., NOBLET, J., van MILGEN, J. and DUBOIS, S. (2001). *Animimal Research* **50:**325-333.

Piglets suckling anterior teats during lactation grow faster but then show a reduced rate of growth

J.C. Kim*, B.P. Mullan**, J. Callesen***, R.H. Wilson**** and J.R. Pluske*

*Murdoch University, Murdoch, WA 6155; ** Department of Agriculture, South Perth, WA 6983; *** Danish Bacon and Meat Council, Copenhagen, Denmark; ****Wandalup Farms, Mandurah, WA 6210.

The anterior teats of sows contain more protein and DNA (Kim *et al*., 2000) and produce up to 200% more milk than the posterior teats (Algers *et al*., 1990). As a consequence, piglets suckling from anterior teats drink more milk and are generally heavier at weaning (Pluske *et al*., 1996; Kim *et al*., 2000). However, the subsequent post-weaning performance of piglets that suckled anterior or posterior teats has not been reported. The purpose of this study was to determine the performance of pigs before and after weaning based on their teat (suckling) order during lactation.

Six Large White x Landrace sows and their litters were used. Litter size was equalised at 10 to 11 piglets per sow within 36 hours of farrowing. On day 12 of lactation a creep feed was introduced to all litters. On days 24 and 27 of lactation, the teat (suckling) order of piglets was assessed during two to three consecutive sucklings. Piglets were categorised as suckling from either the anterior teats (teats 1-4) or posterior teats (teats 5-7) (Pluske *et al*., 1996). The numbers of piglets in each group were 37 and 19 respectively. Piglet live weight and growth rates were determined on several occasions between birth and 59 days of age. Weaning occurred at 31 days of age. The ANOVA analysis of Statview 5.0 for Windows (SAS Inc.) was used for statistical analysis.

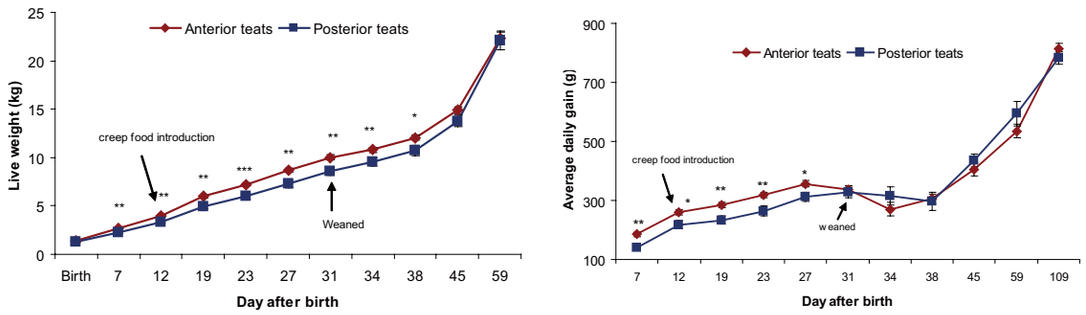


Figure 1. *Pre- and post-weaning performance of piglets suckling from different teats on the udder.*

The mean birth weight of piglets suckling from anterior and posterior teats did not differ (1.38 and 1.26 kg, respectively). Piglets suckling the anterior teats grew 40 g/day more than piglets sucking the posterior teats up to weaning (278 g *vs*. 237 g/day, respectively; P<0.01). After weaning, the growth rate of piglets suckling anterior teats decreased by 67 g/day to day 34, but started to recover from day 38. In contrast, piglets suckling posterior teats maintained their growth rate after weaning. Between weaning and day 59, piglets that drank from posterior teats grew 40 g/day faster than piglets that suckled from the anterior teats (P>0.05). Consequently the live weight difference at weaning between piglets suckling from anterior and posterior teats disappeared by day 59. On day 109, live weights of anterior and posterior suckling piglets were not different (63 and 61 kg, respectively, P=0.323). Piglets drinking milk from the anterior teats grew faster to weaning because anterior teats produce more milk (Pluske *et al*., 1996), but the benefit of being a heavier piglet at weaning was not related to subsequent growth rate after weaning. Curiously, the consumption of more milk in lactation may limit the intake of creep feed and this in turn can exacerbate the after weaning growth check. However, these differences disappear over time.

The Australian Research Council, Danish Bacon and Meat Council and Wandalup Farms are thanked for their support with this work.

References

ALGERS, B., JENSEN, P. and STEINWALL, L. (1990). *Applied Animal BehaviourScience* **26**:143-155.

KIM, S.W., HURLEY, W.L., HAN, I.K. and EASTER, R.A. (2000). *Journal of Animal Science* **78**:131-1318.

PLUSKE, J.R. and WILLIAMS, I.H. (1996). *Australian Journal of Agricultural Research* **47**:513-523.

The effect of an exogenous hydrophylic emulsifier on weaner pig performance

C.J. Soede

Nutrifeed, Veghel, The Netherlands.

Fats and oils are used to increase the energy density of weaner diets to compensate for the low feed intake of weaned piglets. However, fats and oils differ widely in their digestible energy values due to differences in chemical structure, inclusion rate in the diet and composition of other dietary components. In addition to these feed factors, piglet-specific factors like health status and age also contribute to the digestible energy value of dietary fat. For example, Cera *et al.* (1988) demonstrated that saturated animal fats have a lower digestibility than unsaturated vegetable oils during the post-weaning period. The lower digestibility of saturated fats in young pigs is associated with an impaired micelle quality due to the molecular structure of the fatty acids and low bile salt levels (Freeman, 1984). Including exogenous emulsifiers, particularly those with hydrophilic properties, in the diet, may support physiological bile salt levels and improve micelle quality. It is hypothesised that adding an exogenous, hydrophilic emulsifier (Premulac, Nutrifeed) will improve the performance of weaned piglets through an increased digestibility of dietary fat.

Eighty eight crossbred piglets (Pietrain x Dutch Landrace) were allocated into two different dietary treatments, each treatment consisting of four replicate pens. The piglets were weaned at 25 days of age with an average weaning weight of 7.3 ± 0.02 kg (mean ± SEM). Littermates were divided equally across pens based on gender (barrows and gilts) and weaning weight. Pens from each weight class were assigned randomly to one of the treatment groups consisting of 1) a basal diet containing 6% total fat including 3.6% added palm oil and 0.4% added coconut oil (control); 2) the basal diet + 200 ppm emulsifier. The basal diet was formulated to contain 14.9 MJ DE/kg and 12 g/kg lysine. The diets were provided during the entire experimental period of 35 days. Statistical analysis was performed using the GLM procedure of SPSS 12.0 (SPSS Inc.).

Table 1. Effects of an exogenous, hydrophilic emulsifier[1] on the performance of weaned piglets fed diets with 3.6% palm oil.

	Control	Emulsifier	SEM	p-value
Weight, 0 d (kg)	7.3	7.2	0.02	0.45
Weight, 14 d (kg)	10.4	10.6	0.34	0.46
Weight, 35 d (kg)	21.8[a]	22.7[b]	0.58	0.03
Daily gain, 0-14 d (g)	228	239	25	0.47
Daily gain, 14-35 d (g)	538[a]	576[b]	25	0.04
Daily gain, 0-35 d (g)	412[a]	441[b]	19	0.04
Daily feed intake, 0-14 d (g)	269	288	16	0.07
Daily feed intake, 14-35 d (g)	771	794	25	0.16
Daily feed intake, 0-35 d (g)	570	592	15	0.06
Feed conversion ratio, 0-14 d	1.18	1.21	0.09	0.69
Feed conversion ratio, 14-35 d	1.43	1.38	0.05	0.09
Feed conversion ratio, 0-35 d	1.38	1.34	0.05	0.21

[1] Premulac, Nutrifeed, The Netherlands
[ab] Within a row, means with different superscripts differ (P<0.05)

Including the emulsifier significantly improved piglet live weight and weight gain during the experimental period. This effect was most pronounced in the period from 14 to 35 days after weaning. A tendency (P=0.06) towards a more favourable feed intake was established over the entire experimental period when the emulsifier was included. This study indicates that including a hydrophilic emulsifier in diets for weaned piglets improves piglet performance, probably due to an increased digestibility of dietary fat.

References

CERA, K.R., MAHAN, D.C. and REINHART, G.A. (1988). *Journal of Animal Science* **66**:1430-1437.

FREEMAN, C.P. (1984). In 'Fats in animal nutrition', pp. 105-122, ed J. Wiseman. (Butterwords, London).

Dietary whey protein concentrate partially alleviates the decrease in bone density observed with high protein diets

M.L. Cox*, J. M. Ferrari*,**, E. Ostrowska*, C. Ketses*,**, M. Muralitharan**, B.G. Tatham*, I. McCauley*, P.J. Eason*, D.J. Kerton* and F.R. Dunshea*,**

*National Centre of Excellence in Functional Foods, Department of Primary Industries, Werribee, Vic. 3030, **Deakin University, Geelong, Vic. 3217.

There has been recent interest in using high protein diets to control food intake and body weight (Anderson and Moore 2004). However, some epidemiological studies have associated long-term consumption of diets rich in animal protein with increased hip fracture rates in the elderly (Abelow *et al.,* 1996), although it is unclear whether this is true for both animal and plant protein sources and in healthy, growing individuals. Therefore, the present study was carried out to examine the effect of dietary protein (DP) source and level on bone density and deposition using growing pigs as a model for rapid growth.

Large White x Landrace pigs (24 boars and 24 gilts) were weaned into individual pens at 21 days of age and allocated one of six dietary treatments formulated to be adequate for all nutrients and to provide either 100, 140 or 180% of DP requirements for the weaner, grower and finisher stages. The major sources of DP were whey protein isolate (WPI (NaturaPro MG2460, MG Nutritionals, Brunswick, Victoria) or soy protein isolate (SPI) (Profarm 974, ADM, Palm Beach, Queensland). The WPI contained 46, 30, and 8% β-lactalbumin, GMP and α-lactoglobulin, respectively. Synthetic amino acids were added to the lowest weaner SPI diet to ensure that no amino acids were limiting. Diets were formulated to contain 1.05 and 0.55%, 0.82 and 0.45% and 0.72 and 0.40% Ca and P for the three growth phases. Bone mineral deposition (BMD) and bone density (BD) were determined periodically by dual energy X-ray absorptiometry (Suster *et al.,* 2003).

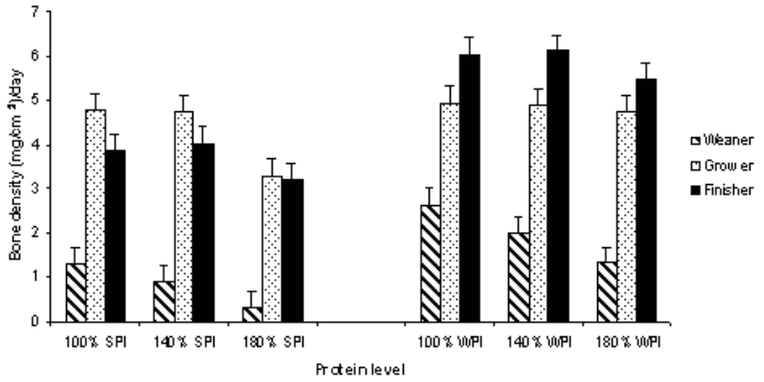


Figure 1. *Effect of stage of growth and amount and type of dietary protein on change in bone density.*

Boars had higher BMD rates than gilts (22.1 *vs.* 20.5 g/d, P<0.001) while pigs consuming WPI had higher BMD rates than those consuming SPI diets (23.0 *vs.* 20.5 g/d, P<0.001). Similarly, boars had a greater change in BD that gilts (3.73 *vs.* 4.32 mg/cm^2/day, P=0.009) and the change in BD was greater in pigs consuming WPI than those consuming WPI (4.69 *vs.* 3.36 mg/cm^2/day, P<0.001; Figure 1). There was a linear decrease in both BMD rate and change in BD with increasing DP content. These data suggest that increasing DP reduces BMD the rate of change in BD during rapid growth but that this can be partially alleviated by WPI.

References

ABELOW, B.J., HOLFORD, T.R. and INSOGNA, K.L. (1992). *Calcified Tissue International* **50**: 14–18.

ANDERSON, G.H., and MOORE, S.E. (2004). *Journal of Nutrition* **134**: 974S–979S.

SUSTER, D., LEURY, B.J., OSTROWSKA, E., BUTLER, K.L., KERTON, D.J., WARK, J.D. and DUNSHEA, F.R. (2003). *Livestock Production Science* **84**: 231-242.

Versatile enzymes increase ileal and faecal digestibility in piglets fed on corn and soybean meal

Y.G. Liu*, X. H. Gu** and X. S. Meng**

*Adisseo Asia-Pacific Pty Ltd, Singapore 507028 **Chinese Academy of Agricultural Sciences, Beijing 100094, China.

The digestive capacity of weaned piglets is restricted due to limited secretion of digestive enzymes and the dietary presence of cellulose, hemi-cellulose, pectin and other non-starch polysaccharides (NSPs). In recent years the benefits of microbial enzymes have been recognised for increasing digestion of NSPs (Liu *et al.*, 2003). In this study we tested the hypothesis that a xylanase-based enzyme preparation from a non-GMO fungus *Penicillium funiculosum* would enhance the digestion of NSPs and nutrients and in doing do increase the digestible energy of the diet for piglets.

The enzyme preparation (Rovabio Excel AP 10%) contains endo-1,4-xylanase 2,200 U/g, endo-1,3(4)-β-glucanase 200 AGL/g plus cellulase and other auxiliary activities. A 4×4 Latin square design, four barrows at average initial weight 12.0 kg were each fitted with a simple T-cannula on the distal ileum and fed with four test diets: 1) Positive Control (PC) diet: corn 61.31%, soybean meal 32.84%, vegetable oil 1.90% plus premixes, DE 13.81 MJ/kg and lysine 1.12%; 2) PC + Enzyme (P+E); 3) Negative Control (NC), diet: corn 64.45%, soybean meal 29.08%, wheat bran 2.00%, vegetable oil 0.28% plus premixes, DE 13.43 MJ/kg and lysine 1.10%; 4) NC + Enzyme (N+E). Each test phase lasted eight days with a four-day adaptation and a four-day sample collection. Ileal digesta and faecal samples were freeze-dried and chromium, proximate nutrients and amino acids were determined.

At the faecal level, enzyme supplementation significantly improved apparent digestibility of dry matter, crude protein, crude fat and ash. The enzyme increased energy digestibility from 83.0-87.0% for the positive control diet and from 83.1% to 87.4% for the negative control diet, with DE increased by 0.586 MJ/kg diet ($P<0.001$). The results are in line with those from our previous study (Liu, 2004).

At the ileal level the inclusion of the enzyme markedly increased the apparent ileal digestibility of dry matter, crude protein, crude fat and ash (Table 1). For the PC and the NC diets, the digestibility of energy was increased by 5.95 and 7.58 percentage units respectively, while the digestibility of the indispensable amino acids was enhanced by 6.93 and 5.84 percentage units respectively.

Table 1. Influence of enzyme addition on the apparent ileal digestibility (%) of nutrients in piglets.

	1. P. C.	2. PC + E	3. N. C.	4. NC+ E	SEM	P
Dry matter	61.93^{d}	68.94^{ab}	63.08^{cd}	71.86^{a}	1.33	<0.001
Gross energy	66.95^{d}	72.90^{ab}	68.23^{cd}	75.81^{a}	1.10	<0.001
Crude protein	69.36^{c}	76.39^{a}	70.94^{bc}	76.21^{a}	1.25	<0.001
Crude fat	47.15^{c}	51.33^{bc}	52.63^{abc}	62.16^{a}	3.42	0.012
Crude ash	12.43^{d}	29.25^{ab}	23.77^{bc}	30.98^{a}	2.21	<0.001
Lysine	82.01^{b}	87.68^{a}	82.63^{b}	87.98^{a}	0.97	<0.001
Methionine	79.01^{c}	86.83^{a}	81.93^{bc}	88.41^{a}	1.32	<0.001
Threonine	63.10^{c}	72.30^{a}	66.59^{bc}	72.94^{a}	1.69	0.001
Indispensable AA	75.22^{b}	82.15^{a}	76.71^{ab}	82.55^{a}	2.07	0.061

*Values in the same row not carrying the same alphabet indicate $P<0.05$. * See text for explanation of treatments.

We conclude that adding a versatile enzyme increases digestibility of proximate nutrients and amino acids in piglets fed on corn-soybean meal diets.

Reference

GERAERT, P. A., MAISONNIER, S., LIU, Y. G. and DALIBARD, P. (2003). *Proceedings of Australia Poultry Science Symposium* Sydney 2003. pp. 123-126.

LIU Y.G. (2004). Asian Pork Magazine, Apr/May, 2004, pp. 30-32.

Impact of daily fluctuations in food intake on the performance and body composition of female pigs

B.P. Mullan, S.M. McCullough, R.R. Nicholls, D.N. D'Souza and M. Trezona

Department of Agriculture, South Perth, WA 6151.

Variation in performance and carcass quality is a major problem for the pig industry (Payne *et al*., 1999) and Edwards (1999) has suggested that some of the variation may be related to daily fluctuations in nutrient intake caused by, for example, competition for feeder space and blocked feeders. Edwards (1999) suggested that when feed intake is sub-optimal maximum protein deposition rates are not achieved and a greater proportion of nutrients are lost to maintenance. However, when intake is above the optimal level then protein deposition is maximised, but excess nutrients are directed to fat deposition. The AUSPIG simulation model was used to predict the impact of restricting feed intake to grower pigs to 36% of normal intake (0.76 *vs.* 2.09 kg/d), followed by a day of engorgement (3.42 kg/d), with this pattern being repeated on a daily basis (Edwards, 1999). Fat deposition was predicted to increase from 194 to 236 g/d while protein deposition was predicted to decline from 155 to 90 g/d. The hypothesis for this experiment was, therefore, that the fat to lean ratio of pigs at slaughter would be higher for animals that had experienced short-term fluctuations in nutrient intake.

Sixty female pigs (Landrace x Large White) from a commercial herd with high health status were housed individually and allocated at 30 kg live weight to one of four treatments. Pigs were either: 1) fed *ad libitum* throughout (High); 2) fed at 85% (Low) of their partner on the High treatment; 3) restricted to 70% on one day and then fed at 100% the following day, with this pattern repeated throughout the experiment (Daily) or; 4) restricted to 70% for three consecutive days and then fed at 100% for the next three days (3-Daily). For the purpose of calculating feed restriction levels, pigs were treated as sub-groups of four. Diet composition was the same for all treatments and the diet was changed at 50 and 70 kg live weight. Pigs were slaughtered when they reached 104 kg live weight and one side of the carcass of all pigs was collected and analysed for fat and lean content via dual energy X-ray absorptiometry (DXA) and the protein and fat content of the viscera measured chemically.

Table 1. Performance and carcass composition of female pigs whose feed intake was either High or Low, or varied daily or every three days (N = 15).

Treatment	High	Low	Daily	3-Daily	s.e.d.	**P - value**
ADG (g)	943[a1]	825[b]	839[b]	828[b]	32.4	0.001
Food intake (kg/d)	2.90[a]	2.51[b]	2.49[b]	2.47[b]	0.066	0.001
Feed : Gain (kg:kg)	3.15	3.06	3.00	3.01	0.591	0.406
P2 (mm)	14.9[a]	11.9[b]	12.7[b]	12.9[b]	0.975	0.021
Fat:Lean ratio						
Shoulder	0.32	0.30	0.29	0.32	0.024	0.645
Loin	0.24	0.22	0.23	0.20	0.032	0.611
Belly	0.33	0.30	0.30	0.27	0.048	0.715
Ham	0.24	0.23	0.20	0.21	0.026	0.458

[1] Means not followed by a similar superscript differ significantly

Average daily gain (ADG) reflected the differences in food intake and the depth of subcutaneous fat (P2) was significantly higher for pigs on the High treatment than the other three treatments (Table 1). Fluctuating feed intake (Low *vs.* Daily or 3-Daily) did not increase the fat to lean ratio of the carcass or affect the fat content of the viscera significantly. The results of this experiment therefore do not support the hypothesis that short-term fluctuations in food intake would increase the fat:lean ratio of the carcass. However, it is possible that fluctuations in food intake are larger in commercial piggeries than those used in this experiment.

References

EDWARDS, A.C. (1999). *Recent Advances in Animal Nutrition in Australia* **12**:137-144.

PAYNE, H.G., MULLAN, B.P., TREZONA, M. FREY, B. (1999). In 'Manipulating Pig Production VII', pp. 13-26, ed P.D. Cranwell. (Australasian Pig Science Association: Werribee).

Reducing nitrogen and phosphorus excretion through diet manipulation

T.J. Wester, P.C.H. Morel and A.N. Shilton

*Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.

Reducing environmental pollution while maintaining profitability is one of the major challenges facing the pig industry today. The aim of this study was to demonstrate that matching diet composition to pig genotype would decrease nitrogen (N) and phosphorus (P) excretion, as suggested by previous growth model simulation studies (Morel and Wood, 2005).

Sixty-four, 30 kg live weight, Large White x Landrace pigs (32 female and 32 male) were kept in four pens of eight females and four pens of eight males and fed *ad libitum* until slaughter at 85 kg live weight. Feed intake, live weight and total effluent output were recorded weekly. The experiment was carried out in two runs. Following the first experimental run, diets were modified based on the results of model simulations to reduce N and P excretion without affecting pig production performance. The diets fed to the pigs before (R1) and after the modifications (R2) are presented in Table 1. The grower diet was fed for five weeks and the finisher diet was then fed until average pig weight per pen reached 85 kg. For the second experimental run (R2), energy content of the diets was increased and crude protein decreased but ingredients with a higher digestibility were used to provide the same amount of ileal digestible essential amino acid (i.e. the amount of protein fed to the pigs decreased across runs but the amount able to be used by the pigs remained the same). Gross phosphorus content was also decreased during R2.

Table 1. Diet composition used during the first (R1) and second (R2) experimental runs.

	Grower R1	Grower R2	Finisher R1	Finisher R2
Digestible energy (MJ/kg)	13.45	13.75	12.75	12.85
Digestible lysine (g/kg)	8.7	8.95	7.7	7.7
Crude protein (g/kg)	186.7	169.1	168.1	153.4
Digestible essential amino acids (%)	73.5	79.6	75	79.1
Phosphorus (g/kg)	9.2	6.3	9.2	6.3
Cost per tonne (NZ$)	557	566	534	540

Pigs in R2 returned more profit and excreted 3% less N and 27% less P than pigs in R1 (Table 2). Nitrogen excretion in both runs was about 37% of N intake and this is the minimal value achievable under commercial conditions in New Zealand when an improved pig genotype is fed properly formulated diets. For comparison, N excretion on a commercial farm was as high as 48.9% when feed was not matched to the particular genotype (Shilton *et al.*, 2003). It is concluded that matching diet composition to the pig genotype can reduce N and P excretion of pigs.

Table 2. Economic and effluent parameters per kg live weight of pig produced during the first (R1) and second (R2) experimental runs.

	R1*	R2	R2/R1
Cost (ct/kg)	348	347	100
Return (ct/kg)	393	387	98
Profit (ct/kg)	44.5	40.4	91
N intake (g/kg)	65.7	63.8	97
P Intake (g/kg)	21.5	15.7	73
N Excretion (g/kg)	24.81	24.06	97
P Excretion (g/kg)	8.54	6.25	73

*See text for explanation of R1 and R2

Supported by New Zealand MAF Sustainable Farming Fund and the New Zealand Pork Industry Board.

References

SHILTON, A.N., MOREL, P.C.H. AND WESTER, T.J. (2003). In 'Manipulating Pig Production IX', p. 159. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

MOREL, P.C.H. AND WOOD, G.R. (2005). *Acta Horticulturae* **674**: 269-275.

Apparent digestibility of nutrients in sows fed wheat-based diets supplemented with phytase

M. Pettitt*, E. Beltranena* and J.S. Sands**

*Prairie Swine Centre Inc., Saskatchewan, S7H 5N9 Canada. **Danisco Animal Nutrition, Wiltshire, SN8 1XN United Kingdom.

While phytase is known to improve phosphorus (P) use in growing and finishing pigs, only a few studies have been done on the use of phytase in sows. The efficiency of use of P in sows is lower than that of growing-and finishing pigs and gestating sows are less efficient than lactating sows (Kemme *et al.,* 1997). Thus, a higher level of supplemental phytase may be required in sow diets. Forty PIC sows (PIC Canada Inc.) were used to study the effect of increasing levels of phytase (Phyzyme XP 5000L, Danisco Animal Nutrition) on: the apparent dry matter of the total tract (DM); nitrogen (N); calcium (Ca); and P digestibility in sows. The sows were assigned to one of four dietary treatments: 1) Positive Control ([PC] 0.70% Ca, 0.57% P, 0.35% available P); 2) Negative Control ([NC] 0.50% Ca and 0.36 P, 0.14% available P); 3) NC plus phytase at 500 FTU/ kg of diet and NC plus phytase at 1000 FTU/ kg of diet in a randomised complete block design. The diets were based on wheat, barley, soybean meal and met the requirements for all nutrients except for Ca and P (NRC, 1998). Chromic oxide was added to the diets as a marker. Diets were fed during gestation (day 60 ± 2 to day 114 ± 2) and lactation (day 1 to day 20). The diets were fed as a single meal of 2 kg daily from day 60 to day 95 and 3 kg daily from day 95 to day 114 during mid and late gestation, respectively. Lactation diets were fed to appetite in three daily portions from day one to day 20 after birth. Water was freely available throughout the study. Faecal samples were collected twice daily and feed intake measured on days 73 to 77 ± 2 days of gestation and days 13 to17 ± 2 days of lactation. Data were analysed using the GLM procedures of SAS. Individual sows were the sampling and experimental units.

During the sampling period in mid gestation, there was no significant effect (P>0.05) on the digestibility of DM or on the dry matter digestibility of CP, Ca and P (Table 1). Litter weights and litter weight gains were not affected by dietary treatment. During the lactation period, digestibility of DM was significantly higher for the 1000 FTU/kg diet than the NC diet (Table 1). Digestibility of CP was higher for the 500 and 1000 FTU/kg diets than the NC diet during lactation. Digestibility of Ca during lactation was significantly higher for the NC and 1000 FTU/kg diet than the PC or 500 FTU/kg diets. Digestibility of P increased linearly in response to phytase and was significantly higher for the 500 and 1000 FTU/kg diets than the NC diet during lactation.

Table 1. Digestibility of dry matter, crude protein, calcium and phosphorus in lactating sows.

Treatment	PC	NC	500	1000	s.e.m.	PC	NC	500	1000	SEM
Item	Gestation					Lactation				
No. of sows	8	8	8	8		7	7	8	8	
DM, %[35]	85.1	83.1	81.9	84.9	1.03	85.3	86.5	87.41	88.3	0.44
CP, % DM[5]	87.6	84.7	83.8	86.4	1.12	85.2	85.5	89.27	88.8	0.73
Ca, % DM[146]	30.9	22.9	25.3	24.9	2.16	44.1	61.8	47.65	64.2	2.23
P, % DM[35]	26.3	16.9	13.1	24.5	3.74	38.7	38.0	55.25	59.3	1.80

Superscripts indicate in Gestation: [1]PC vs NC at (P < 0.05); [2]NC *vs.* 1000 at (P < 0.05); [3]500 vs 1000 at (P < 0.05)
Superscripts indicate in Lactation: [4]PC vs NC at (P < 0.05); [5]NC *vs.* 1000 at (P < 0.05); [6]500 vs 1000 at (P < 0.05)

Supplemental Phytase at 500 and 1000 FTU/kg increased P and crude protein digestibility during lactation when compared to negative control diets, however, there was no effect of supplemental phytase on nutrient digestibility during gestation. The results during gestation support previous studies (Kemme *et al.*, 1997) and are possibly due to a metabolic priority to mobilise body reserves to compensate for low dietary nutrient supply. The improvements in DM and N digestibility in lactating sows, as seen in this study and in previous reports (Kemme *et al.*, 1997; Baidoo *et al.*, 2003), suggest supplemental phytase increases the use of P and other nutrients.

References

BAIDOO, S.K., YANG, Q.M., and WALKER, R.D. (2003). *Animal Feed Science Technology* **104**:133-141.

KEMME, P.A., JONGBLOED, A.W., MROZ, Z., and BEYNEN, A.C. (1997). *Journal of Animal Science* **75**:2129-2138.

NATIONAL RESEARCH COUNCIL (1998). Nutrient Requirements of Swine. 10th ed. National Academy Press, Washington, DC.

Influence of storage temperature of pig colon and faecal digesta without cryogenic pre-treatment on *in vitro* fermentation of potato starch

W.F. Pellikaan, A. Awati, M.W.A. Verstegen and W.J.J. Gerrits

Wageningen University, 6700 AH Wageningen, The Netherlands.

When using digesta samples as inocula for *in vitro* systems it is important to determine the influence of storage temperature. Standardised procedures (feed evaluation), international research consortiums (exchange materials) and ethical considerations (reduced number of experimental animals) have generated interest in the long-term storage of inocula. The effect of storage temperature on faecal and colon chyme from pigs was investigated in an *in vitro* batch culture system, using native potato starch as a substrate.

Faeces and chyme from the proximal colon were obtained from three pigs (45 kg body weight) receiving a diet based on barley, wheat and gelatinised potato starch. The samples were pooled, homogenised, split into two aliquots and stored at either -20° C or -80° C without cryogenic pre-treatment. Two weeks later similar samples were obtained from another three pigs kept under similar conditions. These samples were divided into two aliquots and stored either on ice (0° C) for two hours or kept at 37° C. Frozen samples were allowed to thaw at 37° C for one hour. Inoculates were introduced in airtight 100 ml bottles containing 0.5 g native potato starch in a pre-warmed buffered medium. Cumulative gas production was measured for 96 hours and a mono-phasic model was fitted to the data with inclusion of a lag time. Volatile fatty acids (VFA) and ammonia (NH_3) were determined after fermentation.

Table 1. Fermentation end-products after 96 h *in vitro* incubation using faecal and colon inocula stored at different temperatures.

Temp	tVFA[1]	NH_3[1]	AP[2]	PP[2]	BP[2]	BCP[3]	Temp	tVFA[1]	NH_3[1]	AP[2]	PP[2]	BP[2]	BCP[3]
Faecal inoculum							Proximal colon inoculum						
37 °C	10.3^{a}	4.4	0.467^{a}	0.155^{ab}	0.304^{a}	5.0^{a}	37 °C	10.3^{a}	4.2^{a}	0.512^{a}	0.163	0.262^{a}	3.9^{a}
0 °C	10.4^{a}	3.9	0.453^{a}	0.173^{a}	0.294^{a}	5.1^{a}	0 °C	10.3^{a}	3.9^{ab}	0.500^{a}	0.169	0.267^{a}	3.8^{a}
-20 °C	13.6^{b}	2.6	0.669^{b}	0.123^{b}	0.154^{b}	2.0^{b}	-20 °C	12.0^{ab}	3.0^{b}	0.679^{b}	0.162	0.112^{b}	1.6^{b}
-80 °C	13.3^{b}	3.3	0.650^{b}	0.142^{ab}	0.148^{b}	2.0^{b}	-80 °C	13.6^{b}	3.3^{ab}	0.679^{b}	0.168	0.108^{b}	1.7^{b}
SEM	0.3	0.3	0.015	0.009	0.006	0.1	SEM	0.4	0.3	0.016	0.010	0.007	0.1

[1] tVFA=total volatile fatty acids (mmol/g OM); NH_3=ammonia (mmol/g OM); [2] molar proportions of acetic (AP), propionic (PP) and butyric (BP) acid. [3] Branched chain fatty acid proportion. Different superscripts indicate differences between temperature treatments within inocula type ($P<0.05$).

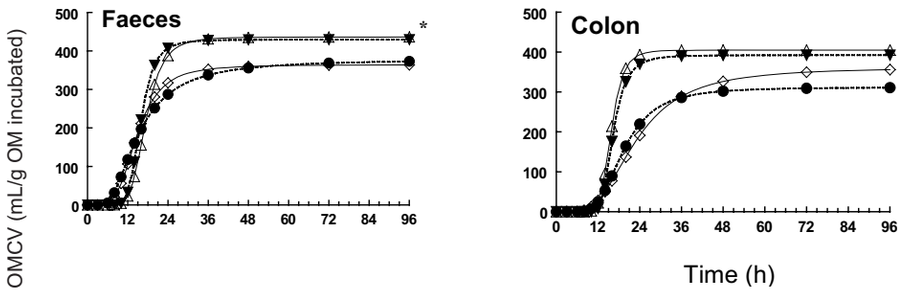


Figure 1. *Cumulative gas production of starch organic matter (OMCV) incubated with inocula from faeces or colon, kept at 37° C (—△—), or stored at 0° C (-- ▼--), -20° C (— ◇—) or -80° C (-- ●--).*

Figure 1 displays the effect of storage temperature on the fermentation kinetics of the potato starch. The cumulative gas production (OMCV) at 96 hours incubation was significantly reduced ($P<0.05$) when faecal inocula was stored frozen. No significant effects were observed for lag time and maximum rate of gas production. Total VFA was higher when using frozen inocula (Table 1) and this was consistent with the decrease in OMCV. The molar proportions of VFA shifted to significantly higher values for acetic acid (AP), mainly at the expense of the butyric acid proportion (BP). The higher BCP observed in the inocula stored at 0° C and 37° C and the trend for increased values for NH_3 reflects an increase in protein fermentation for these inocula. This was caused partly by an increased use of the N-source present in the medium as a source of energy. Our observations show that storing inocula at 3° C and 0° C does not change fermentation kinetics but storage at -20° C and -80° C alters the type of fermentation as well as fermentation kinetics.

Response of 80-100 kg boar and gilt pigs to dietary lysine concentration

M.K. O'Connell*, P.B. Lynch*, J.V. O'Doherty** and P.G. Lawlor*

Moorepark Research Centre, Cork, Ireland. ** University College, Dublin, Ireland.

Differences in the nutritional requirements of boars and gilts have been detected from weights as low as 35 kg (Campbell *et al*., 1988). The differences of boars and gilts become increasingly divergent as protein and lysine content in the diet increases because gilts have a lower potential rate of nitrogen deposition and therefore excrete protein at lower dietary levels than boars (Van Lunen and Cole, 1996). However, for convenience, many producers feed the same diet to both sexes even though this may result in failure of boars to express their potential and in increased nitrogen excretion by gilts. The objectives of this experiment were: 1) To determine, using response curves, the optimal dietary lysine level for maximising growth rate or minimising feed conversion ratio of pigs from 80 to 100 kg live weight. 2) To determine if boars and gilts differ in their lysine requirement.

Same-sex pairs (n=144) were used in a randomised block design with eight treatments from initial weight 80.7 kg (SD 3.8) to 101.9 kg (SD 4.0 kg). Iso-energetic diets (13.8 MJ DE/kg) were based on equal amounts of barley and wheat; soybean meal; vitamins; minerals and amino acids. Dietary total lysine concentrations were: 7.0, 7.9, 8.8, 9.7, 10.7, 11.7, 12.5 and 13.5 g/kg. Methionine, methionine plus cystine, threonine and tryptophan were maintained at ratios to lysine of 0.30, 0.60, 0.66, and 0.20 respectively, while lysine was maintained at 0.06 of crude protein. Back fat and muscle depth were measured using the Hennessy Grading Probe (HGP - Hennessy and Chong, Auckland, NZ). Statistical analysis was by the GLM procedure of SAS (2001) for equally spaced treatments and the NLIN procedure for fitting response curves.

Treatment x sex interactions for average daily gain (ADG, $P<0.01$) and feed conversion ratio (FCR, $P<0.05$) indicated that boars grew faster and had better FCR than gilts and that the difference between sexes was greater at higher lysine levels. Equations were: Boars ADG: $y=-24.3\ (377.2) + 171.9x\ (76.0) - 7.18x^2\ (3.69)$, $R^2 = 0.78$, $P<0.05$; Gilts ADG: $y=-25.1\ (323.2) + 175.4x\ (65.1) - 8.84x^2\ (3.17)$, $R^2=0.64$, $P=0.08$; Boars FCR: $y=6.26\ (1.44) - 0.64x\ (0.29) + 0.027x^2\ (0.014)$, $R^2=0.76$, $P<0.05$; Gilts FCR: $y=5.33\ (0.54) - 0.46x\ (0.11) + 0.023x^2\ (0.005)$, $R^2=0.79$, $P<0.05$. The value in brackets is the standard error of each estimate. Maximum ADG was predicted at 11.8 g (1004 g/d) and 9.9 g (845 g/d) lysine/kg and minimum FCR at 11.9 g (2.47) and 10.0 g (3.03) lysine/kg for boars and gilts respectively (Figure 1). The higher lysine requirement of boars compared with gilts may reflect a growth spurt around puberty.

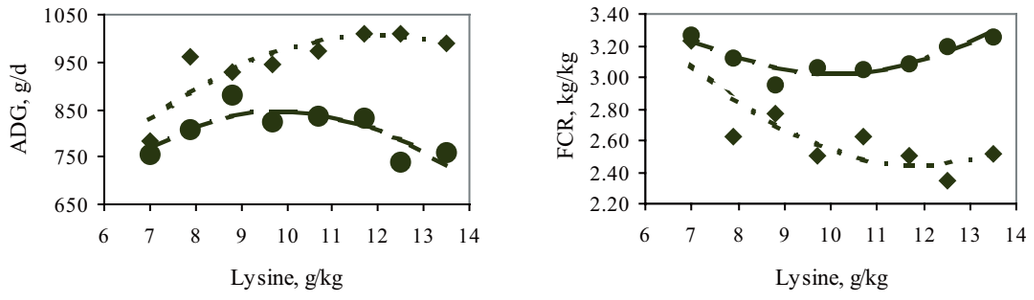


Figure 1. *Quadratic response curves fitted to observed values of ADG and FCR of boars (-◆-) and gilts (-●-).*

There was a linear ($P<0.01$) response in back fat depth to lysine concentration but the sex effect was not significant. Gilts had a higher muscle depth than boars (58.3 mm *vs.* 56.4 mm; SE 0.47; $P<0.01$) but lysine level had no significant effect. Interaction terms were not significant ($P>0.05$) for back fat or muscle depth.

In conclusion, boars grew faster and more efficiently than gilts, with interactions indicating a greater sex difference in performance at high lysine levels. The results support the feeding of different diets to boars and gilts from modern, lean genotypes. Optimum levels of other amino acids for heavy boars need to be determined.

References

CAMPBELL, R.G., TAVERNER, M.R. AND CURIC, D.M. (1988). *Animal Production* **46**:123-130.

SAS (2001). Statistical Analysis Systems, SAS Institute, Cary, North Carolina, USA

VAN LUNEN, T.A. AND COLE, D.J.A. (1996). *Animal Science* **63**:465-475

9 Mycotoxins, environment and feed additives

Recent advances in understanding mycotoxicoses in swine

T.K. Smith, G. Diaz and H.V.L.N. Swamy

Department of Animal and Poultry Science, University of Guelph, Guelph, Ontario, Canada.

Abstract

Mycotoxins are fungal metabolites that can reduce the performance and alter the metabolism of livestock and poultry. The pathological states that arise from consuming feeds contaminated by mycotoxins are referred to as mycotoxicoses. The two major categories of mycotoxins affecting pig production in the Australasian region are aflatoxin and the *Fusarium* mycotoxins. Our understanding of aflatoxicosis in pigs is much more advanced than our understanding of *Fusarium* mycotoxicoses. Determining the minimal concentrations of mycotoxins that will affect pig performance is complicated by the synergistic toxicological interactions that occur when combinations of mycotoxins exist. We now know that a given mycotoxin will almost never be present in isolation. Estimates of safe levels of mycotoxins in pig feeds must be reduced to 50 ppb for aflatoxin and ochratoxin and 200 ppb for T-2 toxin, deoxynivalenol (DON, vomitoxin), nivalenol, zearalenone and fumonisin. This review describes the most recent reports of aflatoxicosis and *Fusarium* toxicosis in pigs by focusing on the impact of these mycotoxins on pig productivity and metabolism.

Introduction

Mycotoxins are fungal metabolites that can reduce the performance and alter the metabolism of livestock and poultry (Wannemacher *et al.*, 1991). The pathological states that arise from consuming feeds contaminated by mycotoxins are referred to as mycotoxicoses. Mycotoxins can be formed in the field before harvest and may continue to be formed under suboptimal storage conditions following harvest. High moisture content often predisposes feedstuffs to fungal growth and mycotoxin formation. Temperature is another key factor controlling fungal growth. *Aspergillus flavus* are usually found in tropical and semi-tropical climates and produce the carcinogenic hepatotoxin aflatoxin. In contrast, *Fusarium* fungi are more common in temperate climates and *Fusarium* mycotoxins are the most common mycotoxins on a global basis (Wood, 1992).

Recent surveys of mycotoxin contamination

Mycotoxin contamination of feedstuffs and the severity of mycotoxicoses in livestock and poultry appear to have increased worldwide during recent years. This may partly be due to the increased monitoring of suspect feedstuffs and the increased awareness of veterinarians and producers regarding the symptoms of mycotoxicoses. Global climate change has also contributed to an increased frequency of mycotoxin contamination of feed grains. Drought, excessive rainfall and flooding can all promote mold growth. A recent survey of corn-based foods and feeds in Indonesia indicated the presence of zearalenone, an estrogenic Fusarium mycotoxin, in 36% of samples (Nuryono *et al.*, 2004). Contamination ranged from 5.5 – 526 ug/kg with poultry feeds being most commonly contamined (>85.7%). Corn samples collected in Karnataka, India, analysed for mycotoxins were found to have contamination rates of 15.2% for ergosterol, 17.7% for aflatoxin, 4.5% for T-2 toxin, 1.5% for ochratoxin, 9.6% for zearalenone and 1.0% for deoxynivalenol (DON) (Janardhana *et al.*, 1999). Fusarium mycotoxins analysed in Nepalese corn and wheat by Desjardins *et al.* (2000) with fumonisins found in 22% of corn samples and DON and nivalenol found in 16% of corn samples. Wheat was less contaminated. Salay and Mercadante, (2002) reviewed the surveys of mycotoxin contamination of Brazilian corn used for animal feed and found high levels of fumonisin contamination were reported. In addition, significant contamination with DON and zearalenone was also observed as well as pockets of aflatoxin contamination.

The European animal feedstuff market is surveyed continuously for mycotoxins including aflatoxin, ochratoxin and Fusarium mycotoxins (Creppy, 2002). Corn harvested in northern Italy during the period 1995 – 1999 was most often contaminated with fumonisins although aflatoxin, DON and zearalenone were also detected (Pietri *et al.*, 2004). Birzele *et al.* (2002) monitored DON content of winter wheat harvested in Germany from 1995-1998 and found the highest level of DON contamination was in 1998 (310 ug/kg). Other, less commonly reported Fusarium mycotoxins, such as beauvericin and enniatins, have also been reported in European wheat (Logrieco *et al.*, 2002). Agricultural practices such as no tillage can increase the frequency of infection post harvest with Fusarium fungi (Edwards, 2004).

A comment must be made about the limitations of analytical methdology for identifying mycotoxins in swine feedstuffs. The concept of 'masked' mycotoxins has recently been introduced, which are newly described chemical

forms of mycotoxins undetectable by conventional analytical techniques. An example is a modified form of DON that has very recently been described in naturally contaminated corn and wheat (Berthiller *et al*., 2005). This is a glucose conjugate that is biologically active in the pig but will not be detected by analytical techniques current available. New protocols need to be developed to quantify free mycotoxins, conjugated mycotoxins and total mycotoxins to accurately determine the hazard posed by the feeding of contaminated feedstuffs. A summary of different fungi, the mycotoxins they produce and the effects of these mycotoxins on pigs is given in Table 1.

Table 1. Common mycotoxins and their effects on pigs.

Fungi	Mycotoxins	Symptoms
Aspergillus flavus	Aflatoxin B_1, B_2, G_1, G_2	Liver necrosis, fatty infiltration of the liver. Immunosuppression
Aspergillus ochraceus catum	Ochratoxin A	Renal nephropathy. Immunosuppression
Fusarium moniliforme	Fumonisin	Pulmonary oedema. Immunosuppression.
	Fusaric acid	Vomiting, lethargy, loss of muscle coordination.
Fusarium graminearum	Deoxynivalenol (DON, vomitoxin)	Vomiting, intestinal lesions. Immunosuppression.
Fusarium roseum	Zearalenone	Hyperestrogenism, abortions, infertility.

Aflatoxicosis in swine

Aflatoxins are produced mainly by *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxicosis has been investigated in many different animal species and in more depth than other mycotoxins (Smith and Ross, 1991) because they are among the most acutely toxic of mycotoxins - causing extensive liver pathology. There is also concern about residues of aflatoxin and metabolites in foods because of the well-documented carcinogenic nature of these compounds.

Effects of dietary aflatoxin on growing and finishing pigs

Bonomi *et al*. (1992) described the chronic aflatoxin B_1 toxicity in growing and finishing pigs (40-140 kg live weight). Feeding 500, 650 and 800 ug/kg aflatoxin B_1 reduced weight gain, feed use, lipid digestion and renal function. In another experiment of similar design, Bonomi *et al*. (1993) showed that the chronic feeding of aflatoxin G_1 damaged liver function more severely than aflatoxin B_1. However, feeding up to 400 ug aflatoxin B_1/kg had little effect on performance and mycotoxin tissue residues in pigs grown from 65-95 kg live weight (Wu *et al*., 1989). Feeding corn contaminated with aflatoxin at a dietary concentration of 922 ug/kg aflatoxin $B_1$to weaning and growing pigs reduced growth rates and feed consumption and elevated serum gamma-glutamyltransferase activities (Schell *et al*., 1993a). Feeding 800 mg aflatoxin / kg for four weeks to starter pigs affected several serum parameters including albumin and total protein concentrations and activities of gamma glutamyltransferase and alkaline phosphatase, which indicated liver damage (Schell *et al*., 1993b). Lindemann *et al*. (1997) found starter pigs fed 500 ug aflatoxin/kg for 34 days showed a 27.8% reduction in average daily weight gain.

Feeding growing boars doses of aflatoxin (2.5 mg/kg) for 32 days reduced serum tocopherol and retinol concentrations (Harvey *et al*., 1995). It was concluded that feeding diets contaminated with aflatoxin might exacerbate Vitamin A and E deficiencies in pigs. Whole body autoradiography of ^{3}H-labeled aflatoxin B_1 in young pigs showed localisation in the nasal olfactory and respiratory mucosa as well as in the liver (Larson and Tjalve, 1996). A significant aspect of aflatoxicosis in swine may be reduced immune function. A study of weanling pigs fed up to 280 ug aflatoxin/ kg indicated a linear reduction in skin thickness at 12 and 24 hours following injection of phytohemagglutinin (Van Heugten *et al*., 1994). Reduced skin thickness was an indication of immuno-suppression and the animals did not respond to the xenobiotic challenge. However, the economic significance of such immuno-suppression will vary with the degree of disease challenge in different production units.

Effects of aflatoxin on swine reproduction

Although low concentrations of aflatoxin are tolerated, the combined adverse effects of aflatoxin on hepatic metabolism, protein synthesis and immune status reduce swine reproductive efficiency. Sows fed graded levels of aflatoxin B_1 up to 400 ug/kg from the first day of gestation to the end of lactation showed no significant effects on number of piglets per litter, piglets weaned at 28 days or survival rate (Wu *et al*., 1992). Residues of aflatoxins B_1 and M_1 were detected in colostrum 14 days after birth with the concentrations rising with dietary aflatoxin concentration. Increasing the dietary dose of aflatoxin B_1 up to 800 ug/kg resulted in reduced numbers of piglets per litter and piglets weaned at 28 days (Bonomi *et al*., 1995a). Similar effects were noted with the feeding of aflatoxin G_1 (Bonomi *et al*., 1995b). The effects of aflatoxin B_1 and aflatoxin G_1 on swine reproduction also appear to be additive (Bonomi *et al.,* 1996).

Action levels for aflatoxin

Fifty ppb (ug/g feed) is the proposed dietary aflatoxin concentration at which intervention should be attempted to prevent adverse effects on production. This also takes account of possible additive or synergistic effects of other mycotoxins, which could result in immunosuppression. A low action level will also minimise aflatoxin residues in tissue to ensure healthiness of pork products and a high standard of food safety.

Ochratoxicosis in swine

Ochratoxin A is produced by several species of Aspergillus and Penicillium. Acute ochratoxicosis is characterised by nephrophathy, enteritis and immunosuppression (Terao and Ohtsubo, 1991). Interest in this mycotoxin has focused on the carcinogenic nature of the compound and associated food and human health issues.

Effects of dietary ochratoxin on growing and finishing pigs

Lippold *et al.* (1992) determined the impact of feeding up to 2.5 mg ochratoxin A/kg to barrows of 15 kg initial weight over 21 days Weight gain, feed intake and feed efficiency were reduced particularly at the highest dietary concentration of ochratoxin A. Impaired renal function, as indicated by hyperproteinemia and azotemia, was noted at 0.5 mg ochratoxin A/kg. A detailed description of nephropathy caused by ochratoxin A in growing pigs in Hungary has been provided by Glavits (1993). The feeding of as little as 800 ug ochratoxin A/kg to growing pigs results in renal lesions over a one year period (Stoev *et al.*, 2002). There have been attempts to correlate pig blood concentrations of ochratoxin A with contamination levels of the feed. In Sweden, 14% of pigs had up to 2 ng ochratoxin A/mL of blood with a maximum concentration of 215 ng/mL (Hult *et al.*, 1992). In western Canada, 36% of serum samples collected from 1600 pigs contained detectable levels of ochratoxin A (Ominski *et al.*, 1996). The Danish swine industry uses renal ochratoxin A residues to minimise potentially harmful residues in pork products (Jorgensen and Peteresen, 2002). Interest has also centered on the immunosuppressive effects of ochratoxin A in swine and Harvey *et al.* (1992) showed that feeding 2.5 mg ochratoxin A/kg to growing gilts for 35 days suppressed cell-mediated immune responses.

Research into the impact of dietary ochratoxin A on swine reproduction has focused on boar semen quality. Feeding ochratoxin A at a rate of five and ten times the human tolerable daily intake reduced initial sperm motility and impaired longevity although it was not clear if ochratoxin A had a direct effect on the germinal epithelium or if it only delayed sperm cell maturation (Solti *et al.*, 1999). In a subsequent study, Biro *et al.* (2003) came to similar conclusions.

Action levels for ochratoxin

Dietary ochratoxin levels in pig feeds should not exceed 50 ppb. Like aflatoxin, ochratoxin residues in foods present a public health hazard and but a low action level will minimise this problem. The possibility that ochratoxin could also contribute to immuno-suppression caused by combinations of mycotoxins adds validity to the argument for low levels.

Fusarium mycotoxicoses in swine

Effects of fumonisin on swine production

The fumonisins are a small family of *Fusarium* mycotoxins that have been discovered relatively recently. Fumonisins are produced mainly by *F. moniliforme*. The chemical structure of the fumonisins enables them to inhibit lipid synthesis in biological membranes. This can result in the lethal condition equine leucoencephalomalacia, which is characterised by massive atrophy of the brain and sudden death. Swine, however, are much less sensitive than horses to acute fumonisin toxicosis, which is characterised by pulmonary edema (Haschek *et al.*, 2001). However the effect of fumonisins on swine immunity is likely to be of greater economic significance.

Feeding 330 mg fumonisin B_1 per kg of feed to weaned piglets resulted in hydrothorax and pulmonary edema and mortality within 5-6 days (Fazekas *et al.*, 1998). However, Zomborszky *et al.* (2000) found feeding weaned piglets up to 40 mg fumonisin B_1 resulted in no significant effect on weight gain or feed consumption and no mortality. In a more chronic study, Rotter *et al.* (1996) fed starter pigs up to 10 mg fumonisin B_1 for eight weeks and found average daily gain was reduced by 11% with the maximum dose of fumonisin. In addition, the he ratio of free sphinganine to free sphingosine increased in liver, pancreas and adrenal glands. This ratio of membrane lipid metabolites is used as a biomarker of fumonisin exposure. An eight-week feeding trial of up to 10 mg fumonisin B_1 did not result in performance impairment or clinical signs (Zomborszky-Kovacs *et al.*, 2002a). The effects of feeding up to 1 mg fumonisin B_1 to growing and finishing pigs to market weight did not significantly affect carcass quality (Rotter *et al.*, 1997).

Smith *et al.* (2000) found daily intravenous injections of fumonisin B_1 to pigs altered cardiovascular function and that the fumonisin-induced pulmonary edema was caused by left-sided heart failure and not by altered endothelial permeability. This was further characterised by Constable *et al.* (2003) as being due to decreased cardiac output and characteristic impedance. The specific nature of fumonisin toxicity in swine has enabled development of a biomarker to measure exposure of pigs to fumonisin. The ratio of serum sphinganine to sphingosine in tissue can be used to indicate whether animals have consumed feeds contaminated with fumonisin (Riley *et al.*, 1993). This is a great advantage as it overcomes problems related to sampling error during feed analysis. Pharmacokinetic studies have indicated that systemic bioavailability of fumonisin B_1 in swine is limited to 3-6% following intragastric administration (Prelusky *et al.*, 1994). Prelusky *et al.* (1996) examined residues of fumonisin B_1 in pig tissues found that the compound accumulated only in liver and kidney with residues only barely detectable nine days after removing contaminated feed from affected animals. Tornyos *et al.* (2003) found it was not possible to demonstrate humoral or cellular specific and non-specific immune responses when weaned pigs were fed up to 100 mg fumonisin B_1 for eight days or lower concentrations for longer periods (up to 10 mg/kg for up to 4 months). This is in contrast to reports with other animal species that have indicated a degree of immuno-suppression. Recent reports indicate that oral doses of fumonisin B_1 given to weanling pigs predispose the animals to infectious disease including intestinal colonisation by pathogenic *E. coli* associated with extraintestinal infection (Oswald *et al.*, 2003).

Effects of fumonisin on swine reproduction

To determine a non-lethal dietary concentration of fumonisin Becker *et al.* (1995) fed lactating sows fed fumonisin B_1 at concentrations of up to 175 mg/kg and found fumonisin B_1 was not detectable in sows' milk and that there was no evidence of toxicosis or immuno-suppression in piglets. However, administration of fumonisin B_1 to gestating sows caused considerable damage to foetuses in *utero* (Zomborszky-Kovacs *et al.*, 2000b).

Action levels for fumonisin

Residues of fumonisin in pork products do not present the same public health hazard as those posed by aflatoxin and ochratoxin. While this enables a higher action level for fumonisin, the immuno-suppressive effects of fumonisin must also be considered and an action level of 200 ppb has been proposed. Fumonisin may serve as a marker mycotoxin indicating the presence of other *Fusarium* mycotoxins..

Effects of deoxynivalenol on swine production

Deoxynivalenol (DON, vomitoxin) is one of the most common *Fusarium* trichothecene mycotoxins. This large family of compounds is generally associated with feed refusal, vomiting and lesions of the gastrointestinal tract in swine. Intravenous infusion of deoxynivalenol to pigs resulted in peak concentrations in cerebral spinal fluid 30 – 60 minutes following infusion (Prelusky *et al.*, 1990). This was considerably longer than that observed for sheep (5-10 minutes) and was thought to be due to the very rapid and extensive tissue distribution of deoxynivalenol in swine. A subsequent study monitored tissue distribution of deoxynivalenol following intravenous dosing with deoxynivalenol and it was observed that there was no extensive uptake or retention by any tissue. This suggested that residue accumulation would not occur in swine upon chronic consumption of deoxynivalenol (Prelusky and Trenholm, 1991). Subsequent long-term swine feeding trials (up to seven weeks) confirmed this (Prelusky and Trenholm, 1992).

Bergsjo *et al.* (1992) determined the impact of feeding growing swine up to 4 mg deoxynivalenol / kg in the form of oats for eight weeks. Feeding 1 mg deoxynivalenol / kg had no effect but feeding the highest dose decreased weight gain, feed intake and feed efficiency. Feeding up to 3.5 mg deoxynivalenol / kg to growing pigs increased liver weights, decreased serum protein and albumin concentrations (Bergsjo *et al.*, 1993). The sub acute toxic effects of dietary deoxynivalenol (up to 3 mg/kg) were examined over a thirty-two day feeding period in grower pigs. Feeding of grains that were naturally contaminated with deoxynivalenol resulted in a more severe feed refusal than when an equivalent amount of purified deoxynivalenol was fed (Prelusky *et al.*, 1994). Rotter *et al.* (1995) fed growing pigs (initial weight 18 kg) diets contaminated with deoxynivalenol (4 mg/kg) for forty-two days and monitored the animals' performance and blood parameters. Although serum protein concentrations were reduced following feeding, the pigs recovered to control levels. They concluded that although deoxynivalenol reduced hepatic protein synthesis the animals were able to adapt to this challenge. Prelusky (1997) compared oral administration of deoxynivalenol with intraperitoneal (IP) infusion in growing pigs and found weight gain was less affected by IP administration - suggesting that the toxic effects of deoxynivalenol are highest with oral administration. House *et al.* (2002) fed growing and finishing pigs to market weight with barley that was naturally contaminated with deoxynivalenol and found that a dietary concentration of 2 mg deoxynivalenol/kg did not reduce growth rates or alter carcass composition of barrows. House *et al.* (2003) found deoxynivalenol was evenly distributed throughout the barley based on abrasive de-hulling.

In studies carried out by Danicke *et al.* (2004), feeding deoxynivalenol to starter and grower pigs in the form of naturally contaminated wheat at 4.6 mg/kg resulted in almost total feed refusal. However, feed intakes recovered quickly when non-contaminated wheat was fed. The concentration of deoxynivalenol in serum increased in a dose-response-related manner as dietary deoxynivalenol concentration increased. This parameter, however, was only weakly correlated with any of the examined performance or serum characteristics.

Prelusky *et al.* (1992) tested the effect of acute intravenous administration of deoxynivalenol on brain regional neurochemistry in pigs and found the changes measured were not indicative of known neurochemical changes associated with chemical-induced anorexia. However Prelusky (1993) administered chronic, low-level concentrations of deoxynivalenol to pigs and noted changes in cerebral spinal fluid that were indicative of elevated brain serotonin turnover in response to reduced feed intake. No changes were measured in pig blood concentrations of serotonin or metabolites following deoxynivalenol administration suggesting that peripheral effects of deoxynivalenol did not account for increased serotonergic activity (Prelusky, 1994). Using an in vitro assay for membrane receptor binding Prelusky (1996) showed that deoxynivalenol possessed only weak affinity for the serotonin-receptor subtypes. Therefore, unless deoxynivalenol concentrations are relatively high, pharmacological effects may be mediated by mechanisms other than interaction with serotonergic receptors. Prelusky and Trenholm (1993) found certain specific serotonin-receptor antagonists prevented vomiting in pigs induced by deoxynivalenol. Overnes *et al.* (1997) determined the effects of feeding graded levels of oats contaminated naturally with deoxynivalenol on the immune responses in growing pigs. Up to 4.7 mg deoxynivalenol was fed for nine weeks. A dose-dependent reduction in secondary antibody response to tetanus toxoid was observed but few other changes were noted. Low doses of deoxynivalenol have been shown to stimulate the immune system in swine (Zielonka *et al.*, 2003). For example, Drochner *et al.* (2004) measured increased serum IgA concentrations in piglets fed up to 1.2 mg deoxynivalenol eight weeks.

Action levels for deoxynivalenol (DON, vomitoxin)

Deoxynivalenol can be used as a marker for *Fusarium* mycotoxins as it the most common of these compounds. However caution is required with the recent report of the naturally occurring, 'masked' deoxynivalenol (Berthiller *et al.,* 2005). This, combined with the immuno-suppressive properties of deoxynivalenol, merits a conservative action level for this compound of 200 ppb.

Effects of zearalenone on swine production

Zearalenone is a *Fusarium* fungal metabolite with estrogenic properties and animal reproduction can be affected adversely by feedstuffs contaminated with zearalenone (Etienne and Dourmad, 1994). Clinical manifestations include vulval swelling and reddening and rectal and vaginal prolapse (Rainey *et al.*, 1991). Kordic *et al.* (1990) determined the impact of feeding less than 1.1 mg zearalenone/kg feed to gilts, gestating sows and lactating sows on reproductive performance. Hyperestrogenic syndrome in the form of vulvovaginitis was observed in 0.24% of gilts with this syndrome receding with time. Similar results were recorded in a study of the effect of very low levels of zearalenone on reproductive efficiency of gilts. It was concluded that feeding 0.5 mg zearalenone/kg of feed would have no serious effects on reproductive efficiency of young gilts (Friend *et al.,* 1990). Green *et al.* (1990) fed pre-pubertal gilts 10 mg zearalenone/kg of feed for two weeks and found that, although zearalenone suppressed mean serum concentrations of leuteinizing hormone, the compound did not delay attainment of puberty or adversely affect subsequent reproduction. Rainey *et al.* (1990) administered lower doses of zearalenone (up to 2 mg/kg) and noted that the hypothalamo-hypophysial axis was affected by zearalenone consumption but later reproductive performance was not affected. Feeding up to 22 mg zearalenone / kg to breeding gilts had an obvious and harmful effect on reproductive performance including decreased number of corpora lutea, decreased weight of ovaries, decreased number of live embryos, increased number of piglets born dead and incidence of abortions (Kordic *et al.*, 1992).

Although zearalenone is metabolised to alpha and beta zearalenols in the pig, Obremski *et al.* (2003) were not able to correlate blood concetrations of zearalenone and alpha zearalenol with external symptoms of hyperestrogenism. Zollner *et al.* (2002) analysed zearalenone and metabolites in the urine and tissues of pigs fed oats contaminated with zearalenone and found 60% of zearalenone in the urine was in the form of alpha and beta zearalenol.

Glucuronide conjugates of zearalenone and the zearalenols were also found in urine and liver. Bieh *et al.* (1993) found there was also extensive biliary secretion and enterohepatic cycling of zearalenone and metabolites in pigs. Identification of zearalenone toxicosis in swine is complicated by the presence of zearelenone glycosides, which release free zearalenone into the digestive tract upon digestion (Gareis *et al.*, 1990).

Action levels for zearalenone

The adverse effects of zearalenone on pig reproduction have been well documented. Like deoxynivalenol, zearalenone has been found in masked forms, which further complicates analysis. The proposed action level for is zearalenone 200 ppb.

Toxicological synergism arising from combinations of mycotoxins

The international trading of feedstuffs has contributed to the severity of mycotoxicoses. Blends of feed ingredients from various geographical regions increase the chance that the feed will contain mixtures of different mycotoxins. This could result in toxicological synergies that increase the severity of mycotoxicoses (Speijers and Speijers, 2004).

Despite feed analyses indicating only very low concentrations of mycotoxins, symptoms typical of swine mycotoxicoses often occur (Trenholm *et al.*, 1983). In these situations it is not always clear if a mycotoxin problem really exists or if the poor performance is due to management or nutritional factors. It is now known that unexpected toxic symptoms may be due to exaggerated toxicological interactions between different mycotoxins. The likelihood of this occurring is highest for the *Fusarium* mycotoxins. It has been shown that feedstuffs contaminated naturally with mycotxins produce higher toxicity than equivalent amounts of purified mycotoxins (Trenholm *et al.*, 1994). Fusaric acid, the most common of the *Fusarium* mycotoxins (Bacon *et al.*, 1996), increases the toxicity of deoxynivalenol in starter pigs (Smith *et al.*, 1997) but is seldom analysed for in swine feeds due to its low toxicity when consumed in the absence of other toxins (Smith and MacDonald, 1991; Smith and Sousadias, 1993).

Interactions between aflatoxin and Fusarium mycotoxins

Harvey *et al.* (1990) evaluated the effects of aflatoxin and T-2 toxin alone and in combination in growing swine and found that when fed in combination, each mycotoxin appeared to have a sparing action on certain effects of the other and that the responses were either additive or less than additive. In a later experiment, Harvey *et al.* (1995) found feeding combinations of aflatoxin B_1 and fumonisin B_1 to growing pigs either alone or in combination adversely affected the pigs' clinical performance, biochemical, hematologic and immunologic values. In general, responses were affected more by the combination diet than the single mycotoxins and the toxic responses could be described as additive or more than additive, particularly for induction of liver disease. More recent studies have also failed to describe major synergistic effects of aflatoxin and fumonisin in growing pigs (Dilkin *et al.*, 2003). Liu *et al.* (2002) investigated the effects of aflatoxin B_1 and fumonisin B_1 on primary swine alveolar macrophages and suggested that both fumonisin B_1 and aflatoxin B_1 were immunotoxic to swine alveolar macrophages but that they exert their toxic effects through different biochemical mechanisms.

Interactions between ochratoxin and other mycotoxins

Several researchers have examined the potential interactions between ochratoxin A and *Fusarium* mycotoxins in swine. Harvey *et al.* (1994) evaluated the effects of ochratoxin and T-2 toxin alone and in combination in growing barrows and found they affected pig performance, serum biochemistry, hematology, immunology and organ weights. The effects were described as additive and not synergistic. Lusky *et al.* (1998) fed combinations of 0.1 mg ochratoxin A / kg and 1.0 mg deoxynivalenol/kg to growing pigs for 90 days and found the mycotoxins were toxic when fed both in combination and alone - indicating that toxicological synergy was absent. Mueller *et al.* (1999) fed weaner pigs combinations of ochratoxin A, deoxynivalenol, T-2 toxin and fumonisin in quantities expected in feeds of central European origin. Apart from ochratoxin alone, little impact of the mycotoxins was found - suggesting that synergistic effects on immunosuppression of these myctoxins in combination should not be expected. Lusky *et al.* (2001) tested the effects of ochratoxin A, deoxynivalenol and zearalenone for 90 days in growing pigs and found no detectable residues of deoxynivalenol or zearalenone in tissues. However, there was an effect of simultaneous administration of deoxynivalenol and zearalenone on secretion of ochratoxin A.

Stoev *et al.* (2001) investigated the potential synergistic effects of ochratoxin A and penicillic acid in young pigs by feeding diets contaminated with *Aspergillus ochraceus*. The microscopic lesions that developed differed from classic Danish procine nephrophathy and the researchers concluded that there could be a synergistic effect between ochratoxin A and penicillic acid. In addition, Sandor *et al.* (1991) tested the impact of combined ochratoxin A and citrinin in swine but no obvious toxicological synergy was detected.

Interactions between fumonisin and other Fusarium mycotoxins

Harvey *et al.* (1996) determined the effects of dietary fumonisin B_1 and deoxynivalenol alone or in combination on the performance, serum biochemistry, immunological responses and histopathological lesions of growing barrows. They concluded that for many variables the response was additive but for some parameters the response was more than additive. It was suggested that caution should be exercised when feeding combinations of fumonisin and deoxynivalenol. However, in a later study, Harvey *et al.* (2002) could not demonstrate a synergic interaction when fumonisin was fed to growing barrows in combination with miniliformin.

Interactions between deoxynivalenol and other Fusarium mycotoxins

Friend *et al.* (1992) evaluated the toxicological interactions between deoxynivalenol and T-2 toxin in growing pigs. Deoxynivalenol was fed up to 2.5 mg/kg and T-2 toxin up to 3.2 mg/kg but there was little evidence of toxicological synergy. A sophisticated attempt to demonstrate toxicological synergy between deoxynivalenol and different *Fusarium* mycotoxins was undertaken in growing swine by Rotter *et al.* (1992). Deoxynivalenol was fed alone or in combination with sambucinol, 15-acetyldeoxynivalenol, 3-acetyldeoxynivalenol and culmorin, however no significant interactions were measured.

Feeding feedstuffs contaminated naturally with combinations of mycotoxins

Corn contaminated naturally with nivalenol (11.5 mg/kg) and zearalenone (3 m/kg) and fed to grower and pregnant pigs during five experiments reduced feed intake, average daily gains and feed efficiencies (Williams and Blaney, 1994). However feeding contaminated corn had no adverse impact on conception rate or number and weights of foetuses at slaughter. When diets containing blends of corn and wheat contaminated naturally with deoxynivalenol, 15-acetyldeoxynivalenol, zearalenone and fusaric acid were fed to starter pigs, marked reductions in growth and feed intake were measured coupled with changes in brain neurochemistry (Swamy *et al.*, 2002). In another experiment Swamy *et al.* (2004) found starter pigs and broiler chickens displayed different sensitivities to combinations of mycotoxins due their differential effects on the brain neurochemistry of the two species. Pair feeding studies indicated that most of the reduced growth rate of pigs fed contaminated grains was due to reduced feed intake (Swamy *et al.*, 2003).

The significance of mycotoxin interactions

The presence of synergistic and additive toxicological interactions between different mycotoxins in feeds makes accurate determination of action levels for individual mycotoxins more difficult. It is therefore necessary to be very conservative in setting action levels to ensure prevention of mycotoxicoses.

Strategies for preventing mycotoxicoses in pigs

Many strategies can be used to prevent the adverse effects of feed-borne mycotoxins. These include dilution of contaminated feeds with non-contaminated feedstuffs, diversion of contaminated feeds to less sensitive species, processing techniques such as screening and de-hulling and the use of organic acids as inhibitors of mold growth. Perhaps the most commonly used strategy is the use of specialty feed additives referred to as mycotoxin adsorbents (Ramos *et al.,* 1996). These are non-digestible, large molecular weight polymers that pass down the digestive tract intact and are excreted in the faeces. Inorganic silica (clay) polymers and organic carbon (plant and yeast fibers) are the most common of these adsorbents. They adsorb mycotoxin molecules in the lumen of the intestine and prevent them from being taken up into blood and transferred to target tissues. The mycotoxins are excreted harmlessly and their effects on pig performance and harmful residues in pork products are minimised. The use of mycotoxin adsorbents has reduced the harmful effects of mycotoxins greatly on global pork production.

Conclusions

Mycotoxicoses research in swine has progressed from the initial descriptive studies of the pathologies arising from acute and chronic administration of individual mycotoxins to focus more on the toxicological synergies arising from combinations of myotoxins. This is most readily achieved by feeding animals feedstuffs contaminated naturally with mycotoxins as this most closely mimics conditions faced by the swine production industry worldwide. Advances in analytical chemistry are needed to more adequately characterise the mycotoxin challenge posed by feeds naturally contaminated with mycotxins. An area yet to be fully explored is the significance of secondary mycotoxic diseases arising from mycotoxin-induced immunosuppression. Quantification of economic losses arising from such diseases will be important to better understand the significance of feed-borne mycotoxins in swine production.

References

BACON, C.W., PORTER, J.K., NORRED, W.P. and LESLIE, J.F. (1996). Production of fusaric acid by Fusarium species. *Applied and Environmental Microbiology* **62**:4039-4043.

BECKER, B.A., PACE, L., ROTTINGHAUS, G.E., SHELBY, R., MISFELDT, M. and ROSS, P F. (1995). Effects of feeding fumonisin B1 in lactating sows and their suckling pigs. *American Journal of Veterinary Research* **56**:1253-1258.

BERGSJO, B., LANGSETH, W., NAFSTAD, I., JANSEN, J. and LARSEN, H.J.S. (1993). The effects of naturally deoxynivalenol-contaminated oats on the clinical condition, blood parameters, performance and carcass composition of growing pigs. *Veterinary Research Communications* **17**:283-294.

BERGSJO, B., MATRE, T. and NAFSTAD, I. (1992). Effects of diets with graded levels of deoxynivalenol on performance in growing pigs. *Journal of Veterinary Medicine Series A* **39**:752-758.

BERTHILLER, F., DALL'ASTA, C., SCHUMACHER, R., LEMMENS, M., ADAM, G. AND KRSKA, R. (2005). Masked Mycotoxins: Determination of a Deoxynivalenol Glucoside in Artificially and Naturally Contaminated Wheat by Liquid Chromatography-Tandem Mass Spectrometry. *Journal of Agricultural and Food Chemistry* **53**:3421-3425.

BIEHL, M.L., PRELUSKY, D.B., KORITZ, G.D., HARTIN, K.E., BUCK, W.B. and TRENHOLM, H. L. (1993). Biliary excretion and enterohepatic cycling of zearalenone in immature pigs. *Toxicology and Applied Pharmacology* **121**:152-159.

BIRO, K., BARNA-BETRO, I., PECSI, T., SZABO, E., WINKLER, G., FINK-GREMMELS, J. and SOLTI, L. (2003). Evaluation of spermatological parameters in ochratoxin A—challenged boars. *Theriogenology* **60**: 199-207.

BIRZELE, B., MEIER, A., HINDORF, H., KRAMER, J. (2002). Epidemiology of *Fusarium* infection and deoxynivalenol content in winter wheat in the Rhineland, Germany. *European Journal of Plant Pathology* **108**:667-673.

BONOMI, A., QUARANTELLI, A., MAZZALI, I., LUCCHELLI, L., CABASSI, E., CORRADI, A., UBALDI, A., FUSARI, A and CHIZZOLINI, R. (1992). *Rivista della Societa Italiana di Scienza dell'Alimentazione* **21**:501-534.

BONOMI, A., QUARANTELLI, A., MAZZALI, I., CABASSO, E., CORRADI, A., UBALDI, A., FUSARI, A and CHIZZOLINI, R. (1993). *Rivista di Scienza dell'Alimentazione* **22**:351-377.

BONOMI, A., QUARANTELLI, A., ZAMBIN, E. M., CABASSI, E., CORRADI, A., DI LECCE, R., UBALDI, A. and FUSARI, A. (1995a). *Rivista di Scienza dell'Alimentazione* **24**:361-384.

BONOMI, A., QUARANTELLI, A., ZAMBINI, E. M., CABASSI, E., CORRADI, A., DI LECCE, R., UBALDI, A. and FUSARI, A. (1995b). *Rivista di Scienza dell'Alimentazione* **24**:385-405.

BONOMI, A., QUARANTELLI, A., ZAMBINI, E.M., CABASSO, E., CORRADI, A., DI LECCE, R., UBALDI, A. and FUSARI, A. (1996). *Rivista di Scienza dell'Alimentazione* **25**:169-189.

CONSTABLE, P.D., SMITH, G.W., ROTTINGHAUS, G.E., TUMBESON, M.E. and HASCHECK, W.M. (2003). Fumonisin-induced blockade of ceramide synthase in sphingolipid biosynthetic pathway alters aortic input impedance spectrum of pigs. *American Journal of Physiology* **284**:H2034-H2044.

CREPPY, E.E.,(2002). Update of survey, regulation and toxic effects of mycotoxins in Europe. *Toxicology Letters* **127**:19-28.

DANICKE, S., VALENTA, H., KLOBAWA, F., DOLL, S., GABTER, M. AND FLACHOWSKY, G. (2004). Effects of graded levels of Fusarium toxin contaminated wheat in diets for fattening pigs on growth performance, nutrient digestibility, deoxynivalenol balance and clinical serum characteristics. *Archives of Animal Nutrition* **58**:1-17.

DESJARDINS, A.E., MANANDHAR, G., PLATTNER, R.D.., MARAGOS, C.M., SHRESTHA, K., and MCCORMICK, S.P. (2000). Occurrence of Fusarium species and mycotoxins in nepalese maize and wheat and the effect of traditional processing methods on mycotoxin levels. *Journal of Agricultural and Food Chemistry* **48**:1377-1383.

DILKIN, P., ZORZETE, P., MALLMANN, C.A., GOMES, J.D.F., UTIYAMA, E.E., OETTING, L.L. and CORREA, B. (2003). Toxicological effects of chronic low doses of aflatoxin B(1) and fumonisin B(1)-containing Fusarium moniliforme culture material in weaned piglets. *Food and Chemical Toxicology* **41**:1345-1353.

DROCHNER, W., SCHOLLENBERGER, M., PIEPHO, H.-P., GOTZ, S., LAUBER, U., TAFAJ, M., KLOBASA, F., WEILER, U., CLAUS, R. and STEFFL, M. (2004). Serum IgA-promoting effects induced by feed loads containing isolated deoxynivalenol (DON) in growing piglets. *Journal of Toxicology and Environmental Health Part A* **67**:1051-1067.

EDWARDS, S.G. (2004). Influence of agricultural practices on fusarium infection of cereals and subsequent contamination of grain by trichothecene mycotoxins. *Toxicology Letters* **153**:29-35.

ETIENNE, M. and DOURMAD, J.-Y. (1994). Effects of zearalenone or glucosinolates in the diet on reproduction in sows: A review. *Livestock Production Science* **40**:99-113.

FAZEKAS, B., BAJMOCY, E., FLAVITS, R., FENYVESI, A. and TANYI, J. (1998). Fumonisin B1 contamination of maize and experimental acute fumonisin toxicosis in pigs. *Journal of Veterinary Medicine Series B* **45**:171-181.

FRIEND, D.W., THOMPSON, B K., TRENHOLM, H.L., BOERMANS, H.J., HARTIN, K.E. and PANICH, P.L. (1992). Toxicity of t-2 toxin and its interaction with deoxynivalenol when fed to young-pigs. *Canadian Journal of Animal Science* **72**:703-711.

FRIEND, D.W., TRENHOLM, H.L., THOMPSON, B.K., HARTIN, K.E., FISER, P.S., ASEM, E.K. and TSANG, B.K. (1990). The reproductive efficiency of gilts fed very low-levels of zearalenone**.** *Canadian Journal of Animal Science* **70**:635-646.

GAREIS, M., BAUER, J., THIEM, J., PLANK, G., GRABLEY, S. and GEDEK, B. (1990). Cleavage of zearalenone-glycoside, a "masked" mycotoxin, during digestion in swine. *Journal of Veterinary Medicine Series B* **37**:236-240.

GLAVITS, R., MOLNAR, T., and SALYI, G. (1993). Nephropathy caused by ochratoxin-a in a hungarian swine stock. *Magyar Allatorvosok Lapja* **48**:343-349.

GREEN, M.L., DIEKMAN, M.A., MALAYER, J.R., SCHEIDT, A.B. and LONG, G.G. (1990). *Journal of Animal Science* **68**:171-178.

HARVEY, R.B., EDRINGTON, T.S., KUBENA, L.F., ELISSALDE, M.H., CASPER, H.H., ROTTINGHAUS, G.E. and TURK, J.R. (1996). Effects of dietary fumonisin B1-containing culture material, deoxynivalenol-contaminated wheat, or their combination on growing barrows. *American Journal of Veterinary Research* **57**:1790-1794.

HARVEY, R.B., EDRINGTON, T.S., KUBENA, L.F., ELISSALDE, M.H. and ROTTINGHAUS, G.E. (1995). Influence of aflatoxin and fumonisin B1-containing culture material on growing barrows. *American Journal of Veterinary Research* **56**:1668-1672.

HARVEY, R.B., EDRINGTON, T.S., KUBENA, L.F., ROTTINGHAUS, G.E., TURK, J.R., GENOVESE, K.J., ZIPRIN, R.L. and NISBET, D.J. (2002). Toxicity of fumonisin from Fusarium verticillioides culture material and moniliformin from Fusarium fujikuroi culture material when fed singly and in combination to growing barrows. *Journal of Food Protection* **65**:373-377.

HARVEY, R.B., ELLISSALDE, M.H., KUBENA, L.F., WEAVER, E.A., CORRIER, D.E. and CLEMENT, B.A. (1992). Immunotoxicity of ochratoxin A to growing gilts. *American Journal of Veterinary Research* **53**:1966-1970.

HARVEY, R.B., KUBENA, L.F. and ELISSALDE, M.H. (1995). The effects of aflatoxin on tocopherol and retinol concentrations in growing barrows. *Agri-Practice* **16**:12-14.

HARVEY, R.B., KUBENA, L.F., ELISSALDE, M.H., ROTTINGHAUS, G.E. and CORRIER, D.E. (1994). Administration of ochratoxin A and T-2 toxin to growing swine. *American Journal of Veterinary Research* **55**:1757-1761.

HARVEY R.B., KUBENA, L.F., HUFF, W.E., CORRIER, D.E., ROTTINGHAUS, G E. and PHILLIPS, T.D. (1990). Effects of treatment of growing swine with aflatoxin and T-2 toxin. *American Journal of Veterinary Research* **51**:1688-1693.

HASCHEK, W.M., GUMPRECHT, L.A., SMITH, G., TUMBLESON, M.E. and CONSTABLE, P.D. (2001). Fumonisin toxicosis in swine: an overview of porcine pulmonary edema and current. *Environmental Health Perspectives* **109**:251-257.

HOUSE, J.D., ABRAMSON, D., CROW, G.H. and NYACHOTI, C.M. (2002). Feed intake, growth and carcass parameters of swine consuming diets containing low levels of deoxynivalenol from naturally contaminated barley. *Canadian Journal of Animal Science* **82**:559-565.

HOUSE, J.D., NYACHOTI, C.M. and ABRAMSON, D. (2003). Deoxynivalenol removal from barley intended as swine feed through the use of an abrasive pearling procedure. *Journal of Agricultural and Food Chemistry* **51**:5172-5175.

HULT, K., HOKBY, E., SELLYEY, G., RUTQVIST, L. and GATENBECK, S. (1992). Ochratoxin A occurrence in slaughter-pigs in Sweden and its use as a tool for feed screening programs. *Journal of Environmental Pathology Toxicology and Oncology* **11**:39-40.

JANARDHANA, G.R., RAVEESHA, K.A. and SHETTY, H.S. (1999). Mycotoxin contamination of maize grains grown in Karnataka (India). *Food and Chemical Toxicology* **37**:863-868.

JORGENSEN, K and PETERSEN, A. (2002). Content of ochratoxin A in paired kidney and meat samples from healthy Danish slaughter pigs. *Food Additives and Contaminants* **19**:562-567.

KORDIC, B., PRIBICEVIC, S., MUNTANOLA-CVETKOVIC, M., NIKOLIC, P. and NIKOLIC, B. (1992). Experimental study of the effects of known quantities of zearalenone on swine reproduction. *Journal of Environmental Pathology, Toxicology and Oncology* **11**:53-55.

KORDIC, B., SIMIC, M. and LAZAREVIC, D. (1990). *Veterinarski Glasnik* **44**:151-155.

LARSSON, P. and TJALVE, H. (1996). Bioactivation of aflatoxin B1 in the nasal and tracheal mucosa in swine. *Journal of Animal Science* **74:**1672-1680.

LINDEMANN, M.D., BLODGETT, D.J., HARPER, A.F., KORNEGAY, E.T. and DOERR, J.A. (1997). Appraisal of the value of selected clays and minerals in diets with and without aflatoxin-contaminated maize fed to young pigs. *Journal of Animal and Feed Sciences* **6**:507-519.

LIPPOLD, C.C., STOTHERS, S.C., FROHLICH, A.A., BOILA, R.J. and MARQUARDT, R.R. (1992). Effects of periodic feeding of diets containing ochratoxin-a on the performance and clinical-chemistry of pigs from 15 to 50 kg body-weight. *Canadian Journal of Animal Science* **72**:135-146.

LIU, B.-H., YU, F.–Y., CHAN, M.-H. and YANG, Y.-L. (2002). The effects of mycotoxins, fumonisin B1 and aflatoxin B1, on primary swine alveolar macrophages. *Toxicology and Applied Pharmacology* **180**:197-204.

LOGRIECO, A., RIZZO, A., FERRACONE, R. and RITIENI, A. (2002). Occurrence of beauvericin and enniatins in wheat affected by Fusarium avenaceum head blight. *Applied and Environmental Microbiology* **68**:82-85.

LUSKY, K., GOEBEL, R., TESCH, D., KOBERSCHUETZ, K. –D., LUSKY, K. and HAIDER, W. (2001). The effects of mycotoxins OTA, ZEA and DON on the health and residue values of pigs. *Tieraerztliche Umschau* **56**:15-20.

LUSKY, K., GOEBEL, R., TESCH, D., TENNER, G., HAIDER, W., KRUEGER, M. and LIPPERT, A. (1998). Studies on the effects of ochratoxin A and deoxynivalenol toxicity on the health of pigs and tissue residue concentrations. *Tieraerztliche Umschau* **53**:623-630.

MUELLER, G., KIELSTEIN, P., ROSNER, H., BERNDT, A., HELLER, M. and KOEHLER, H. (1999). Studies on the influence of combined administration of ochratoxin A, fumonisin B1, deoxynivalenol and T2 toxin on immune and defence reactions in weaner pigs. *Mycoses* **42**:485-493.

NURYONO, N., NOVIANDI, J, BOHM, J. and RAZZAZI-FAZELI, E. (2005). A limited survey of zearalenone in Indonesian maize-based food and feed by ELISA and high performance liquid chromatography. *Food Control* **16**:65-71.

OBREMSKI, K., GAJECKI, M., ZWIERZCHOWSKI, W., BAKULA, T., APOZNANSKI, J and WOJCIECHOWSKI, J. (2003). The level of zearalenone and a-zearalenol in the blood of gilts with clinical symptoms of toxicosis, fed diets with a low zearalenone content. *Journal of Animal and Feed Sciences* **12**:529-538.

OMINSHI, K.H., FROHLICH, A.A., MARQUARDT, R.R., CROW, G.H. and ABRAMSON, D. (1996). The incidence and distribution of ochratoxin A in western Canadian swine. *Food Additives and Contaminants* **13**:185-198.

OSWALD, I.P., DESAUTELS, C., LAFFIEET, J., FOURNOUT, S., PERES, S.Y., ODIN, M., LE BARS, P., LE BARS, J. and FAIRBROTHER, J.M. (2003). Mycotoxin fumonisin B1 increases intestinal colonization by pathogenic Escherichia coli in pigs. *Applied and Environmental Microbiology* **69**:5870-5874.

OVERNES, G., MATRE, T., SIVERTSEN, T., LARSEN, H.J.S., LANGSETH, W., REITAN, L.J. and JANSEN, J.H. (1997). Effects of diets with graded levels of naturally deoxynivalenol-contaminated oats on immune response in growing pigs. *Journal of Veterinary Medicine Series A*: **44**:539-550.

PIETRI, A., BERTUZZI, T., PALLARONI, L. and PIVA, G. (2004). Occurrence of mycotoxins and ergosterol in maize harvested over 5 years in Northern Italy *Food Additives and Contaminants* **21**:479-487.

PRELUSKY, D.B. (1993). The effect of low-level deoxynivalenol on neurotransmitter levels measured in pig cerebral spinal-fluid. *Journal of Environmental Science and Health Part B: Pesticides, Food Contaminants and Agricultural Wastes* **28**:731-761.

PRELUSKY, D.B. (1994). The effect of deoxynivalenol on serotoninergic neurotransmitter levels in pig-blood. *Journal of Environmental Science and Health Part B: Pesticides, Food Contaminants and Agricultural Wastes* **29**:1203-1218.

PRELUSKY, D.B. (1996). A study on the effect of deoxynivalenol on serotonin receptor binding in pig brain membranes. *Journal of Environmental Science and Health Part B: Pesticides, Food Contaminants and Agricultural Wastes* **31**:1103-1117.

PRELUSKY, D.B. (1997**).** Effect of intraperitoneal infusion of deoxynivalenol on feed consumption and weight gain in the pig. *Natural Toxins* **5**:121-125.

PRELUSKY, D.B., GERDES, R.G., UNDERHILL, K.L., ROTTER, B.A., JUI,, P.Y. and TRENHOLM, H.L. (1994). Effects of low-level dietary deoxynivalenol on haematological and clinical parameters of the pig. *Natural Toxins* **2**:97-104.

PRELUSKY, D.B., HARTIN, K.E. and TRENHOLM, H.L. (1990). Distribution of deoxynivalenol in cerebral spinal fluid following administration to swine and sheep. *Journal of Environmental Science and Health Part B: Pesticides, Food Contaminants and Agricultural Wastes* **25**:395-414.

PRELUSKY, D.B., MILLER, J.D. and TRENHOLM, H.L. (1996). Disposition of C-14-derived residues in tissues of pigs fed radiolabelled fumonisin B-1. *Food Additives and Contaminants* **13**:155-162.

PRELUSKY, D.B. and TRENHOLM, H. L. (1991). Tissue distribution of deoxynivalenol in swine dosed intravenously. *Journal of Agricultural and Food Chemistry* **39**:748-751.

PRELUSKY, D.B. and TRENHOLM, H.L. (1992). Nonaccumulation of residues in swine tissue following extended consumption of deoxynivalenol-contaminated diets. *Journal of Food Science* **57**:801-802.

PRELUSKY, D.B. and TRENHOLM, H.L. (1993). The efficacy of various classes of anti-emetics in preventing deoxynivalenol-induced vomiting in swine. *Natural Toxins* **1**:296-302.

PRELUSKY, D.B., TRENHOLM, H.L. and SAVARD, M.E. (1994). Pharmacokinetic fate of 14C-labelled fumonisin B1 in swine. *Natural Toxins* **2**:73-80.

PRELUSKY, D.B., YEUNG, J.M., THOMPSON, B.K. and TRENHOLM, H.L. (1992). Effect of deoxynivalenol on neurotransmitters in discrete regions of swine brain. *Archives of Environmental Contamination and Toxicology* **22**:36-40.

RAINEY, M.R., TUBBS, R.C., BENNETT, L.W. and COX, N.M. (1990). *Journal of Animal Science* **68**:2015-2022.

RAINEY, M.R., TUBBS, R.C., HARDIN, D.K. and COX, N.M. (1991). *Agri-Practice* **12**:35-36.

RAMOS, A.J., FINK-GREMMELS, J. AND HERNANDEZ, E. (1996). *Journal of Food Protection* **59**:631-641.

RILEY R.T., AN, N.–H., SHOWKER, J.L., YOO, H.–S., NORRED, W.P., CHAMBERLAIN, W.J., WANG, E., MERRILL, A.H. and MOTELIN, G. (1993). *Toxicology and Applied Pharmacology* **118**:105-112.

ROTTER, B.A., PRELUSKY, D.B., FORTIN, A., MILLER, J.D. and SAVARD, M.E. (1997). Impact of pure fumonisin B-1 on various metabolic parameters and carcass quality of growing-finishing swine - Preliminary findings. *Canadian Journal of Animal Science* **77**:465-470.

ROTTER, B.A., THOMPSON, B.K. and LESSARD, M. (1995). Effects of deoxynivalenol-contaminated diet on performance and blood parameters in growing swine. *Canadian Journal of Animal Science* **75**:297-302.

ROTTER, B.A., THOMPSON, B.K., PRELUSKY, D.B., TRENHOLM, H.L., STEWART, B., MILLER, J.D. and SAVARD, M.E. (1996). Response of growing swine to dietary exposure to pure fumonisin B1 during an eight-week period: Growth and clinical parameters. *Natural Toxins* **4**:42-50.

ROTTER, R.G., THOMPSON, B.K., TRENHOLM, H.L., PRELUSKY, D.B., HARTIN, K.E. and MILLER, J.D. (1992). A preliminary examination of potential interactions between deoxynivalenol (don) and other selected fusarium metabolites in growing pigs. *Canadian Journal of Animal Science* **72**:107-116.

SALAY, E. and MERADANTE, A.Z. (2002). Mycotoxins in Brazilian corn for animal feed: occurrence and incentives for the private sector to control the level of contamination. *Food Control* **13**:87-92.

SANDOR, G., BUSCH, A., WATZKE, H. REEL, J. and VANYI, A. (1991). Subacute toxicity testing of ochratoxin-a and citrinin in swine. *Acta Veterinaria Hungarica* **39**:149-160.

SCHELL, T.C., LINDEMANN, M.D., KORNEGAY, E.T. and BLODGETT, D.J. (1993a). Effects of feeding aflatoxin-contaminated diets with and without clay to weanling and growing pigs on performance, liver function, and mineral metabolism. *Journal of Animal Science* **71**:1209-1218.

SCHELL, T.C., LINDEMANN, M.D., KORNEGAY, E.T., BLODGETT, D.J. and DOERR, J.A. (1993b). Effectiveness of different types of clay for reducing the detrimental effects of aflatoxin-contaminated diets on performance and serum profiles of weanling pigs. *Journal of Animal Science* **71**:1226-1231.

SMITH, G.W., CONSTABLE, P.D., EPPLEY, R.M., TUMBLESON, M.E., GUMPRECHT, L.A. and HASCHEK-HOCK, W.M. (2000). Purified fumonisin B(1) decreases cardiovascular function but does not alter pulmonary capillary permeability in swine. *Toxicological Sciences* **56**:240-249.

SMITH, J.E. and ROSS, K. (1991). In "Mycotoxins And Animal Foods", PP. 101-139, eds J.E. Smith and R.S. Henderson. (CRC Press: Boca Raton).

SMITH, T.K. and MACDONALD, E.J. (1991). Effect of fusaric acid on brain regional neurochemistry and vomiting behavior in swine. *Journal of Animal Science* **69**:2044-2049.

SMITH, T.K., MCMILLAN, E.G. and CASTILLO, J.B. (1997). Effect of feeding blends of Fusarium mycotoxin-contaminated grains containing deoxynivalenol and fusaric acid on growth and feed consumption of immature swine. *Journal of Animal Science* **75**:2184-2191.

SMITH, T.K. and SOUSADIAS, M.G. (1993). Fusaric acid content of swine feedstuffs. *Journal of Agricultural and Food Chemistry* **41**:2296-2298.

SOLTI, L, PECSI, T., BARNA-VETRO, I., SZASZ, F., BIRO, K. and SZABO, E. (1999). Analysis of serum and seminal plasma after feeding ochratoxin A with breeding boars. *Animal Reproduction Science* **56**:123-132.

SPEIJERS, G.J.A. and SPEIJERS, M.H.M. (2004). Combined toxic effects of mycotoxins. *Toxicology Letters* 153:91-98.

STOEV, S.D., PASKALEV, M. MACDONALD, S. and MANTLE, P.G. (2002). Experimental one year ochratoxin A toxicosis in pigs. *Experimental and Toxicologic Pathology* **53**:481-487.

STOEV, S.D., VITANOV, S., ANGUELOV, G., PETKOVA-BOCHAROVA, T. and CREPPY, E.E. (2001). Experimental mycotoxic nephropathy in pigs provoked by a diet containing ochratoxin A and penicillic acid. *Veterinary Research Communications* **25**:205-223.

SWAMY, H.V.L. N., SMITH, T.K. and MACDONALD, E.J. (2004). Effects of feeding blends of grains naturally contaminated with Fusarium mycotoxins on brain regional neurochemistry of starter pigs and broiler chickens. *Journal of Animal Science* **82**:2131-2139.

SWAMY, H.V.L.N., SMITH, T.K., MACDONALD, E.J., BOERMANS, H.J. and SQUIRES, E.J. (2002). Effects of feeding a blend of grains naturally contaminated with Fusarium mycotoxins on swine performance, brain regional neurochemistry, and serum chemistry and the efficacy of a polymeric glucomannan mycotoxin adsorbent. *Journal of Animal Science* **80**:3257-3267.

SWAMY, H.V.L.N., SMITH, T.K., MACDONALD, E.J., KARROW, N.A., WOODWARD, B. and BOERMANS, H.J. (2003). Effects of feeding a blend of grains naturally contaminated with Fusarium mycotoxins on growth and immunological measurements of starter pigs, and the efficacy of a polymeric glucomannan mycotoxin adsorbent. *Journal of Animal Science* **81**:2792-2803.

TERAO, K. and OHTSUBO, K. (1991). In "Mycotoxins And Animal Foods", pp. 455-488, eds J.E. Smith and R.S. Henderson. (CRC Press: Boca Raton).

TORNYOS, G., KOVACS, M., RUSVAI, M. HORN, P., FODOR, J and KOVACS, F. (2003). Effect of dietary fumonisin B-1 on certain immune parameters of weaned pigs. *Acta Veterinaria Hungarica* **51**:171-179.

TRENHOLM, H.L., COCHRANE, W.P., COHEN, H., ELLIOTT, J.I., FARNWORTH, E.R., FRIEND, D.W., HAMILTON, R.M.G., STANDISH, J.R. and THOMPSON, B.K. (1983). Survey of vomitoxin contamination of 1980 ontario white winter-wheat crop - results of survey and feeding trials. *Journal of the Association of Official Analytical Chemists* **66**:92-97.

TRENHOLM, H.L., FOSTER, B.C., CHARMLEY, L.L. THOMPSON, B.K., HARTIN, K.E., COPPOCK, R.W. and ALBASSAM, M.A. (1994). Effects of feeding diets containing fusarium (naturally) contaminated wheat or pure deoxynivalenol (don) in growing pigs. *Canadian Journal of Animal Science* **74**:361-369.

WANNEMACHER, R.W., BUNNER, D.L. and NEUFELD, H.A. (1991). In 'Mycotoxins and Animal Feeds', pp. 499-522, eds J.E. Smith and R.S. Henderson. (CRC Press: Boca Raton).

WILLIAMS, K.C. and BLANEY, B.J. (1994). Effect of the mycotoxins, nivalenol and zearalenone, in maize naturally infected with *Fusarium graminearum* on the performance of growing and pregnant pigs. *Australian Journal of Agricultural Research* **45**:1265-1279.

WOOD, G.E. (1992). Mycotoxins in foods and feeds in the United States *Journal of Animal Science* **70**:3941-3949.

WU, J.-F., CHENG, C.-S., YU, I-T., YEN, C-C. and KUO, C.-C. (1992). Effects of low dietary levels of aflatoxin on the reproductive-performance of sows. *Journal of the Agricultural Association of China New Series* **159**:82-90.

WU, J.-F., YU, I.-T., CHENG, C.-S., YEN, C.-C. and KUO, C.-C. (1989). Effects of low dietary levels of aflatoxin on performance and tissue residues of finishing pigs. *Journal of the Agricultural Association of China* **146**:42-50.

ZIELONKA, L., GAJECKI, M., OBREMSKI, K and ZWIERZCHOWSKI, W. (2003). Influence of low doses of deoxynivalenol applied per os on chosen indexes of immune response in swine. *Polish Journal of Veterinary Sciences* **6**:74-77.

ZOLLNER, P., JODLBAUER, J., KLEINOVA, M., KAHLBACHER, H., KUHN, T., HOCHSTEINER, W. and LINDNER, W. (2002). Concentration levels of zearalenone and its metabolites in urine, muscle tissue, and liver samples of pigs fed with mycotoxin-contaminated oats. *Journal of Agricultural and Food Chemistry* **50**:2494-2501.

ZOMBORSZKY, M.K., VETESI, F., REPA, I., KOVACS, F., BATA, A., HORN, P., TOTH, A. and ROMVARI, R. (2000a). Experiment to determine limits of tolerance for fumonisin B1 in weaned piglets. *Journal of Veterinary Medicine Series B* **47**:277-286.

ZOMBORSZKY-KOVACS, M., VETESI, F., HORN, P., REPA, I. and KOVACS, F. (2002). Effects of prolonged exposure to low-dose fumonisin B1 in pigs. *Journal of Veterinary Medicine Series B* **49**:197-201.

ZOMBORSZKY-KOVACS, M., VETESI, F., KOVACS, F., BATA, A., TOTH, A. and TORNYOS, G. (2000b). Preliminary communication: examination of the harmful effect to fetuses of fumonisin B(1) in pregnant sows. *Teratogenesis Carcinogenesis and Mutagenesis* **20**:293-299.

Diurnal fluctuations in body weight could cause variation in growth records

B. Schauer, N. Cogger, D.E. Lawton and G. Skilton*

Massey University, Palmerston North, New Zealand. *Aorere Farms, Wanganui, New Zealand.

On commercial pig farms, sample weights are important indicators of herd growth performance. However, sample weights can vary due to random or assignable causes and as a result of sampling procedure. In this study we assessed the diurnal fluctuation in body weights of finisher pigs to evaluate its potential impact on variation in growth records.

Experimental animals were housed in a commercial piggery in the North Island of New Zealand. There were about 10 pigs per pen as mixed gender groups. All pigs had continuous access to water and received a barley-based finisher diet. The study distinguished between two groups according to the feeding systems used on the farm. One group was fed *ad libitum* (AL-group) from a double space wet and dry feeder. The second group was fed four times a day via a computerised liquid feeding system (CL-group). The feeding times for the CL-group were 0830, 1015, 1430 and 2000. During a four-day period pigs were weighed individually at 0700, 1030, 1400 and 1730. The experiment included two replicates in September of subsequent years. Differences in body weight of pigs nested within a pen were assessed for each feeding system using repeated measure analysis. Analysis was carried out using SAS 9.1. The level of significance was 0.05.

A total of 81 pigs were used in the experiment. Thirty-seven pigs were in the AL-group (15 pigs in 2003 and 22 pigs in 2004) and 44 pigs in the CL-group (19 pigs in 2003 and 25 pigs in 2004). Figures 1 and 2 depict the fluctuation in body weight for pigs in the AL-group and CL-group, by replicate. Weight changed with time of day and the changes were consistent within the feeding system used and between replicates. The weight changes were different between the two feeding systems.

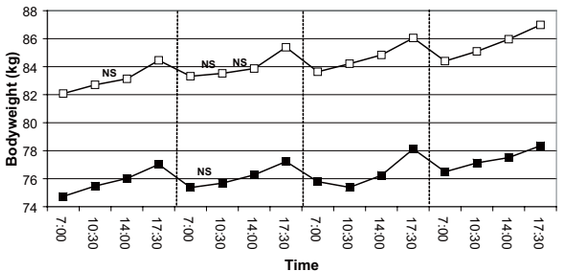


Figure 1. *Diurnal fluctuation in the mean body weight of finisher pigs within an ad libitum feeding system (AL-group), for Replicate 1 (□, n=15) and Replicate 2 (■, n=22). Non-significant differences between subsequent body weights are denoted by NS. Dashed lines denote the different days.*

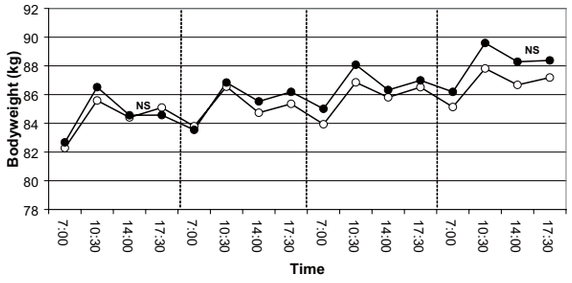


Figure 2. *Diurnal fluctuation in the mean body weight of finisher pigs within a computerised liquid feeding system (CL-group) for Replicate 1 (○, n=19) and Replicate 2 (●, n=25). Non-significant differences between subsequent body weights are denoted by NS. Dashed lines denote the different days.*

In the CL-group, the pattern of diurnal weight change corresponded with the time of feed deliveries. Between 0700 and 1030, when two feeds had been delivered, body weight increased on average by 2.9 kg (95% CI: 2.7-3.1) for Replicate 1 and 3.4 kg (95% CI: 3.2-3.6) for Replicate 2. In contrast, the mean weight in the AL-group increased steadily throughout the day and dropped over night. This suggests that variation in sample weights may be influenced by gut fill. Future research could investigate if food withdrawal before weighing reduces variation in body weight by reducing the variation in gut fill. In conclusion, weighing times as well as feeding times in a CL-situation need to be kept constant to minimise variation in body weight due to sampling procedures.

Identifying the key factors influencing air temperature variation in piggeries

T. Banhazi, D. Rutley and W. Pitchford

University of Adelaide, SA 5371.

The thermal environment influences the production efficiency and welfare of pigs significantly (Morrow-Tesch *et al*., 1994). In piggery buildings, the thermal environment is controlled mainly through building engineering design and installed ventilation systems. Modern piggery buildings are designed to maintain pigs within their optimal thermal zone. However, studies carried out in Australia indicate that the air temperatures in many pig-housing facilities are often outside the optimal range (Banhazi *et al*., 2000). The aim of this study was to determine the key factors affecting the internal temperature in piggery buildings.

As the methodology used in this study has been published, only a brief description will be given here (Banhazi *et al*., 2004). Internal and external temperatures were recorded for 149 piggery buildings, across all seasons, between 1997 and 1999 using temperature data loggers (Tinytalk-2, Hasting Dataloggers, Australia). A general linear model procedure was used to analyse the data in a stepwise manner (SAS, 1989). The significance of different effects was tested using Type III sums of squares. The models were developed from seven fixed effects and 11 co-variates along with their first order interactions.

External temperature alone explained 67% of the variation in the internal temperatures. When the building type (weaner, grower/finisher, dry sow, farrowing and deep-bedded shelters or DBS) was included in the model, 75% of the variation was explained. All other combined significant (P<5%) effects explained only an additional 12% of the variation in internal building temperature (Table 1). Some interactions were important at the 5% and 1% significance levels.

Table 1. General linear models developed for air temperature (°C).

Significance (%)	R^2 (%)	Main effects included in the models	Model df*
P < 5%	87	Vertical height of wall opening	23
P < 1%	84	Building age, stocking density, ridge vent height and control type	17
P < 0.1%	78	Type of wall insulation	8
P < 0.01%	75	External temperature, type of building	5

* df=degree of freedom

The models developed at higher significance levels included the effects of all models developed at lower significance levels.

External temperature was the overriding effect influencing internal temperature. The next most important factor was the type or classification of the pig buildings (built to house a particular type of pig and therefore built with different thermal control capacities). By defining the building type, steps can be taken to improve its thermal control capacity by using improved wall insulation and ensuring the ridge vent height is appropriate. The height of the vertical openings (essentially the size of air inlets) in the wall should also be considered, as larger ventilation inlets will reduce the thermal control capacity of the buildings. The overriding effect of the external environment on the internal temperature of piggeries indicates thermal control could be limited but could be achieved in piggery buildings applying mainstream building engineering practices.

Supported in part by the Australian Pork Limited.

References

BANHAZI, T., CARGILL, C. and WEGIEL, J. (2000). *Proceedings of the 16th International Pig Veterinary Society Congress*, Melbourne, Australia, p. 149.

BANHAZI, T., SEEDORF, J., RUTLEY, D.L. and BERCKMANS, D. (2004). *Proceedings of International Society for Animal Hygiene Conference* St Malo, France, pp. 215-217.

MORROW-TESCH, J.L., McGLONE, J.J. and SALAK-JOHNSON, J.L. (1994). *Journal of Animal Science* **72:** 2599-2609.

SAS (1989) SAS/STAT® User's Guide, Version 6, Fourth Edition, Volume 2, Cary, NC: SAS Institute

Australian Pork

Identifying key factors influencing bacteria concentrations in piggeries

T. Banhazi, D. Rutley and W. Pitchford

University of Adelaide, SA 5371.

Significant numbers of airborne bacteria can be found in the airspace of piggery buildings. In turn, high concentrations of airborne bacteria can affect production efficiency, human and animal health, animal welfare and the external environment (Banhazi *et al*., 2004a,). Continuous versus all-in-all-out management systems as well as environmental and housing factors can influence the concentrations of different pollutants within piggery buildings (Attwood *et al*., 1987). However, these factors have not been identified simultaneously in the one study. The aim of this experiment study was to determine the key housing design and management factors that affect the internal concentrations of viable bacteria in commercial piggeries.

As the methodology used for this study has been published previously, only a brief description will be given here (Banhazi *et al*., 2004b). Airborne bacteria were sampled using a standard Anderson sampler and horse-blood-agar plates (HBA) (Medvet Science, Australia) in 122 piggery buildings. On each farm, data relating to housing and environmental factors were collected, including building dimensions, animal weight, temperature, humidity and measurements of ventilation airflow. The level of hygiene was assessed visually and categorised into three distinct classes (good, fair and poor). The dependent variable was log-transformed and analysed using the step down process in the general linear models procedure (SAS, 1989), considering a number of main effects and co-variates.

Building type (weaner, grower/finisher, dry sow, farrowing and deep-bedded shelters or DBS) and hygiene classification together explained 50% of the variation in bacteria concentration (Table 1) and both effects were equally important. Including management effects and ventilation airflow explained an additional 10% of the variation in bacteria concentrations.

Table 1. General linear models developed for bacteria concentration (cfu/m^3).

Significance (%)	R^2 (%)	Main effects included in the models	Model df*	Total ss**
P < 5%	60	Management, ventilation airflow	14	46.01
P < 0.01%	50	Building type, hygiene classification	6	53.13

* df=degree of freedom ** ss=sums of squares

Deep-bedded shelters had the highest airborne bacteria concentrations, while the bacterial concentrations between traditional buildings did not differ significantly. The presence of bedding materials is thought to be the main reason for the high concentrations of airborne bacteria measured in deep-bedded shelters. Further studies on the relationship between the quality and management of bedding and airborne bacteria concentrations would be useful. Incorporating vegetable oil or plant extract to entrap particles and/or reduce bacterial growth in the bedding might be a practical way to reduce viable and non-viable airborne particles in deep-bedded systems (Banhazi *et al*., 1999).

Pen cleanliness affected bacteria concentration since viable airborne particles are readily generated from dried faeces on pen floors. Faeces dried on the skin of the animals can also be a major source of dust and bacteria. Sub-optimal hygiene in traditional buildings is one of the main causes of high bacteria concentrations and improving pen hygiene will contribute significantly to cleaner conditions and improve production efficiency, human and animal health and welfare and the external environment.

Supported in part by the Australian Pork Limited.

References

ATTWOOD, P., BROUWER, R., RUIGEWAARD, P., VERSLOOT, P., DE WIT, R., HEEDERIK, D. and BOLEIJ, J.S. (1987). *American Industrial Hygiene Association Journal* **48:** 745-751.

BANHAZI, T., SEEDORF, J., RUTLEY, D.L. and BERCKMANS, D. (2004a). *Proceedings of International Society for Animal Hygiene Conference*, St Malo, France, pp. 215-217.

BANHAZI, T., SEEDORF, J., RUTLEY, D., CARGILL, C. and HARTUNG, J. (2004b). *Proceedings of International Society for Animal Hygiene Conference*, St Malo, France, pp. 187-191.

BANHAZI, T., CARGILL, C., MASTERMAN N. and WEGIEL, J. (1999). *Manipulating Pig Production VII.* Adelaide, Australia. p. 28.

SAS (1989) SAS/STAT® User's Guide, Version 6, Fourth Edition, Volume 2, Cary, NC: SAS Institute

Mycotoxins in straw used in Australian deep-litter pig housing systems

D.D. Moore

Biomin Australia, Vic. 3806.

Mycotoxins are secondary metabolites produced when fungi contaminate pastures and feed grains (Bryden 1998). When ingested, mycotoxins cause insidious livestock losses, ill thrift and reduced disease resistance (Bryden *et al.,* 1987). For example, zearalenone (ZEA) causes hyper-estrogenism in pigs and can reduce fertility in sows and boars (Binder 2004). ZEA and deoxynivalenol (DON) are produced by *Fusarium* spp. of fungi. For example, *F. pseudograminearum* produces DON and ZEA in decreasing levels up the tiller of winter cereals that are affected by crown rot (Blaney *et al.,* 1987). While many researchers have investigated the occurrence of mycotoxins in grain, little is known about the prevalence of mycotoxins in the straw of crops. Housing of pigs on straw is becoming a favoured production system due to its perceived benefits to animal welfare and environmental pressures. Consumption of straw by weaner pigs on straw-based systems accounts for 11.5% of total feed intake (Barneveld *et al.* 2004) and these animals could therefore be at risk of increased mycotoxin ingestion. In this experiment we investigated the occurrence of mycotoxins in straw used in deep litter systems for pig production.

Thirty samples of straw were collected at random from pig production units in Queensland, New South Wales, Victoria, South Australia and Western Australia. Representative samples of 0.5-1.0 kg of straw were collected either from inside production sheds or from unused storage bales. Sub-samples of 200 g were ground using a Romer Mill and were analysed for DON, ZEA, aflatoxin B1, B2, G1, G2, ochratoxin A and fumonisins by Romer Labs Singapore using HPLC. Detection limits for the mycotoxins were Aflatoxin B1 <5 ug/kg, Aflatoxin B2, G1, and G2 <3 ug/kg, Ochratoxin <2 ug/kg; ZEA <32 ug/kg; DON <50 ug/kg; and Fumonisin B1 and B2 <100 ug/kg.

Aflatoxins were detected in 13 samples. Twenty-three samples were contaminated with ZEA and 16 with DON. *F. pseudograminearum* could be the source of DON and ZEA, as crown rot is common in wheat and barley tillers in Australia. The source of AFB1 and AFB2 is less clear, as straw is not generally regarded as a suitable substrate for AF production by *Aspergillus flavus,* however the contamination could also have from grain and feed residues contaminating the litter. This study highlights the potential risk that straw used as deep litter for pigs could be contaminated with mycotoxins.

Table 1. Mycotoxin concentration (μg/kg) of straw samples from deep-litter systems across Australia

	AFB1	AFB2	AFG1	AFG2	ZEA	DON	FUM B1	UMB2
Samples analysed	30	30	30	30	29	30	30	30
Samples positive	13	2	-	-	23	16	0	0
Average concentration in positive samples	8.77	16.00	-	-	533	489	-	-
Maximum concentration	11.00	17.00	-	-	3551	1860	-	-
SEM	0.455	1.00	-	-	162	113	-	-

References

BARNEVELD, R. and DOVE, H. (2003). In 'Manipulating Pig Production IX', p.122. ed J.E. Paterson. (Australasian Pig Science Association: Werribee).

BINDER, E. M. (2004). *Australian Pork Journal* **26:** 26-30.

BLANEY, B. and TYLER, A. (1987). *Herbivore Nutrition Research* 37-38.

BRYDEN, W.L. (1998). In 'Toxic Plants and Other Natural Toxicants'. pp. 464-468. eds T. Gardland and A.C. Barr) CAB International.

BRYDEN, W.L., LOVE, R.J. and BURGESS, L.W. (1987). *Australian Veterinary Journal* **64:** 225-226.

Copper and fat interactions in weaner pigs

A. Murphy, A.C. Philphotts and D.J. Henman

QAF Meat Industries, Corowa, NSW 2646.

Young pigs have a reduced capacity to use dietary fat for the first two weeks after weaning and this could explain the poor growth rates of animals observed during this period. Adding copper to high fat diets has been shown to stimulate production of lipases and phospholipases in the small intestine, which results in fats being broken down and used by the weaner pig (Luo and Dove, 1996; Dove, 1995). In this 2 x 2 x 2 factorial experiment we evaluated the impact of adding copper and fat to weaner diets on the performance of weaner pigs. The hypothesis for this experiment was that adding high levels of copper to a high fat diet would improve the growth performance of the weaner pig.

One hundred and twenty male weaners (Large White x Landrace) were placed into individual metabolism crates for 21 days. There were 15 weaners per treatment. The treatments were: 1) no tallow and 10 ppm inorganic copper; 2) no tallow and 250 ppm inorganic copper; 3) no tallow and 10 ppm organic copper 4) no tallow and 100 ppm organic copper; 5) 5% added tallow and 10 ppm inorganic copper; 6) 5% added tallow and 250 ppm inorganic copper; 7) 5% added tallow and 10 ppm organic copper and; 8) 5% added tallow and 100 ppm organic copper. Zinc oxide was included in the diets at 3 000 ppm. Measurements of live weight and feed intake were taken on days one, seven, 14 and 21 and a feed conversion calculated for each period.

Table 1. Growth performance of weaner pigs during days 0-21 fed inorganic or organic sources of copper.

Treatment	Fat level	Copper type	Copper level (ppm)	Start weight (kg)	Final weight (kg)	Rate of gain (kg/day)	Average daily intake (kg/day)	Feed conversion ratio
1	0%	Inorganic	10	6.87	12.94	0.289	0.381	1.32
2	0%	Inorganic	250	6.83	13.05	0.296	0.357	1.21
3	0%	Organic	10	6.84	12.27	0.259	0.318	1.24
4	0%	Organic	100	6.81	12.77	0.283	0.356	1.27
5	5%	Inorganic	10	6.87	12.84	0.284	0.330	1.16
6	5%	Inorganic	250	6.93	14.02	0.338	0.385	1.14
7	5%	Organic	10	6.85	13.19	0.302	0.368	1.23
8	5%	Organic	100	6.83	13.44	0.315	0.367	1.17
Statistics								
Fat[1]				NS	*	**	NS	**
Copper type[1]				NS	NS	NS	NS	NS
Copper level[1]				NS	*	*	NS	NS
SEM				0.048	0.132	0.005	0.007	0.014

[1]NS not significant,*****P<0.05, **P<0.01, ***P<0.001.

Combining high fat diets with growth promoting levels of copper improved the performance of weaner pigs (Table 1). Adding fat to the diet significantly improved weaner performance with a 12% increase in rate of gain (P=0.009) and a 7% improvement in feed to gain (P=0.003). Adding high levels of copper improved the rate of gain by 8% (P=0.019). The improvement was highest for weaners fed diets high in fat and inorganic copper. Therefore, adding inorganic copper to high fat diets for weaner pigs may be useful in promoting pig growth performance during the first 21 days after weaning. However, we estimate that incorporating inorganic copper would result in a 1% increase in copper output across the piggery and if this was deemed unfeasible then organic copper would have to replace inorganic copper during the first 21 days, resulting in a loss of performance.

References

LUO, X.G., and DOVE, C.R. (1996). *Journal of Animal Science* **74**:1888:1896.

DOVE, C.R. (1995). *Journal of Animal Science* **73(1)**:166:171.

Influence of a multi-enzyme preparation on the gut morphology of piglets

S. Jakob*, J.Wolinski**, R.Zabielski** and D.Laubitz**

*Adisseo France S.A.S, Commentry, France. **The Kielanowski Inst. of Animal Physiology and Nutrition, Jablonna, Poland.

It has become common practice to supplement the diet of pigs with carbohydrolases to decrease the anti-nutritive properties of non-starch polysaccharides (NSP) and in doing so increase nutrient digestibility and pig performance. However, it is not known whether the hydrolysis of NSP with carbohydrolases causes a morphological change of the small intestine in piglets. The aim of this study was to determine whether supplementing a diet based on wheat and barley with a multi-enzyme preparation would increase villi length and crypt depth in the small intestine of piglets.

The aim of this experiment was to evaluate the influence of a multi-enzyme preparation (Rovabio™ Excel LC; ADISSEO, France; 17 enzymatic activities, main activities Endo-1.4-β-Xylanase & Endo-1.3(4)-β-Glucanase) on the gut morphology of weaning piglets. Twelve piglets (9.95 ± 0.7 kg) aged 28 days were divided into two groups consisting of six animals each and assigned to two different treatments: 1) a basal diet of wheat, barley and soybean meal (ME 13.9 MJ/kg; XP 17.7%; Lys 11.4 g/kg) and 2) an experimental diet consisting of the basal diet supplemented with Rovabio™ Excel LC at a level of 200 ml/kg. The piglets were housed individually, fed their diets *ad libitum* for three weeks and were euthanised on day 23. Samples from the duodenum, jejunum and ileum were taken and villi length and crypt depth were determined using confocal microscopy. Data were analysed by using ANOVA with enzyme supplementation as the main effect. The interaction between enzyme supplementation and site of absorption was analysed for the ratio of villi length and crypt depth.

Animals remained healthy during the experiment, with no signs of sickness or diarrhoea observed. Villi length in the duodenum did not differ between the two groups (control: 507 μm ± 135; enzyme: 489 μm ± 144). However in the jejunum, villi length was significantly ($P<0.05$) higher in animals fed the diet supplemented with the multi-enzyme preparation (control: 333 μm ± 59; enzyme: 369 μm ± 54) and the increase in villi length of animals fed the enzyme was even more pronounced in the ileum ($P<0.01$). While the enzyme increased ($P<0.10$) the crypt depth in the duodenum (control: 292 μm ± 37; enzyme: 306 μm ± 28), there was no change in crypt depth for either the jejunum or the ileum. There was an interaction ($P<0.05$) between enzyme supplementation and site of absorption. Thus, for control animals, the ratio of villi length and crypt depth dropped from the proximal to the distal parts of the small intestine, while in animals fed the enzyme the ratio remained unchanged (Figure 1).

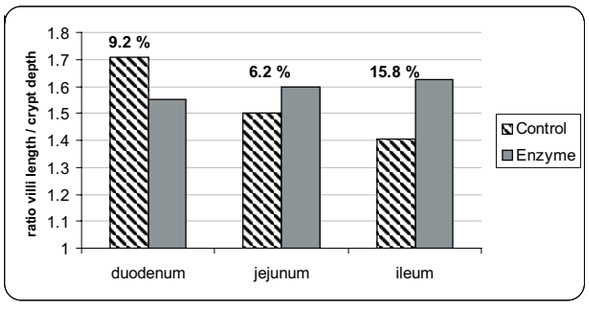


Figure 1: *Ratio of villi length and crypt depth in the duodenum, jejunum and ileum of weaning piglets fed diets supplemented with and without the enzyme preparation Rovabio™ Excel.*

As proposed by Rådberg *et al.* (2001), an increased villi length throughout the small intestine results in an increased absorptive area and increases the capacity of the gut to absorb digested nutrients. In the present experiment, it is significant that villi length of enzyme-supplemented pigs was increased most in the jejunum and ileum, the regions of the small intestine with the highest absorption capacity. We conclude that gut integrity, and therefore optimal function of the small intestine, is improved by adding a multi-enzyme preparation to pig feed.

References

RÅDBERG K., BIERNAT M., LINDEROTH A., ZABIELSKI R., PIERZYNOWSKI S.G. and WESTRÖM B.R. (2001). *Journal of Animal Science* **79**: 2669-2678.

10 Functional foods, meat quality and feed evaluation

Pork as a functional food

D.N. D'Souza*, B.P. Mullan** and F.R. Dunshea***

*Alltech Biotechnology PL, Dandenong South, Vic. 3175. **Department of Agriculture, South Perth, WA 6151.
***Department of Primary Industries, Werribee, Vic. 3030.

Abstract

There is increasing concern about the health status of the human population in many developed countries, in particular the proportion of the population that are classed as being overweight, having metabolic diseases or being at higher risk of developing cancers and cardiovascular disease. There is a misconception among many in the community that consuming meats such as pork is part of the reason for these problems. However, there is growing evidence that a diet high in protein and low in carbohydrate and fat, in conjunction with regular exercise, can actually reduce the incidence of obesity in humans. Pork that is available to consumers today is leaner and contains an excellent balance of essential nutrients for the health and well being of consumers. In addition, with the growth of the functional food sector, there are several opportunities to develop and market pork products with enhanced nutritional value and with a higher bioavailability of nutrients. Nutritionally enhanced pork as part of our everyday diet may also have a far greater impact on the general health of the population than the alternative approach of using dietary supplements.

Introduction

The notion of eating foods that have beneficial effects on consumer health above that of adequate nutritional value is one that sits well with today's consumers. Consequently the development of 'functional foods' and their impact on our health has received much attention over the past 5 to 10 years from scientists, health professionals, health policy makers, agricultural and food producers, and still remains a 'buzzword' within the food and health sector. The majority of the functional foods on the market today aim to offset the incidence of a number of major human non-communicable diseases, with the main constituents of the functional food products being dietary fibre, pro-biotic cultures, minerals and vitamins, oligosaccharides, unsaturated fatty acids and omega-3 fatty acids.

The role of meat in human nutrition and its impact on consumer health is one that has been widely researched and hotly debated by both the food and health industry. Epidemiological studies often find that consuming meat, including pork, positively correlates with the incidence of coronary heart disease (Hu *et al.,* 1999). However, this is not evident when reviewing clinical and animal studies. Furthermore, it is often overlooked that pork is one of the most versatile protein sources available and has a number of 'functional' properties that make it an important nutrient for human health and development. Many of the messages and recommendations that consumers hear are over-simplified and fail to indicate the many health benefits from consuming meat, eggs and dairy products (Dunshea *et al.*, 2005a). This paper will focus on some of the functional properties of pork and will review the general body of research that has been directed towards the further development of pork as a 'functional food' with validated health benefits to the end consumer.

Nutritional profile of pork

Meat consumption, including pork, has been linked with an increased incidence of obesity (Table 1), Type 2 diabetes, and cardiovascular disease. However, dietary fat intake rather than protein has been shown to play the primary role in the increased incidence of these three conditions (Scrimshaw and Guzman, 1968). Since the late 1970s Australian pork producers have undertaken significant steps to ensure leaner pork in keeping with consumer demands and to take advantage of favourable consumer attitudes towards allegedly leaner white meats such as chicken and fish. As a consequence, the overall fat content of pork has been reduced by between 60-65% during this period (Barnes *et al.*, 1996) through a combination of genetic selection, nutrition and the use of metabolic modifiers (Dunshea *et al.,* 2005b).

Table 1. Proportion of men and women aged 20 years and over in Australia that are overweight or obese (Cameron *et al.*, 2003).

	Men		Women	
	Overweight	Obese	Overweight	Obese
1989	37.9	8.8	22.9	10.2
1995	41.2	11.9	25.7	12.6
2001	42.9	16.0	25.8	17.4

Overweight – BMI > 25 kg/m^2; Obesity - BMI>30 kg/m^2.

In addition, comparisons between cooked meats (Table 2) indicates that pork is now significantly lower in fat than lean beef, lamb and skinless chicken breast (Barnes *et al*., 1996) and is a good source of iron and zinc. As a consequence, the National Heart Foundation Food Council has approved 14 of the 22 cuts of 'New-Fashioned Pork' (Barnes *et al*., 1996).

Table 2. Comparison of the nutrient values of 100-gram portions of different cooked meats[a] (Barnes *et al*., 1996).

	Pork	Chicken	Beef	Lamb
Energy (kj)	440	660	529	596
Protein (g)	22.2	22.3	22.7	22.0
Total fat (g)	1.2	1.6	3.9	6.0
Unsaturated fat (g)	0.7	1.0	2.3	3.6
Iron (mg)	1.0	0.6	2.2	2.2
Zinc (mg)	2.9	0.8	6.2	4.7
Sodium (mg)	53	55	52	63

[a] All lean defatted cuts (100 g portions)

Protein, satiety and weight loss

As indicated previously, the incidence of obesity continues to rise at an alarming rate in Australia and many other developed countries, even though there has been significant public awareness and efforts to control weight gain. Although obesity is identified as a disorder of energy balance, there is still much confusion regarding the causes and solutions. The lack of consensus within the scientific community regarding the cause of the rising incidence of obesity, including diet and the optimal method for controlling weight loss has also seen the prevalence of non-scientific remedies for weight loss and prevention of weight regain.

To maintain or reduce body weight, it is generally accepted that energy intake must be controlled and the most effective way to do this has been to restrict fat intake. The recommended dietary guidelines suggest daily intake of carbohydrate, fats and proteins should be about 55%, 30% and 15%, respectively, of the energy intake. However, in the case of weight loss, an increasing body of epidemiological, clinical and experimental studies is challenging this balance of carbohydrate, fats and protein intake. From a public viewpoint high protein diets remain extremely popular and perhaps the most effective, with the Atkins and the Protein Power diets being examples of high protein dietary programs for weight loss. A number of reviews have highlighted the effectiveness of high protein diets over high carbohydrate diets on weight loss (Eistenstein *et al*., 2002; Anderson and Moore, 2004; Layman and Baum, 2004). It has been hypothesised that the effect of high protein diets on weight loss is possibly mediated by the effects of increased satiety, reduced energy efficiency (increased thermogenesis) and increased glycemic control and these will be examined in this section of the paper.

Satiety

Several studies have investigated the effect of high protein diets on hunger, satiety and energy intake (reviews by Eistenstein *et al*., 2002; Layman and Baum, 2004). Although not conclusive, the general consensus of studies indicate that high protein meals have the potential to suppress hunger to a greater degree and result in enhanced sensations of satiety and lower energy intake leading to greater weight loss.

Stubbs *et al*. (1997) reported that men who consumed a high protein breakfast had lower subjective hunger, and high fullness scores compared to men who had a high fat or high carbohydrate diet. However, the effects were relatively short-term as the lower hunger scores did not affect lunch or dinner intakes. In contrast, Latner and Schwartz (1999) reported that women on a short-term high protein lunch consumed 31% less energy and exhibited less pre-dinner hunger sensations compared to women on a high carbohydrate diet. Similarly, Poppitt *et al*. (1998) reported that women on a high protein diet had higher satiety and lower energy intakes than women on a high carbohydrate diet. High protein, low fat diets (meat based) in preschool children resulted in higher short-term satiety and lower food intake in the subsequent meal compared to a high carbohydrate low fat diet. When comparing the effect of high protein versus high fat diets, Porrini *et al*. (1997) found that subjects on high protein diets exhibited higher degrees of intra-meal and post-meal satiety than those on the high fat diet.

In addition to increasing satiety and reducing energy intake, several studies also reported that subjects were more likely to adhere to a high protein diet compared to a high carbohydrate diet. Skov *et al*. (1999) found that men and women on a high protein (meat and dairy), reduced-fat diet, with no control on level of intake, lost 8.9 kg in six months compared to 5.1 kg on the high carbohydrate diet. In addition, more subjects on the high protein diet adhered to the diet and achieved the clinically relevant weight loss (35%) compared to those on the high carbohydrate diet (9%).

A study investigating the impact of a high protein meat diet compared to a high carbohydrate vegetarian diet on satiety and weight loss found that subjects on the meat diet at lunch ate 12% less at dinner compared to the vegetarian diet resulting in greater weight loss (Barkeling *et al.*, 1990).

Anecdotal observations indicate that different types of meat protein effect satiety and weight loss to varying degrees, with red meat (beef, lamb and pork) being more filling and resulting in greater weight loss than white meat (chicken and fish). However, Melanson *et al.* (2003) reported similar weight and fat loss in subjects fed beef or chicken meals. Similarly, Uhe *et al.* (1992) reported no difference in satiety between beef and chicken, but found that satiety was greater in subjects following the fish meal compared to the beef or chicken meal. Therefore, it would seem that high protein meals based around consumption of lean meat does have beneficial effects on satiety and total energy intake, and perhaps more importantly have the prospect of people adhering to the dietary regimen over a long enough time to provide long-lasting benefits to control of body weight.

The role of high protein diets, especially the Atkins and the Protein Power diets, in weight loss have however been widely criticised in the popular press for a possible adverse effect on calcium balance, cardiovascular disease and renal and liver function. However, numerous reports have found that the data did not support the adverse claims made. On the contrary, a number of studies have found that high protein diets had a positive effect on weight loss as well as blood lipid profiles and calcium excretion (Farnsworth *et al.*, 2003). Similarly, Layman *et al.* (2003) comparing a high carbohydrate versus a high protein diet in adult women reported that the carbohydrate group lost 6.9 kg while the protein group lost 7.5 kg, and also had lower triacylglycerols (21%) and TAG/HDL cholesterol (23%). As momentum for high protein diets increases, more studies will no doubt be conducted on the other effects of a high protein diet besides those related to satiety and energy intake. However, some epidemiological studies have associated long-term consumption of diets rich in animal protein with increased hip fracture rates in the elderly (Abelow *et al.,* 1996). Also, using the growing pig as a model Cox *et al.* (2005) found that high protein diets (based on whey and soy protein isolates (WPI and SPI)) reduced bone mineral density although this was partially alleviated in pigs consuming animal protein as WPI.

Increased thermogenesis

It has also been hypothesised that consuming high protein diets results in higher postprandial energy expenditure (Eistenstein *et al.*, 2002). Most research to date has focused on the increased energy expenditure after eating and is referred to as the thermic effect of feeding, representing 10-15% of total energy expenditure (Sims and Danforth, 1987). Robinson *et al.* (1990) reported that men fed a high protein diet had a higher thermic response and whole-body nitrogen turnover than men on a high carbohydrate diet. In addition, the metabolic cost of protein synthesis for the high protein diet was 68% compared to 36% for the high carbohydrate diet. Similarly, Karst *et al.* (1983) reported that the energy expenditure following a high protein meal was three times larger than a high carbohydrate meal. Nair *et al.* (1984) also noted that the thermic response was greater and also more prolonged in subjects fed a high protein diet compared to a high carbohydrate diet. The above studies clearly indicate that compared to high carbohydrate diets, consumption of high protein diets by normal or obese subjects has a greater effect on postprandial energy expenditure. However, the contribution of this increased thermogenesis to weight loss is unknown, as most of the studies highlighted were short-term studies lasting a maximum of 24 hours.

Increased glycaemic control

Diets designed to lower the insulin response to ingested carbohydrate (i.e. low glycaemic index (GI) foods) may improve access to stored metabolic fuels, decrease hunger, and promote weight loss (Ludwig, 2000). The GI is a property of carbohydrate-containing foods that predicts the body's blood glucose response. As protein has a minimal short-term effect on blood glucose compared to carbohydrates, meals high in protein have been used to reduce the GI of meals. Farnworth *et al.* (2003) reported that subjects fed a high protein diet had a lower glycaemic response (smaller plasma glucose curve, and plasma glucose response decreased to a greater extent) compared to subjects fed a standard protein high carbohydrate diet. Similarly, Layman *et al.* (2003) reported that women who consumed a high protein diet had a lower insulin response to meals, greater satiety (subjective score), lost more body weight and had a greater fat/lean ratio compared to women on the high carbohydrate diet. Recent studies also indicate that a high protein diet based on WPI or SPI increased insulin sensitivity with respect to both glucose and amino acid metabolism in the obese mini-pig (Ferrari *et al.,* 2005).

The high and seemingly increasing incidence of obesity in people of all ages is clearly of great concern. Diets that are relatively high in protein and lower in total fat and carbohydrate have been shown to have a positive effect on total energy intake and expenditure. Lean meats such as pork can be an important component of high protein diets whilst at the same time supplying a range of other important micronutrients with benefits to human health.

Pork and micronutrients

The incidence of people consuming vegetarian diets in Australia is increasing for a number of reasons. One reason is that diets with little or no animal derived foods are considered by many a healthier meal option. However, a number of studies have in fact found that the movement towards a plant-based diet is not without nutritional risk. Reviews of the scientific literature by Fairfield and Fletcher (2002), Hunt (2002), and Biesalski (2004) concluded that plant based diets may be inadequate or have low bioavailability for several different micronutrients. This section of the paper will focus on the bioavailability of key micronutrients in meats such as pork, and their impact on human health.

Vitamins

Vitamin A refers to a family of fat-soluble compounds called retinoids and the recommended dietary intake for adults for Vitamin A is 5000IU. Preformed Vitamin A is only found in animal products such as meat, eggs and milk (Fairfield and Fletcher, 2002) and is essential for vision, immune response, epithelial cell growth and repair. Plant derived Vitamin A (ß – carotene) has an extremely poor conversion rate and hence has to be taken in high amounts compared to that found in meat. Therefore, it is important to consider the source of Vitamin A when calculating the daily requirement for particular foods if our requirement is to be met.

Pork is a good source of methyl donors such as folate and vitamin B_{12} (2 Ref). Vitamin B_{12} is another micronutrient that is found only in meat, but unlike Vitamin A there is no plant-derived provitamin B_{12}. It should also be noted that folate derived from meat has a significantly better bioavailability compared to that from fruits and vegetables (Biesalski, 2004). The effect of these micronutrients on the incidence of cancer has been well researched. Studies by Giovannucci *et al.* (1993, 1998) and Benito *et al.* (1991) have found that increased intake of folate and vitamin B_{12} resulted in a reduction in the incidence of colon adenomas. Similarly, the increased breast cancer risk associated with alcohol consumption, was reduced in women who consumed at least 300 ug folate per day. The impact of folate intake on reducing the cancer risk was only evident following a 15-year period suggesting the bioactive ingredient needed to be present in the diet for a very prolonged period. Unfortunately, the average folate intake in Australia is around 200 mg, which is half of the 400 mg recommended daily intake for the average person in the US and it is highly likely that Australia may soon revise its guidelines to follow suit. Therefore encouraging consumers to increase the intake of foods high in folate, such as pork or folate fortified foods may be a viable way to boost folate intake. If this is the case then it would seem a good case for dietary intake via consumption of meat products as opposed to daily supplements, and that this approach would be of far greater benefit to the wider community.

Minerals

Iron and zinc deficiencies are associated with a number of health problems in humans including stunted growth, increased morbidity, and reduced neuro-cognitive development and learning capacity. In Western countries and developing countries, iron deficiency and low iron stores are prevalent in infants, teenagers, and women of childbearing age (Dallman *et al.*, 1980). This may be due to a diet that provides insufficient amounts of available iron to cover the extra needs for growth and menstrual losses. Iron and zinc bioavailability depends on several factors including the mineral's absorption from each food and the inhibitors present in the meal. Plant foods contain a high proportion of mineral absorption inhibitors such as phytic acid, many polyphenols and dietary fibre, with phytic acid perhaps the most potent inhibitor of iron and zinc absorpotion (Rosado *et al.,* 2005). Recent studies have shown that intake of these inhibitors are also more common in diets that contain no animal products (Rosado *et al.,* 2005). As indicated in Table 2 pork is a good source of both iron and zinc, with each serve supplying approximately 10-15% of the recommended daily intake for these minerals. In addition, muscle protein (beef, veal, pork, lamb, chicken, and fish) has long been known to enhance absorption of non-heme (Cook and Monsen, 1976) and heme iron (Halberg *et al.*, 1979). Because non-heme iron accounts for 85–90% of the iron content in a typical Western diet, enhancement of non-heme iron absorption from the diet is of particular importance. Baech *et al.* (2003) reported that the addition of a small amount of pork (50 g) significantly increased the non-heme iron absorption in women fed a meal rich in phytic acid.

Minerals and vitamins play an important role in human nutrition. Pork is a good source of many of these, such as iron, zinc, folate and vitamin B_{12}. On the other hand, some components in plants can reduce the biological availability of minerals in particular. While supplements are available for each of these compounds, ultimately the greatest potential to improve the health of the general population is via major ingredients such as pork. In addition, it may be possible to further increase the concentrations of these compounds in pork.

Nutritional enhancement of pork

The food manufacturing industry has in the last five years paid significant attention to the fortification and the nutritional enhancement of a number of functional properties of everyday foods. The premise is that the majority of people would probably prefer to eat food with added functional aspects rather than take supplements. Given this trend, pork is in a good position for the phrase 'a pig is what it eats' is certainly true due to the fact that the pigs is a monogastric and therefore the ease in which pork can be fortified with key trace elements or have its nutritional value enhanced to convey functional properties. An excellent example of this is the way that we are able to increase the selenium (Se) content of pork.

Selenium

In many countries such as Australia and New Zealand, the relatively low level of Se in soils means that primary products from these countries are also low in Se. While it is generally accepted that Se intakes of Australian and New Zealand consumers are sufficient to ensure that there are no overt signs of deficiency, there is a growing feeling that the relatively low intakes may contribute to elevated levels of risk for some cancers (Dunshea *et al.*, 2005). The effect of Se on human health has been widely researched and has been the subject of numerous reviews (McCartney, 2005; Rayman, 2004; Schrauzer, 2003). The majority of the studies conducted to date indicate that increased dietary Se intakes, particularly in the form of organic Se, confer additional health benefits on the immune system, including reduced viral virulence, reduced cancer risk, reduction in HIV symptoms and progression, reduced risk of cardiovascular disease, and reduced pain from rheumatoid arthritis as outlined in Table 3.

Table 3. Summary of human health benefits from enhanced selenium status (McCartney, 2005).

Benefit related to	Authors	Comment
Immune system/HIV	Burbano *et al.* (2002) Rayman, (2002) Olmsted *et al.* (1988; 1989)	Se from yeast (200 μg/day) proved beneficial in AIDS and ARC
Cancer prevention	Giovannucci, (1998) Yoshizawa *et al.* (1998) Yu *et al.* (1999)	[2]Supplementation with 200 μg Se from yeast daily for up to 7 years with no toxic effects
	Helzlsouer *et al.* (2000) Nomura *et al.* (2000) Brooks *et al.* (2001) Van den Brandt *et al.* (2003) Li *et al.* (2004) Clark *et al.* (1996) Wei *et al.* (2004)	Se from yeast in a controlled study that indicated inverse relationship between baseline serum Se and mortality from oesophageal/stomach cancer
Reduced viral virulence	Beck *et al.* (1995, 1998, 2001) Broome *et al.* (2004)	
Rheumatoid arthritis	Peretz *et al.* (1992) Aaseth *et al.* (1998)	Daily supplement of 200 or 600 μg Se from Se yeast reduced pain
Immune stimulation	Peretz *et al.* (1991)	Daily supplement of 100 μg Se from Se yeast restored immune response to mitogen challenge in elderly subjects
Prevention of cardio vascular disease (CVD)	Blankenberg *et al.* (2003) Alfthan *et al.* (1997) Uthus *et al.* (2002)	CVD mortality increases with plasma homocysteine (HC), and Se deficiency hinders conversion of HC to methionine

Notes: HIV = Human Immunodeficiency Virus; AIDS = Acquired Immune Deficiency Syndrome; ARC = Aids Related Complex

In addition to the ever-accumulating body of epidemiological data suggesting that a higher Se intake is linked to lower cancer mortalities, Schrauzer (2004) reports that mortality from breast, prostate, and skin cancer is much higher in countries with low blood Se (e.g. US and Australasia) and much lower in countries with high blood Se, reflecting higher dietary Se intakes (e.g Japan, Mexico, Thailand). Japan, for example, has a high dietary Se intake, and lower cancer mortality than Western countries. Schrauzer (2004) also links longevity with blood Se and calculates that increasing blood Se from 100 μg/L to 300 μg/L adds four years to life expectancy for both men and women, and recommended a daily Se intake of 200-300 μg. While the conclusions made from studies that associate epidemiological data to, for example, Se status are often challenged as being unscientific, human studies such as those described by Stratton *et al.* (2003) will go a long way to improving our understanding of the role of Se in human health.

Milk, eggs and meat are excellent foods that can be fortified with additional Se (McCartney, 2005). However, as outlined in Table 4, the source (inorganic or organic) can have a significant impact on a range of animal tissue Se levels following Se supplementation. Mahan and Parrett (1996) studied Se content of tissues in a series of studies involving both young growing and finishing pigs. The loin Se content following three dietary Se doses (0.1, 0.3 and 0.5 ppm Se) from inorganic (selenite) and organic (Sel-Plex®) sources were compared in pigs slaughtered at 105 kg live weight. These data indicate a linear dose response with increasing Se dose from either source resulting in significantly improved muscle Se. However, Sel-Plex® supplementation resulted in significantly higher loin Se at each dose of Se compared to sodium selenite. In addition, more Se was excreted in urine in the case of selenite-fed pigs and more Se was retained in muscle in the case of pigs fed Sel-Plex®. This strongly points to the fact that Sel-Plex® is a more efficient way of enhancing the Se content of pork, and using inorganic Se leads to excess Se excretion mainly via the urine, and unnecessary environmental loading.

Table 4. Estimated EU Se consumption from animal products supplemented with 0.3 ppm Se from either selenite or Sel-Plex® (from McCartney, 2005).

EU-15 Daily consumption	Animals supplemented with selenite		Animals supplemented with Sel-Plex	
	Se Concentration	Daily Se contribution (μg)	Se concentration	Daily Se contribution (μg)
Milk, 0.7 l/day	14.3 μg/l	10	29.7 μg/l	21
Eggs, 35 g/day	0.180 μg/g	6	270 μg/g	10
Pork, 115 g/day	0.124 μg/g	14	0.272 μg/g	31
Poultry, 57 g/day	0.150 μg/g	9	350 μg/g	20
Beef, 66 g/day	0.090 μg/g	6	200 μg/g	13
Total daily Se from animal foods		56 μg per day		107 μg per day

Conjugated linoleic acid

Conjugated linoleic acid (CLA) is a mixture of positional and geometric isomers of linoleic acid with conjugated double bonds located at positions 7,9-, 8,10-, 9,11-, 10,12- or 11,13- on the carbon chain. Conjugated linoleic acid was first identified as an anti-mutagenic agent in fried ground beef (Ha *et al.*, 1987). The cis/trans-9,11 isomer has been shown to have specific health benefits such as anti-cancer properties (Pariza *et al.*, 2001; Whigham *et al.*, 2000) and activities against atherosclerosis, the onset of diabetes and obesity (Belury, 2002a,b). The effect of CLA in the inhibition of mammary carcinogenesis in both animal and human studies is perhaps the most extensively documented physiological effect of CLA (Belury, 1995).

Conjugated linoleic acids are usually found in ruminant fats, particularly those from grazing animals. Pork, however, is an ideal candidate for CLA enrichment by feeding synthetic CLA to pigs, because CLA will not be further saturated before absorption. Therefore, CLA deposits in tissues with relatively high efficiency, meaning that pork could become a physiologically significant source of CLA for human consumption. In fact recent studies indicate that the use of CLA as a diet supplement in pigs have resulted in increased CLA concentrations of pork to levels comparable to other major sources of CLA such as milk and beef (Dunshea *et al.*, 2005). Dietary CLA is incorporated into adipose tissue and to a lesser extent into intramuscular fat of pigs in a dose-dependant manner (Eggert *et al.,* 2001; Ostrowska *et al.*, 2003). Numerous pigs studies conducted to date indicate that dietary CLA supplementation had a positive impact on the growth performance of pigs. Dietary CLA supplementation in pigs improved feed efficiency, increased lean tissue deposition and significantly decreased fat deposition in pigs, thus making pork a much healthier meat option for consumers (Ostrowska *et al.*, 1999; Ostrowska *et al.*, 2005).

Lecithin

There is a general consensus within the human health research community that LDL cholesterol is a critical component of both prevention and treatment of coronary heart disease. While therapeutic changes have formed the basis of intervention for lowering LDL, a number of low-risk changes in diet are emerging as key long-term prevention strategies in the fight to lower LDL cholesterol. One of these 'new' strategies is the use of soy lecithin. Soy lecithin, a potent emulsifier, has been shown to influence the absorption of fatty acids in the small intestine. Dietary lecithin supplementation in humans has been shown to reduce cholesterol levels and increases the ratio of polyunsaturated fatty acids (PUFA) to saturated fatty acids (SFA) in both serum and erythrocytes (Spilburg *et al.*, 2003). The most likely rationale for this is that lecithin, an emulsifying agent, allows triglycerides to be digested better. Therefore, in plant-based diets that are higher in PUFA than SFA, a supplement of lecithin should lead to a higher absorption and deposition of PUFAs such as linoleic acid. By contrast, when lecithin combines with cholesterol the resulting micelle

is much larger than one formed with bile salts alone. In this case, lecithin is likely to reduce rather than increase the absorption of cholesterol.

Recent studies have shown that dietary lecithin supplementation influenced the collagen properties of pork and improved the eating quality of pork by reducing the chewiness and hardness of pork (D'Souza *et al.,* 2005b and Edmunds *et al*., 2005). D'Souza *et al.* (2005a) also reported that dietary lecithin supplementation at 75g/kg significantly increased the % of linoleic acid and reduced the myristic acid content of pork. Pigs fed the 75g/kg lecithin supplemented diet also tended to have lower plasma cholesterol at slaughter compared to those fed the control diet and this has the potential to further improve the 'healthiness' of pork. The use of lecithin supplementation to improve the 'healthiness' of pork or pork products, while also improving the tenderness of pork, will provide the pork industry with significant marketing opportunities.

Unsaturated fatty acids

There is increasing evidence from animal and *in vitro* studies which indicates that n-3 fatty acids, especially the long-chain polyunsaturated fatty acids eicosapentaenoic acid and docosahexaenoic acid, present in fatty fish and fish oils inhibit carcinogenesis. Several mechanisms have been proposed whereby n-3 fatty acids may modify the carcinogenic process and n-3 fatty acids suppressing AA-derived eicosanoid biosynthesis; influencing transcription factor activity, gene expression, and signal transduction pathways; modulate estrogen metabolism; increase or decrease the production of free radicals and reactive oxygen species; and influence insulin sensitivity and membrane fluidity (Larsson *et al*., 2004).

In pigs, the amount of fat increases rapidly during growth, and originates both from the diet and from *de novo* synthesis (Dunshea and D'Souza 2003). Madsen *et al.* (1992) reported that 75% of carcass fat resulted from *de novo* synthesis indicating that it is relatively easy to manipulate the fatty acid composition of pork.

Fish meals and oils used in pig diets contain high concentrations of polyunsaturated long-chain fatty acids, C18:2 and C18:3 (>50% of total fatty acids), with a significant number of them having more than 20 carbons with four or more double bonds. Fatty acids C18:2 and C18:3 predominate in some oils whereas eicosapentanoic (C20:5), docosapentanoic (C22:5) and C22:6 occur in high concentrations in others, particularly in fish species from cold waters (Ackman, 1992). All of these fatty acids are readily absorbed by pigs and are deposited in fatty tissues as triacylglycerols where they significantly alter the overall fatty acid composition. Feeding fish oils to pigs is therefore an effective way of increasing the concentrations of these acids in their tissues (Irie and Sakimoto, 1992; Leskanich *et al*., 1994) with the aim of having a beneficial effect on reducing cardio-vascular disease and atherosclerosis. Whilst enrichment of pork can be achieved by adding flaxseed, fish oil, or fishmeal to pig feeds, it should be noted that use of these sources, particularly fishmeal, has been limited by concerns about adverse effects on sensory qualities. However Howe *et al.* (2002) evaluated the use of PorcOmega (POM), a stabilized tuna fishmeal formulation, as a source of DHA for enrichment of pork products. The results indicate that the LC n-3 PUFA (mainly DHA content) of pork products including chops and sausages, were substantially increased (up to seven-fold) by including up to 10% POM in rations. More importantly however, the increases were retained after cooking without anu adverse effect on the sensory qualities of pork.

Most oilseeds and feed grains also contain highly unsaturated lipids (refer Table 5) which, when fed to pigs, can have a marked effect on the fatty acid composition of pork resulting in a beneficial effect on cardio-vascular disease and atherosclerosis. It should, however, be noted that the oils of plant origin do not contain the very long-chain polyunsaturated fatty acids that are present in fish products.

Table 5. Fat content and the fatty acid composition (% distribution) of pig feed ingredients (Madsen *et al*., 1992).

Ingredient	Fat, %	C16:0	C18:0	C18:1	C18:2	C18:3
Barley	3.5	19.8	0.7	10.4	44.2	4.5
Oats, conventional	5.8	14.7	1.0	27.7	35.2	1.6
Oats, naked	11.0	13.8	0.9	36.4	34.7	1.0
Soybean meal	3.1	18.3	4.4	15.9	51.8	6.8
Animal fat	100	23.6	13.3	38.8	6.0	0.5
Soya oil	100	10.0	2.0	29.0	51.0	7.0
Rapeseed	41.1	4.7	1.6	50.2	21.8	9.1
Sunflower seed	31.2	7.0	6.3	22.2	61.6	0.4
Linseed oil	100	6.6	4.9	20.5	14.6	51.9
Palm oil	100	8.3	2.2	23.6	4.0	0.1
Corn oil	100	10.4	1.6	30.7	54.6	1.7

Branding 'functional' pork

While it is possible to further improve the nutritional quality of pork via what we feed the pig, this will come at a cost, which must be recouped, from consumers to make the strategy viable. Therefore, marketing and promotion of functional foods is an important issue to consider at the outset. Two examples of how this is being achieved are with SelenPork and Vitapork™.

SelenPork

SelenPork was developed by a non-government private enterprise scheme in South Korea. Pig producers qualify for the SelenPork brand if the following specifications have been implemented:

- Specified hybrid pig.
- Specified diet (feed must be fed from 60-100 kg live weight and diet to contain 0.5 ppm Se from Sel-Plex®).

Studies to investigate the impact of Sel-Plex® in these production systems have been reported (Table 6) and, on this basis, 0.5 ppm Se from Sel-Plex® was chosen as giving the most economic level of Se in pork. More recently Han (personal communication) found that SelenPork contained almost 20 times more Se than normal pork (4.15 *vs.* 0.22 ppm, respectively). The anti-oxidant properties of Sel-Plex® supplementation have also benefited SelenPork by improving the quality of pork and these include improvements to the tenderness, colour, and drip loss.

Table 6. Selenium content (ppm) of SelenPork (ham and loin) (Han, personal communication).

	Study 1 - Korea	Study 2 - (Ohio State University)
Feed*	0.29	0.22
Feed* + 0.4 ppm Sel-Plex®	5.06	4.87
Feed* + 0.8 ppm Sel-Plex®	2.72	4.47

Contains 0.1 ppm Se from basal ingredients and 0.15 from the mineral premix.

The marketing of SelenPork is strictly controlled and may only be sold through authorised butchers and restaurants. These outlets sell SelenPork exclusively and no other meats. In Korea this is co-ordinated by pig co-operatives who sell to the authorised outlets on behalf of their pig farmer members. They are also responsible for co-ordinating the specified pig and pig feed requirements of their members. Already there are more than 60 outlets widely spread across Korea, all administered through co-operatives. These outlets are jointly owned with the farmer being a shareholder.

The restaurants are typically Korean but the walls are adorned with health information, trial results and other information pertaining to Se. The most popular SelenPork product is the pork skewer and approximately 200-250 g of SelenPork on skewers was found to provide about 50 μg Se, thus making a substantial contribution to daily Se requirements.

Vitapork™

Vitapork™ is another innovative approach to enhancing the 'healthiness' and nutritional value of pork in Belgium. It is a 'brand' of new lean and healthy meat that can be utilised across a range of final product formats (Penny, 2004). The strategy was to increase the most important long chain PUFA, EPA and DHA, and obtain the recommended n-6: n-3 ratio without compromising the physical and organoleptic properties of the carcass. The effect of the increased PUFA levels (soft fat and susceptibility to lipid oxidation) in Vitapork™ pork was overcome by increased antioxidant protection in the pig diets. This included a 5 week Sel-Plex® and vitamin E supplementation period that provided the necessary assurance against lipid oxidation.

Conclusion

Major improvements have been made in the nutritional value of pork in the past 20 years, such that it now meets the needs of consumers in terms of leanness and quality. This review highlights some of the nutritional characteristics of pork that are beneficial to human health. There is good evidence to show that it can also form a very important part of a dietary program to reduce the incidence of obesity and of some human diseases. A growing demand for functional foods, rather than relying on dietary supplements, has seen pork emerge as a very worth candidate in the fight against obesity, and as a vehicle for delivering essential micronutrients such as Se, iron, folate and other functional supplements such as DHA, CLA that in many instances are absent or inadequate in vegetarian diets. Ultimately it may be how well we promote and market such foods that will determine the success of this whole approach to human nutrition.

References

AASETH, J., HAUGEN, M. and FORRE, O. (1998). Rheumatoid arthritis and metal compounds – Perspectives on the role of oxygen radical detoxification. *Analyst* **79**:179-92.

ABELOW, B.J., HOLFORD, T.R. and INSOGNA, K.L. (1992). *Calcified Tissue International* **50:**14–18.

ACKMAN, R.G. (1992). The absorption of fish oils and concentrates. *Lipids* **27**:858-862.

ALFTHAN, G., ARO, A. and GEY, K.F. (1997). Plasma homocysteine and cardiovascular disease mortality. *Lancet* :349:397.

ANDERSON, G.H. and MOORE, S.E. (2004). Dietary proteins in the regulation of food intake and body weight in humans. *Journal of Nutrition* **134:**974S–979S.

BAECH, S.B., HANSEN, M., BUKHAVE, K., JENSEN, M., SORENSEN, S.S., KRISTENSEN, L., PURSLOW, P.P., SKIBSTED, L.H. and SANDSTROM, B. (2003). Non-heme-iron absorption from phytate-rich meal is increased by the addition of small amounts of pork meat. *American Journal of Clinical Nutrition* **77**:173-179.

BARKELING, B., ROSSNER, S. and BJORVELL, H. (1990). Effects of a high-protein meal (meat) and a high-carbohydrate meal (vegetarian) on satiety measured by automated computerized monitoring of subsequent food intake, motivation to eat and food preferences. *International Journal of Obesity* **14**:743-751.

BARNES, J.A., LEWIS, J.L. and BUICK, D.R. (1996). Composition of new-fashioned pork. *Food Australia* **48**:1-16.

BECK, M.A., SHI, Q., MORRIS, V.C. and LEVANDER, O.A., (1995). Rapid genomic evolution of a non-virulent coxsackievirus B3 in selenium-deficient mice results in selection of identical virulent isolates. *Nat Medicine* **1**:405-406.

BECK, M.A., ESWORTHY, R.S., HO, Y.S. and CHU, F.F. (1998). Glutathione peroxidase protects mice from viral-induced myocarditis. *Federation of the American Societies for Experimental Biology Journal* **12**:1143-1149.

BECK, M.A., NELSON, H.K., SHIM Q., VAN DAEL, P., SCHIFFRIN, E.J., BLUM, S., BARCLAY, D and LEVANDER, O.A. (2001). Selenium deficiency increases the pathology of an influenza virus infection. *Federation of the American Societies for Experimental Biology Journal* **15**:1481-1483.

BELURY, M.A. (1995). Conjugated dienoic linoleate: a polyunsaturated fatty acid with unique hemoprotective properties. *Nutrition Reviews* **53**:83-89.

BELURY, M.A. (2002a). Dietary conjugated linoleic acid in health: physiological effects and mechanisms of action. *Annual Reviews in Nutrition* **22**:505-531.

BELURY, M.A. (2002b). Inhibition of carcinogenesis by conjugated linoleic acid: potential mechanisms of action. *Journal of Nutrition* **132**:2995-2998.

BENITO, E., STIGGELBOUT, A. and BOSCH, F.X. (1991). Nutritional factors in colorectal cancer risk: a case-control study in Majorca. *International Journal of Cancer* **49**:161-167.

BIESALSKI, H.K. (2004). Meat as a component of a healthy diet – are there any risks or benefits if meat is avoided in the diet? *Meat Science* **70**:509-524.

BLANKENBERG, S., LUC, G., DUCIMETIERE, P., ARVEILER, D., FERRIERES, J., AMOUYEL, P., EVANS, A., CAMBIEN, F. and TIRET, L. (2003). Interleukin-18 and the Risk of Coronary Heart Disease in European Men: The Prospective Epidemiological Study of Myocardial Infarction. *Circulation* **108**:2453.

BROOME, C.S., MCARDLE, F., KYLE, J.A., ANDREWS, F., LOWE, N.M., HART, C.A., ARTHUR, J.R. and JACKSON, M.J., (2004). An increase in selenium intake improves immune function and poliovirus handling in adults with marginal selenium status. *American Journal of Clinical Nutrition* **80**:154-162.

BROOKS, J.D., METTER, E.J., CHAN, D.W., SOKOLL, L.J., LANDIS, P., NELSON, W.G., MULLER, D., ANDRES, R. and CARTER, H.B. (2001). Plasma selenium level before diagnosis and the risk of prostate cancer development. *Journal of Urology* **166**:2034-2038.

BURBANO, X., MIGUEZ-BURBANO, M.J., MCCOLLISTER, K., ZHANG, G., RODRIGUEZ, A., RUIZ, P., LECUSAY, R. and SHOR-POSNIER, G. (2002). Impact of a selenium chemoprevention clinical trial on hospital admissions of HIV-infected participants. *HIV Clinical Trials* **3**:483-91.

CAMERON, A.J., WELBORN, T.A., ZIMMET, P.Z., DUNSTAN, D.W., OWEN, N., SALMON, J., DALTON, M., JOLLEY, D. and SHAW, J.E. (2003). Overweight and obesity in Australia: the 1999-2000 Australian diabetes, obesity and lifestyle study (AusDiab). *Medical Journal of Australia* **178**: 427-432.

CLARK, L.C., COMBS. G.F JR., TURNBALL, B.W., SLATE, E.H., CHALKER, D.K., CHOW, J., DAVIS, L.S., GLOVER, R.A., GRAHAM, G.F., GROSS, E.G., KRONGRAD, A., LESHER, J.L JR., PARK, H.K., SANDERS, B.B JR., SMITH, C.L. and TAYLOR, J.R. (1996). Effects of selenium supplementation for cancer prevention in patients with carcinoma of the skin. A randomised controlled trial. Nutritional Prevention of Cancer Study Group. *Jama* **276**:1957-1963.

COOK, J.D. and MONSEN, E.R. (1976). Food iron absorption in human subjects. III. Comparison of the effect of animal proteins on nonheme iron absorption. *American Journal of Clinical Nutrition* **29**:859-67.

COX, M.L., FERRARI, J.M., OSTROWSKA, E., KETSES, C., MURALITHARAN, M., TATHAM, B.G., MCCAULEY, I., EASON, P.J., KERTON, D.J. and DUNSHEA, F.R. (2005). Dietary whey protein concentrate partially alleviates the decrease in bone density observed with high protein diet. In 'Manipulating Pig Production X', p. 226. ed. J.Paterson. (Australasian Pig Science Association: Werribee).

DALLMAN, P.R., SIIMES, M.A. and STEKEL, A. (1980). Iron deficiency in infancy and childhood. *American Journal of Clinical Nutrition* **33**:86-118.

D'SOUZA, D.N., EDMUNDS, B.L, WILLIAMS, I.H., MCLEISH, J., MULLAN, B.P., AND DUNSHEA, F.R. (2005). Dietary lecithin improves the healthiness of pork. In 'Manipulating Pig Production X', p. 10. ed J.E. Paterson. (Australasian Pig Science Association, Attwood).

D'SOUZA, D.N., MULLAN, B.P., MCLEISH, J., PETHICK, D.W. and DUNSHEA, F.R. (2005). Dietary lecithin improves the compression properties of pork from the *Semitendinousus* muscle In 'Manipulating Pig Production X', p. 270. ed J.E. Paterson. (Australasian Pig Science Association, Attwood).

DUNSHEA, F.R., OSTROWSKA, E., FERRARI, J.M., HEARD, J.W., DOYLE, P.T. and WALKER, G.P. (2005a). Functional foods from animal production. In Recent Advances in Animal Nutrition in Australia, **15**:1-13.

DUNSHEA, F.R., D'SOUZA, D.N., PETHICK, D.W., HARPER, G.S. and WARNER, R.D. (2005b). Effects of dietary factors and other metabolic modifiers on quality and nutritional value of meat. *Meat Science* **71**; 8-38.

EDMUNDS, B.L, WILLIAMS, I.H. MULLAN, B.P., MCLEISH, J., DUNSHEA, F.R. and D'SOUZA, D.N. (2005). Dietary lecithin improves the compression properties of pork from the *Longissimus Thoracis* muscle In 'Manipulating Pig Production X', p. 271. ed J.E. Paterson. (Australasian Pig Science Association, Werribee).

EGGERT, J.M., BELURY, M.A., KEMPA-STECZKO, A., MILLS, S.E. and SCHINCKEL, A.P. (2001). Effects of conjugated linoleic acid on the belly firmness and fatty acid composition of genetically lean pigs. *Journal of Animal Science.* **79**:2866-2872.

EISTENSTEIN, J., ROBERTS, S.B., DALLAL, G. and SALTZMAN, E. (2002). High protein weight-loss diets: are they safe and do they work? A review of the experimental and epidemiologic data. *Nutrition Reviews.* **60**: 189-200.

FARNSWORTH, E., LUSCOMBE, N.D., NOAKES, M., WITTERT, G., ARGYIOU, E. and CLIFTON, P.M. (2003). Effect of a high-protein, energy-restricted diet on body composition, glycemic control, and lipid concentrations in overweight and obese hyperinsulinemic men and women. *American Journal of Nutrition* **78**: 31-39.

FAIRFIELD, R.H. and FLETCHER, K.P. (2002). Vitamins for chronic disease prevention in adults: clinical applications. *Jama* **287**:3127-3129.

FERRARI, J.M., OSTROWSKA, E., MURALITHARAN, M., TATHAM, B.G., MCCAULEY, I., COX, M.L., EASON, P.J., KERTON, D.J. and DUNSHEA, F.R. (2005). High protein diets containing whey protein concentrate and soy protein isolate improve insulin resistance and reduce weight and fat gain in obese mini-pigs. In 'Manipulating Pig Production X', p. 267, ed. J.E.Patterson. (Australasian Pig Science Association: Werribee).

GIOVANNUCCI, E., STAMPFER, M.J., COLDITZ, G.A., RIMM, E.B., TRICHOPOULOS, D., ROSNER, B.A., SPEIZER, F.E. and WILLETT, W.C. (1993). Folate, methionine, and alcohol intake and risk of colorectal adenoma. *J Natl Cancer Inst* **85**:875-884.

GIOVANNUCCI, E., STAMPFER, M.J., COLDITZ, G.A., HUNTER, D.J., FUCHS, C., ROSNER, B.A., SPEIZER, F.E. and WILLETT, W.C. (1998) Multivitamin use, folate, and colon cancer in women in the Nurses' Health Study. *Ann Intern Med* **129**:517-524.

HALLBERG, L., BJORN-RASMUSSEN, E., HOWARD, L. and ROSSANDER, L. (1979). Dietary heme iron absorption. A discussion of possible mechanisms for the absorption-promoting effect of meat and for the regulation of iron absorption. *Scand J Gastroenterol* **14**:769-79.

HELZLSOUER, K.J., HUANG, H.Y., ALBERG, A.J., HOFFMAN, S., BURKE, A., NORKUS, E.P., MORRIS, J.S. and COMSTOCK, G.W. (2000). Association between alpha-tocopherol, gamma-tocopherol, selenium, and subsequent prostate cancer. *J Natl Cancer Inst* **92**:2018-2023.

HOWE, P.R., DOWNING, J.A., GREYNER, B.F., GRIGONIS-DEANE, E.M. and BRYDEN, W.L. 2002. Tuna fishmeal as a source of DHA for n-3 PUFA enrichment of pork, chicken, and eggs. *Lipids* **37**:1067-1076.

HUNT, J.R. (2002). Moving Toward a Plant-based Diet: Are Iron and Zinc at Risk? *Nutrition Reviews* **60**:127-134.

IRIE, M. and SAKIMOTO, M. (1992). Fat characteristics of pigs fed fish oil containing eicosapentaenoic and docosahexaenoic acids. *Journal of Animal Science* **70**:470-477.

KARST, H., STEINIGER, J., NOACK, R. and STEGLICH, HD. (1984). Diet-induced thermogenesis in man: thermic effects of single proteins, carbohydrates and fats depending on their energy amount. *Ann Nutr Metab* **28**:245-252.

LARSSON, S.C., KUMLIN, M., INGELMAN-SUNDBERG, M. and WOLK, A. (2004). Dietary long-chain n-3 fatty acids for the prevention of cancer: a review of potential mechanisms. *American Journal of Clinical Nutrition* **79**:935-945.

LATNER, J.D. and SCHWARTZ, M. (1999). The effects of a high-carbohydrate, high-protein or balanced lunch upon later food intake and hunger ratings. *Appetite* **33**: 119-128.

LAYMAN, D.K., BOILEAU, R.A., ERICKSON, D.J., PAINTER, J.E., SHIUE, H., SATHER, C. and CHRISTOU, D.D. (2003). A reduced ratio of dietary carbohydrate to protein improves body composition and blood lipid profiles during weight loss in adult women. *Journal of Nutrition* **133**: 411-417.

LAYMAN, D.K., SHIUE, H., SATHER, C., ERICKSON, D.J. and BAUM, J. (2003). Increased dietary protein modifies glucose and insulin homeostasis in adult women during weight loss. *Journal of Nutrition* **133**:405-410.

LAYMAN, D.K. and BAUM, J.I. (2004). Dietary Protein Impact on Glycemic Control during Weight Loss. *Journal of Nutrition.* **134**: Health Module 968S.

LESKANICH, C.O., NOBLE, R.C. and MORGAN, C.A. (1994) Manipulation of the polyunsaturated fatty acid content of pig meat in conformity with dietary guidelines. *Proceedings of the Nutrition Society of UK.* **53**:14A.

LI, H., STAMPFER, M.J., GIOVANNUCCI, E.L., MORRIS, J.S., WILLETT, W.C., GAZIANO, J.M. and MA, J. (2004). A prospective study of plasma selenium levels and prostate cancer risk. *J Natl Cancer Inst* **96**:696-703.

LUGWIG, D.S. (2000). Dietary glycemic index and obesity. *Journal of Nutrition* **130**:280S-283S.

MADSEN, A., JAKOBSEN, K. and MORTENSEN, H. (1992). Influence of dietary fat on carcass fat quality in pigs: A review. *Acta Agric. Scand* **42**:220-225.

MAHAN, D.C. and PARRETT, N.A. (1996). Evaluating the efficacy of selenium-enriched yeast and sodium selenite on tissue selenium retention and serum glutathione peroxidase activity in grower and finisher swine, *Journal of Animal Science* **74:**2967-2974.

MCCARTNEY, E. (2005). Living longer, happier, healthier? Selenium supplementation of human foods – the need to move forward. In Nutritional Biotechnology in the Feed and Food Industry, *Proceedings of Alltech's 21st Annual Symposium, Kentucky,* USA.pp.121-131.

MELANSON, K., GOOTMAN, J., MYRDAL, A., KLINE, G. and RIPPLE, J.M. (2003). Weight loss and total lipid profile changes in overweight women consuming beef or chicken as the primary protein source. *Nutrition* **19**:409-414.

NAIR, K.S., HALLIDAY, D. and GARROW, J.S. (1983). Thermic response to isoenergetic protein, carbohydrate or fat meals in lean and obese subjects. *Clinical Science* **65**:307-312.

NOMURA, A.M., J. LEE, J., STEMMERMANN, G.N. and COMBS, Jr., G.F. (2000). Serum selenium and subsequent risk of prostate cancer. *Cancer Epidemiology Biomark. Previews* **9**:883-887.

OLMSTED, L., SCHRAUZER, G.N., FLORES-ARCE, M. and DOWD, J. (1988). Selenium supplementation of symptomatic immunodeficiency virus infected patients. *Biological Trace Element Research* **15**:167-177.

OLMSTED, L., SCHRAUZER, G.N., FLORES-ARCE, M. AND DOWD, J. (1989). Selenium supplementation of symptomatic human immunodeficiency virus infected patients. *Biological Trace Element Research* **20**:59-65.

OSTROWSKA, E., MURALITHARAN, M., CROSS, R.F., BAUMAN, D.E. and DUNSHEA, F.R. (1999). Dietary conjugated linoleic acids increase lean tissue and decrease fat deposition in growing pigs. *Journal of Nutrition* **129**:2037-2042.

OSTROWSKA E, CROSS R.F., MURALITHARAN M, BAUMAN D.E. and DUNSHEA F.R. (2003) Dietary conjugated linoleic acid differentially alters fatty acid composition and increases conjugated linoleic acid content in porcine adipose tissue. *British Journal of Nutrition* **90:**915-28.

OSTROWSKA, E., CROSS, R.F., WARNER, R.D., MURALITHARAN, M., BAUMAN, D.E. and DUNSHEA, F.R. (2005) Dietary conjugated linoleic acid improves carcass quality without altering meat quality in the growing pig. *Australian Journal of Agricultural Research* **45**:1-7.

PARIZA, M.W., PARK, Y. and COOK, M.E. (2001). The biologically active isomers of conjugated linoleic acid. *Progress in Lipid Research* **40**:283-98.

PENNY, P. (2004). Adding value to pork for producers and consumers: enhancing omega-3 DHA and selenium content of meat. In Nutritional Biotechnology in the Feed and Food Industries, *Proceedings of Alltech's 20th Annual Symposium,* (eds. T.P. Lyons and K.A. Jacques), Nottingham University Press, Nottingham, pp141-147.

PERETZ, A., NEVE, J., DESMEDT, J., DUCHATEAU, J., DRAMAIX, M. and FAMAEY, J.P. (1991). Lymphocyte response is enhanced by supplementation of elderly subjects with selenium-enriched yeast. *American Journal Clinical Nutrition* **53**:1323-1328.

PERETZ, A., NEVE, J., DUCHATEAU, J., and FAMAEY, J.P. (1992). Adjuvant treatment of recent onset rheumatoid arthritis by selenium supplementation: preliminary observations. *British Journal of Rheumatology* **31**:281-286

POPPITT, S.D., MCCORMACK, D. and BUFFENSTEIN, R. (1998). Short-term effects of macronutrient preloads on appetite and energy intake in lean women. *Physiological Behaviour* **64**: 279-285.

PORRINI, M., SANTANGELO, A., CROVETTI, R., RISO, P., TESTOLIN, G. and BLUNDELL, J.E. (1997). Weight, protein, fat, and timing of preloads affect food intake. *Physiological Behaviour* **62**: 563-570.

RAYMAN, M.P. (2002). The argument for increasing selenium intake. *Proceedings of the Nutrition Society*, UK **61**:203-215.

RAYMAN, M.P. (2004). The use of high-selenium yeast to raise selenium status: how does it measure up? *British Journal of Nutrition* **92**:557-73.

ROBINSON, S.M., JACCARD, C., PERSAUD, C., JACKSON, A.A., JEQUIER, E. and SCHUTZ. Y. (1990). Protein turnover and thermogenesis in response to high-protein and high-carbohydrate feeding in men**.** *American Journal of Clinical Nutrition* **52**:72-80**.**

ROSADO, J.L., DIAZ, M., GONZALEZ, I.G., ABRAMS, S.A. and PRECIADO, R. (2005). The addition of milk or yoghurt to a plant-based diet increases zinc bioavailability in women. *Journal of Nutrition* **135**:465-468.

SCHRAUZER, G.N. (2003). The nutritional significance, metabolism and toxicology of selenomethionine. *Adv Food Nutr Res* **47**:73-112.

SCHRAUZER, G. (2004). Selenium in human health – relationship between selenium intake, cancer and viral diseases. Proceedings of the 1st Alltech Latin American Symposium on Biotechnology and Nutrition in Animal Feeds, Curitiba, Brazil, pp. 44-112.

SCRIMSHAW, N.S. and GUZMAN, M.A. (1968). Diet and atherosclerosis. *Lab Invest* **18**:623-8.

SIMS, E.A.H. and DANFORTH, E. (1987). Expenditure and storage of energy in man. *J Clin Invest* **79**:1019-25.

SKOV, A.R., TOUBRO, S., RONN, B., HOLM, L. and ASTRUP, A. (1999). Randomised trial on protein vs carbohydrate in a libitum fat reduced diet for the treatment of obesity. *Int. J. Obes. Relat. Metab. Disord.* **23**:528-536.

SPILBURG, CA., GOLDBERG, AC., MCGILL, JB., STENSON, WF., RACETTE, SB., BATEMAN, J., MCPHERSON, T.B. and OSTLUND, R.E Jr. (2003). Fat-free foods supplemented with soy stanol-lecithin powder reduce cholesterol absorption and LDL cholesterol. *J Am Diet Assoc* **103**:577-581.

STRATTON, M.S., DILLEY, T. and AHMANN, F. (2003). Molecular mechanism of selenium in chemoprevention of prostate cancer. In Nutritional Biotechnology in the Feed and Food Industries, *Proceedings of Alltech's 19th Annual Symposium*, (eds. T.P. Lyons and K.A. Jacques), Nottingham University Press, Nottingham, pp31-50.

STUBBS, R.J., VAN WYK, M.C., JOHNSTONE, A.M. and HARBRON, C.G. (1997). Breakfasts high in protein, fat or carbohydrate: effect on within-day appetite and energy balance. *Eur. J. Clin. Nutr* **50**:409-417.

UHE, A.M., COLLIER, G.R. and O'DEA, K. (1992). A comparison of the effects of beef, chicken and fish protein on satiety and amino acid profiles in lean male subjects. *Journal of Nutrition* **122**:467-472.

UTHUS, E.O., YOKOI, K. and DAVIS, C.D. (2002). Selenium deficiency in Fisher-344 rats decreases plasma and tissue homocysteine concentrations and alters plasma homocysteine and cysteine redox status. *Journal of Nutrition* **132**:1122-1128.

VAN DEN BRANDT, P.A., ZEEGERS, M.P., BODE, P. and GOLDBOHM, R.A. (2003). Toenail selenium levels and the subsequent risk of prostate cancer: a prospective cohort study. *Cancer Epidemiol Biomarkers Prev* **12**:866-871.

WEI, W.Q., ABNET, C.C., QIAO, Y.L., DAWSEY, S.M., DONG, Z.W., SUN, X.D., FAN, J.H., GUNTER, E.W., TAYLOR, P.R. and MARK. S.D. (2004). Prospective study of serum selenium concentrations and esophageal and gastric cardia cancer, heart disease, stroke and total death. *Amer. J. Clin. Nutr***79**:80-85.

WHIGHAM, L.D., COOK, M.E. and ATKINSON, R.L. (2000). Conjugated linoleic acid: implications for human health. *Pharm. Res* **42**:503-510.

YOSHIZAWA, K., WILLETT, W.C., MORRIS, S.J., STAMPFER, M.J., SPIEGELMAN, D., RIMM, E.B. and GIOVANNUCCI, E. (1998). Study of prediagnostic selenium level in toenails and the risk of advanced prostate cancer. *J Natl Cancer Inst* **90**:1219-1224.

YU, M-W., HORNG, I.S., CHIANG, Y.C., LIAW, Y.F. and CHEN, C.J. (1999). Plasma selenium levels and the risk of hepatocellular carcinoma among men with chronic hepatitis virus infection. *American Journal Epidemiology* **150**:367-374.

Digestibility of amino acids and energy in mung bean, chickpea and lablab when fed to pigs

D.N. Singh, R.J. van Barneveld* and Y.J. Ru**

Department of Primary Industries and Fisheries, Qld. 4163. *Barneveld Nutrition Pty Ltd, Qld. 4280. **Danisco Animal Nutrition, Singapore.

While opportunities to use protein sources from non-conventional plants in pig diets arise occasionally, without information on the nutrient digestibility of these grains it is difficult to formulate cost-effective diets. For example, there is limited information on nutrient digestibility of mung bean (MB), chickpea (CP) and lablab (LAB) when fed to pigs. The objectives of this experiment were: to determine the digestible energy (DE) content of CP, MB and LAB and to measure the apparent and true ileal digestibility (AID and TID) of dry matter (DM), nitrogen (N) and amino acids (AA) of these grains. Chickpea comprised 400 g/kg and MB and LAB 360 g/kg of the sugar/starch-based diets and was the sole source of the 115 g/kg of protein in each diet. Celite® was added (20 g/kg) as an acid-insoluble ash marker. The AID of AA and the DE was determined using five Large White male pigs (35-40 kg) fitted with simple T-piece cannulas, as described by van Barneveld *et al.* (1994). Diet allocations were based on a 5 X 5 Latin square design. The additional two treatments comprised soybean meal (SBM), which acted as control and a diet containing enzymically hydrolysed casein (EHC) was included to calculate endogenous losses. The data were analysed using a general linear model in SYSTAT. Means were separated by least significant differences.

Table 1. AID and TID coefficients of energy and some amino acids and of faecal DE for growing pigs fed diets containing soybean meal, chickpeas, mung bean and lablab (proportion of total).

Nutrient	**Soybean meal**		Chickpea		Mung bean		Lablab	
	AID	TID	AID	TID	AID	TID	AID	TID
Energy	0.86^{a}	0.93^{a}	0.82^{b}	0.89^{a}	0.84^{ab}	0.91^{a}	0.72^{c}	0.79^{c}
Protein	0.77^{a}	0.83^{a}	0.71^{ab}	0.79^{a}	0.68^{b}	0.76^{a}	0.18^{c}	0.27^{b}
Cystine	0.70^{a}	0.77^{a}	0.59^{a}	0.67^{ab}	0.43^{b}	0.57^{b}	0.06^{c}	0.05^{c}
Methionine	0.91^{abc}	0.97^{a}	0.90^{ab}	0.99^{a}	0.83^{b}	0.95^{a}	0.35^{c}	0.53^{b}
Threonine	0.77^{a}	0.88^{a}	0.69^{b}	0.85^{a}	0.68^{b}	0.85^{a}	0.30^{c}	0.45^{b}
Alanine	0.72^{a}	0.81^{a}	0.68^{b}	0.80^{ab}	0.56^{c}	0.67^{b}	0.16^{d}	0.17^{c}
Valine	0.86^{a}	0.93^{a}	0.79^{b}	0.88^{a}	0.80^{ab}	0.87^{a}	0.43^{c}	0.51^{b}
Isoleucine	0.81^{a}	0.91^{a}	0.71^{b}	0.85^{a}	0.71^{b}	0.83^{a}	0.29^{c}	0.42^{b}
Lysine	0.88^{a}	0.93^{a}	0.88^{a}	0.92^{a}	0.87^{a}	0.91^{a}	0.64^{b}	0.69^{b}
DE (MJ/kg as fed)	15.32^{a}		14.79^{b}		15.01^{c}		14.50^{d}	

Values in a row with different superscripts in columns AID and TID differ significantly ($P<0.05$)

The DE of all the test legumes was significantly lower than SBM. Mung bean had a significantly higher DE content than CP, which was significantly higher than LAB. The AID and TID of LAB was significantly inferior to the SBM control and the other test legumes. The AID of all AA in CP and MB were comparable to SBM, with the exception of threonine, alanine, valine, isoleucine and tyrosine. AID of cystine, alanine and arginine were significantly higher in CP compared with MB. Many of the differences evident between the AID of AA in SBM, CP and MB were not evident when TID was determined, with only cystine and alanine significantly inferior in MB compared with CP and SBM. Chickpea and MB hold significant potential as alternative protein and energy sources. The data suggest that there are limited influences on protein digestion from any anti-nutritional factors that may be present in these legumes. In contrast, it appears that lablab contains anti-nutritional factors that significantly impede protein and amino acid digestion.

Supported in part by Australian Pork Ltd.

References

van BARNEVELD,R.J., BATTERHAM, THE LATE E.S and NORTON, BW. (1994). *British Journal of Nutrition* **72**:221-241.

Australian Pork

Digestible and net energy contents of two types of extruded rice for weaner and grower pigs

J.C. Kim*, M.M.J.A. Rijnen**, B.P. Mullan*** and J.R. Pluske*

Murdoch University, Murdoch, WA 6155. **Hendrix UTD B.V., Nutreco, The Netherlands. *** Department of Agriculture, South Perth, WA 6151.

Published data regarding the energy content of rice for pigs are scarce, apart from the NRC's (1998) estimate of 14.9 MJ of digestible energy (DE)/kg for white rice. Extruded rice most likely has a higher energy value for pigs than white rice because of its high digestible starch content. Establishing the energy content of extruded rice is needed so diets can be formulated accurately for pigs of different weights. The purpose of this study was to examine the DE and calculated net energy (NE) contents of two types of extruded rice in weaner and grower pigs. The hypotheses tested were 1) rice with a lower amylose-to-amylopectin ratio will have a higher DE and NE content than a variety with a higher amylose-to-amylopectin ratio, and 2) heavier pigs will extract more energy from rice than lighter pigs.

Thirty-two male pigs (Large White x Landrace, 16 pigs per body weight group) were used in a 2 x 2 factorial arrangement of treatments, with the respective factors being a) two rice types (Amaroo medium-grain, lower amylose-to-amylopectin ratio *vs.* Doongara long-grain, higher amylose-to-amylopectin ratio) and b) two body weight groups (weaner and grower). The average body weights for each group were (mean ±SEM) 7.9 (±0.16) kg and 55.4 (±3.10) kg, respectively. The pigs were offered their respective experimental diet at a rate of 5% and 3.75% of body weight for weaner and grower pigs (about 90% of *ad libitum*), respectively. The diet was formulated to contain 859 g rice/kg, 15.3 MJ DE/kg and 0.6 g lysine/MJ DE. Other ingredients used were meat and bone meal, canola meal, canola oil and vitamin/mineral supplements. Titanium dioxide (TiO_2) was added as an inert marker for apparent digestibility estimation. Pigs were adapted to their experimental diets for seven days. Faecal grab samples were then collected at 0800, 1000, 1200, 1400, 1600 h for the next three days, dried and sub-sampled for subsequent analyses. The NE contents of rice were determined using published equations from the Dutch CVB tables and from INRA (Sauvant *et al.*, 2004). The ANOVA analysis of Statview 5.0 for Windows (SAS Inc.) was used for statistical analysis.

Table 1. Main effects of types of extruded rice and body weight of pigs on the digestible energy (DE MJ/kg as is) and net energy (NE MJ/kg as is) contents.

Treatment	Energy digestibility (%)	DE	NE-CVB[1]	NE-INRA[2]
Medium grain rice	91.7[b]	15.1[b]	11.1	11.9
Long grain rice	91.5[b]	15.1[b]	11.2	11.8
Weaner pigs	90.2[a]	14.8[a]	-	11.8[§]
Grower pigs	92.9[c]	15.4[c]	-	12.1[§]
s.e.m.	0.30	0.06		

[1,2]Calculated from CVB and INRA formulae (Sauvant *et al.*, 2004);
[abc]Values within a column without a common superscript are significantly different (P<0.05);
[§]Calculated using determined DE value and INRA equation.

The mean (±SEM) gross energy digestibility and DE content (MJ/kg air-dry basis) of rice were 91.6% (0.30) and 15.1 (0.06), respectively. Variety had no influence on the DE content. Weaner pigs extracted less energy from a given rice than grower pigs (0.6 MJ, P<0.001). The interaction between variety and body weight was not significant. Estimation of the NE content of the two rice types using CVB and INRA formulae showed a mean NE content of 11.5 MJ/kg, although this differed according to whether CVB or INRA equations were used. This study suggests that the DE and NE content of rice might be higher than originally thought (compared to 14.9 MJ and 9.60 MJ/kg, respectively; NRC, 1998), and weaner pigs extract 0.6 MJ/kg less DE from a given rice type than grower pigs.

The Rice Sub-program of the Rural Industries Research and Development Corporation is thanked for funding.

References

NRC (1998). Nutrient requirements of swine (10th Ed.). National Academy Press, Washington, DC.

SAUVANT, D., PEREZ, J.-M. and TRAN, G. (2004). 'Tables of composition and nutritional value of feed materials' (Wageningen Academic Publishers, The Netherlands).

Resistant starch content in different types of rice in response to a range of cooking and cooling conditions

J.C. Kim*, B.P. Mullan**, D.J. Hampson* and J.R. Pluske*

Murdoch University, Murdoch, WA 6155; ** Department of Agriculture, South Perth, WA 6151.

Resistant starch (RS) is fermented by micro-biota in the gastrointestinal tract and is resistant to enzymatic digestion in the small intestine of pigs. Cooked white rice could potentially replace more traditional cereals such as wheat and barley in the diets of young pigs (Pluske *et al*., 2003), particularly when the incidence of post-weaning colibacillosis is high (Pluske *et al*., 2002). However, to be used commercially, heat processing of the rice would probably be necessary for starch gelatinisation. Furthermore, selection of rice type and the appropriate processing and cooking conditions would be required to optimise starch gelatinisation. This *in vitro* study was carried out to test changes in the RS content of rice types differing in their amylose-to-amylopectin content following various processing and cooling combinations. The hypotheses tested were: 1) rice variety will influence the RS content; 2) extrusion cooking will decrease RS content and; 3) cooking and cooling will increase the RS content.

In vitro studies were carried out to examine: 1) the effect of rice type [variety Amaroo (lower amylose, medium-grain), Doongara (higher amylose, long-grain) and parboiled rice (long-grain)]; 2) the effect of extrusion (single-screw extruder) and; 3) the effects of rice-to-water ratio (1:1 or 1:2 w/w) during autoclaving and refrigeration after autoclaving (freshly dried or dried after refrigeration for 24 hrs at 4^{o} C), on the RS content of rice. All samples of rice were cooked in an autoclave (121^{o} C, 20 min steaming plus 20 min drying). The RS contents were determined using the Megazyme RS kit (Megazyme, Ireland). The ANOVA analysis of Statview 5.0 for Windows (SAS Inc.) was used for statistical analysis.

Table 1. Resistant starch content (g 100g^{-1} DM) of three rice samples in response to extrusion, the rice: water ratio during autoclaving and cooling conditions following autoclaving[1].

Treatment		Raw	Extruded	Autoclaved[3]
Rice	Amaroo	0.10^{a}	0.13	0.60^{a}
	Doongara	0.42^{b}	0.16	1.42^{b}
	Parboiled	0.72^{c}	-	3.74^{d}
Rice:water ratio	1:1			1.52^{b}
	1:2			2.31^{c}
Refrigeration [2]	Fresh			1.54^{b}
	Refrigerated			2.29^{c}
SEM		0.05	0.02	0.14

[1]Values from mean of 10 observations for each treatment combinations were used. [abc]Values within a column with different superscripts are significantly different (P<0.05). [2]Fresh: autoclaved and dried at 70 °C, 48 hrs; Refrigerated: autoclaved and stored at 4 °C, 24 hrs and dried at 70 °C, 48 hrs. [3]Values are main effect means.

The higher-amylose, long-grain variety Doongara had four times as much RS and the parboiled rice had seven times as much RS as the lower-amylose variety Amaroo (P<0.001). Extrusion decreased the RS content only in the higher RS rice Doongara (P=0.02). The RS contents were significantly different between rice types following autoclaving (P<0.001) but followed the order of the raw RS contents. Increasing the rice-to-water ratio (1:2) and refrigeration (24 hours at ^{o} C) after autoclaving significantly increased the RS content (P<0.001). Rice with higher amylose content, cooking with more water and cooling after autoclaving increased the RS content, while extrusion decreased the RS content.

The Rice Sub-program of the Rural Industries Research and Development Corporation is thanked for funding.

References

PLUSKE, J.R., BLACK, B., PETHICK, D.W., MULLAN, B.P. and HAMPSON, D.J. (2003). *Animal Feed Science and Technology* **107**:129-142.

PLUSKE, J.R., PETHICK, D.W., HOPWOOD, D.E. and HAMPSON, D.J. (2002). *Nutrition Research Review* **15**:333-371.

Net energy defines growth and carcass quality better than digestible energy

D.J. Cadogan*, C. Smith** and D.J.Henman**

*Feedworks, Eagle Farm, Qld. 4009. **QAF Meat Industries, Corowa, NSW 2646.

Most Australian nutritionists formulate pig diets using digestible energy (DE). Net energy (NE) is the energy available for lean tissue and fat deposition and is calculated by subtracting the energy lost from urine, methane and the heat production from DE. Equations are available to calculate NE from known dietary nutrients (Noblet *et al*., 1994).

To test the hypothesis that growth performance and carcass characteristics are more responsive to NE (calculated using equations) than DE, an equal number of entire males and castrate males, 96 in total, were allocated to four treatments at 64.7±3.6 kg live weight. The treatments consisted of two levels of DE and four decreasing levels of NE. The diets were formulated to an available lysine of 0.54 g/MJ DE, while other essential amino acids were balanced to the ideal protein ratio. Pigs were housed in individual pens and offered diets *ad libitum* for 42 days. Average daily gain (ADG), average daily intake (ADI) and feed conversion ratio (FCR) were measured weekly. On day 43, pigs were killed and carcass weight (CWT), carcass feed conversion ratio (cFCR) and P2 back fat were recorded.

Table 1. Effects of net energy and digestible energy on measured growth and carcass parameters.

DE (MJ/kg)	14.0		12.5			Significance[1]
NE (MJ/kg)	10.4	9.6	9.2	8.6	s.e.m.	
ADG	1063	1040	1062	991	12.9	NE*, Sex*
ADI	2.893	2.889	3.104	3.059	0.055	DE**, Sex***
FCR	2.81	2.78	2.93	3.06	0.046	DE***, NE*, Sex***
*c*FCR	3.03	3.32	3.44	3.69	0.049	DE***, NE***,Sex***
CWT	85.3	81.8	83.0	79.7	0.569	DE*, NE***, Sex***
P2	13.4	11.4	11.8	11.0	0.293	NE*, Sex***

[1]*P<0.05, **P<0.01, ***P<0.001.

Entire males exhibited a superior FCR, *c*FCR and P2, while their castrated counterparts produced a higher ADI, ADG and CWT (P<0.05). Increasing DE from 12.5 to 14 MJ/kg DE decreased ADI (P<0.05) and improved FCR, *c*FCR, and CWT (P<0.05), but had no effect on ADG or P2. The decreasing levels of NE had no effect on ADI, although all other growth and carcass measurements were significantly influenced. No interactions were observed. Analysing NE and DE on a daily intake basis, NE intake was more strongly related to all growth and carcass parameters, except FCR, than daily DE intake. The present study suggests that NE provides a stronger relationship to carcass lean and fat growth.

References

NOBLET, J., FORTUNE, H., SHI, X.S. and DUBIOS, S. (1994). *Journal of Animal Science* **72**:648-657.

Dietary lecithin improves the compression properties of pork from the *semitendinousus* muscle

D.N. D'Souza[*†], B.P. Mullan[*], J. McLeish[**], D.W. Pethick[***] and F.R. Dunshea[****]

*Department of Agriculture, South Perth, WA 6151. **ADM, Bondi Junction, NSW 2022, ***Murdoch University, Murdoch, WA 6150,****Department of Primary Industries, Werribee, Vic. 3030. †Current address - Alltech Biotechnology PL, Dandenong South, VicC. 3175.

Consumers rate tenderness and texture as important eating quality attributes of pork. Pork tenderness and texture are affected by the collagen and myofibril protein components of pork. For example, cross-linking and temporal pattern of thickening of collagen fibrils and the subsequent decline in heat solubility can result in tough and chewy pork (Fang *et al*., 1999). In this experiment we hypothesised that the phospholipid, polyenylphosphatidylcho line (PPC), present in lecithin extracted from soy beans would decrease the cross-linking of collagen fibrils (Lieber *et al.,*1990) and that this would improve the tenderness and texture of pork. The aim was to determine the effect of dietary lecithin supplementation during the grower and finisher growth phases on the compression properties (measure of texture) of pork.

Twenty crossbred (Large White x Landrace x Duroc) female pigs were used with the main nutritional treatments being: 1) control (pigs fed commercial grower and finisher phase diet) and; 2) lecithin (3g/kg) supplementation during the grower and finisher growth phase (soy bean lecithin, ADM Australia Pty Ltd). The pigs were housed individually and had *ad libitum* access to feed and water via nipple drinkers. The pigs were weighed weekly and total feed intake recorded. At about 23 weeks (105 kg ± 2 kg) the pigs were transported to a commercial abattoir and slaughtered according to standard commercial procedures. Twenty-four hours after slaughter the s*emintendinosus muscle* was removed for muscle compression tests (hardness – peak force required to achieve initial penetration, cohesiveness – increase in proportion of work required for a second penetration compared to that required for the first penetration and chewiness – the product of hardness and cohesiveness) (Channon *et al*., 2001). All data were analysed by ANOVA.

Table 1. The effect of dietary lecithin supplementation on the growth performance, carcass quality and *semitendinosus* compression properties of female pigs housed individually.

	Control	Lecithin (3g/kg)	lsd	P-values
Start live weight – Day 68 (kg)	25.5	25.9	2.60	0.786
End live weight – Day 166 (kg)	106.7	104.9	11.1	0.738
ADG (kg) day 68-166	0.828	0.811	0.108	0.734
VFI (kg/d) day 68-166	2.42	2.41	0.242	0.931
FCR day 68-166	2.93	3.03	0.306	0.534
Carcass weight (kg)	72.1	72.9	9.46	0.866
Back fat depth - P2 (mm)	14.9	14.4	3.64	0.795
% Cook loss	31.4	28.7	3.16	0.090
Compression test: Hardness (kg)	3.22	2.80	0.321	0.011
Cohesiveness	0.385	0.381	0.015	0.569
Chewiness	1.26	1.07	0.155	0.021

There was no significant difference in live weight, average daily gain, feed intake, feed conversion ratio, carcass weight and back fat depth in pigs fed the control or lecithin supplemented diet (P>0.05). Although not significant (P = 0.09), pigs fed the lecithin-supplemented diet tended to have lower percentage of cook loss than pigs fed the control diet. The compression tests indicated that pigs fed the lecithin-supplemented diet had significantly lower (P<0.05) hardness and chewiness values for the *semitendinosus* muscle than pigs fed the control diet. Dietary lecithin supplementation did not have a detrimental effect on growth performance or carcass quality and significantly reduced the chewiness and hardness of pork. Lecithin also had the potential to improve the tenderness of pork. The lack of effect of lecithin supplementation on cohesiveness of pork requires further investigation.

References

FANG, S.H., NISHIMURA, T. and TAKAHASHI, K. (1999). *Journal of Animal Science* **77:**120-130.

LIEBER, C. S., DECARLI, L. M., KIM, C. I. and LEO, M. A. (1990). *Hepatology,* **12**:1390-1398

CHANNON, H.A., REYNOLDS, J. and BAUD, S.R. (2001). Pig Research Report 1385. Australian Pork Limited , Canberra.

Dietary lecithin improves the compression properties of pork from the *longissimus thoracis* muscle

B.L. Edmunds, I.H. Williams, B.P. Mullan*, J. McLeish**, D.W. Pethick***, F.R Dunshea**** and D.N.D'Souza*†

University of Western Australia, Crawley, WA 6009. *Department of Agriculture, South Perth, WA 6151. **ADM, Bondi Junction, NSW 2022, ***Murdoch University, Murdoch, WA 6150, ****Department of Primary Industries, Werribee, Vic. 3030. †Current address - Alltech Biotechnology PL, Dandenong, Vic. 3175.

D'Souza *et al.* (2005) showed that dietary lecithin could improve the eating quality of pork from the S*emitendinosus* muscle by reducing chewiness and hardness. It has been hypothesised that the phospholipid, polyenylphosphatidylcho line (PPC), present in lecithin extracted from soy beans may decrease the cross-linking of collagen fibrils (Lieber *et al.,* 1990) and reduce the chewiness and hardness of pork. However muscles vary in collagen content and in the extent of collagen cross-linking. The aim of this experiment was to investigate the effect of lecithin supplementation at varying doses on the pork quality in the *Longissimus thoracis* and *Semitendinosus* muscles.

Forty crossbred (Large White x Landrace x Duroc) female pigs were used. Pigs were fed either: 1) a commercial grower and finisher phase diet (Control); 2) control diet supplemented with 3 g lecithin/kg of feed during the grower and finisher growth phase (soybean lecithin, ADM Australia Pty Ltd); 3) control diet supplemented with 15 g lecithin/kg feed during the grower and finisher growth phase or; 4) control diet supplemented with 75 g lecithin/kg of feed during the grower and finisher growth phase. The pigs were housed individually and had *ad libitum* access to feed and water via nipple drinkers. The pigs were slaughtered at 23 weeks of age (100 kg ± 1 kg) according to standard commercial procedures. Twenty-four hours after slaughter, the *Longissimus thoracis* muscle was removed for objective assessment of pork quality (muscle pH, colour, surface exudate, shear force and compression tests (Channon *et al.*, 2002). All data were analysed by ANOVA. Supplementing lecithin at 15 g/kg or 75 g/kg feed reduced the hardness and chewiness of pork significantly compared to the control treatment (Table 1). There was no effect of lecithin supplementation ($P>0.05$) on muscle cohesiveness, pH, colour, surface exudate, cook loss percentage or shear force.

Table 1: The effect of lecithin supplementation on the objective pork quality of the *Longissimus thoracis* muscle twenty four hours after slaughter.

	Control	Lecithin 3g/kg	Lecithin 15g/kg	Lecithin 75g/kg	lsd.	P - value
pH (24 hr)	5.45	5.47	5.50	5.46	0.099	0.806
L*	50.4	50.3	49.5	51.9	3.50	0.549
a*	6.70	6.84	5.50	6.46	1.23	0.139
b*	3.85	4.03	3.06	4.20	1.15	0.223
Surface exudate (mg)	68.4	59.4	54.0	68.7	23.5	0.523
Cook loss (%)	30.6	30.7	30.5	31.0	2.24	0.965
Shear force (kg)	5.80	6.06	5.76	6.01	1.24	0.947
Hardness (kg)	4.93	4.65	4.32	4.28	0.337	<0.001
Cohesiveness	0.404	0.404	0.398	0.402	0.016	0.819
Chewiness	1.99	1.88	1.73	1.72	0.158	0.002

A dose of 15 g lecithin/kg feed was considered to be the most cost-effective commercial level required to improve the compression characteristics of pork. The impacts of reduced chewiness and hardness on the sensory properties (juiciness, tenderness, flavour and overall acceptability) of pork due to lecithin supplementation need to be quantified. The results from this experiment and D'Souza *et al.* (2005) indicated that varying amounts of lecithin were required to improve the pork compression characteristics of different muscles and that this was most likely dependant on the collagen levels and extent of cross-linking. The lack of effect of lecithin supplementation on cohesiveness of pork requires further investigation.

References

D'SOUZA, D.N., MULLAN, B.P., MCLEISH, J. and DUNSHEA, F.R. (2005). In 'Manipulating Pig Production X', p. 264. ed J.E. Paterson. (Australasian Pig Science Association, Werribee).

LIEBER, C. S., DECARLI, L. M., KIM, C. I. and LEO, M. A. (1990). *Hepatology* **12**:1390-1398

CHANNON, H.A., REYNOLDS, J. and BAUD, S. (2001). Identifying pathways to ensure acceptable eating quality of pork. Final Report, Australian Pork Limited, Canberra.

Apparent digestibility of feed containing alternative fat sources to animal fat in weaners and growers

C. Lauridsen*, T.B. Christensen*, S.K. Jensen* and U. Halekoh**

*Danish Institute of Agricultural Sciences, Department of Animal Health, Welfare and Nutrition, Denmark.
**Department of Genetics and Biotechnology, Tjele, Denmark.

The BSE crisis in the European Union has reduced the amount of animal fat available for animal feeds and alternative fat sources are of increasing importance. The chemical composition of dietary fats differs widely between different sources and this may affect the digestibility and hence nutritional value of dietary fats (Jørgensen and Fernandez, 2000). To our knowledge, no recent studies have considered the digestibility of different fat sources in pigs using the same individuals throughout the weaning and growing periods. The objective of the present study was to compare the apparent fat digestibility (AFD) of feeds made with either vegetable or animal fats. The hypothesis was that the digestibility of fat would differ between sources.

Forty eight piglets (Danish Landrace x Danish Yorkshire x Duroc) from eight litters with an initial weight of 8.3 ± 1.1 kg were obtained at weaning (28 days of age) and moved to metabolism cages for two days of adaptation. Three consecutive balance periods, each of five days, were then conducted during the weaning phase and again during the growing phase (50 kg live weight). The experiment used six different fat sources added at a level of 5 % 1) animal fat (of swine, AF), 2) palm oil (PO), 3) palm oil mix (primarily palm oil fatty acid distillates, PFAD), 4) vegetable oil mix (50 % palm oil mix + 50 % palm oil, VMIX), 5) coconut oil (CO) and 6) rapeseed oil (RO). During the growing phase, coconut oil was replaced with a fat blend (FB) (65 % palm oil, + 15 % palm oil mix + 20 % mixed fatty acids). All vegetable oil sources were obtained from DLG (Dansk Landbrugs Grovvareselskab, Copenhagen, Denmark), and animal fat from DAKA (Randers, Denmark). Before acid hydrolysis, fat in diets and in faeces was extracted after modification of Bligh and Dyer (1959). Because each pig received one type of dietary fat during the entire experimental period, the data for fat digestibility were analysed according to a statistical model. All interactions were significant at the 5% level.

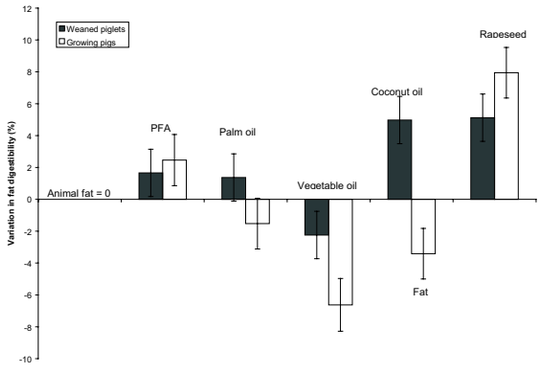


Figure 1. *Difference in apparent fat digestibility (AFD) of dietary fat sources in weaned piglets and growing pigs relative to AFD of animal fat. Animal fat had an AFD of 75.1 (SE=1.1) % (weaning) and 65.6 (SE=1.1) % (growing).*

Surprisingly, weaner pigs digested fats better than growing pigs (Figure 1). RO diets had the highest AFD of all dietary fat sources in both weaned piglets (P< 0.001, 5.1 % units better than AF, Figure 1) and growing pigs (P<0.001, 7.9 % units better than AF). The CO diet had a higher AFD (5 % units) than AF in weaned piglets (P=0.001). The FB had a lower AFD (3.4 % units) in growing pigs than the AF (P=0.034). The VMIX diet showed the lowest AFD - 2.2% lower than for AF for weaned piglets (P=0.134) and 6.6% lower for growing pigs (P<0.001). The PFAD diets resulted in a higher AFD (P=0.128) in both weaned piglets and growing pigs than the AF diet. Diets containing PO did not differ in AFD compared to diets with AF (P=0.33). The AFD was generally lower during the growing phase than during the weaning phase. It was concluded that relative to AF, RO and CO (for weaners) were good fat alternatives, while PFAD and PO were similar to the AFD of AF, and FB and VMIX were not suitable alternative fat sources. However, to assess the suitability of the fat sources for swine nutrition other factors such as palatability of feed and cost effectiveness must also be considered.

References

JØRGENSEN, H. and FERNANDEZ, J. (2000). *Animal Science* **50**:129-136.

BLIGH, E.G. and DYER, W.J. (1959). *Canadian Journal of Biochemistry and Physiology* **37**:911-917.

High protein diets containing whey protein concentrate and soy protein isolate improve insulin resistance and reduce weight and fat gain in obese minipigs

J. M. Ferrari*,**, E. Ostrowska*, M. Muralitharan**, B.G. Tatham*, I. McCauley*, M.L. Cox*, P.J. Eason*, D.J. Kerton* and F.R. Dunshea*,**

*Department of Primary Industries, Werribee, Vic. 3030. **Deakin University, Geelong, Vic. 3217.

Milk contains a mixture of proteins, each having unique attributes for nutritional, biological and human food ingredient applications (Smithers *et al.,* 1996). The major proteins present in milk include β-lactalbumin, α-lactoglobulin, immunoglobulin, bovine serum albumin, and the caseins: κ-casein, β-casein, and the α-caseins (Etzel 2004). In addition, whey contains glycomacropeptide (GMP) that is cleaved from κ-casein by chymosin to initiate precipitation of the caseins forming curd. When casein is removed from whole milk, liquid whey remains, and a number of different proprietary processes exist to further treat or purify whey protein resulting in various whey protein isolates, some of which may be rich in specific bioactive peptides such as GMP. Recently, there has been evidence to support a role for dietary proteins in the regulation of food intake and weight maintenance (Anderson and Moore, 2004). In addition, some protein sources contain specific peptides that may elicit direct effect upon satiety. For example, GMP is thought to have a satiating effect and there are whey protein isolates (WPI) that are relatively high in GMP. Therefore, the following study was done to determine if high protein diets, including a WPI rich in GMP, have any effect on feed intake, body weight and other indices of obesity in minipigs. The minipig is an excellent model for obesity as it contains 50% body fat and displays insulin resistance (Dunshea *et al.,* 2005).

Sixteen obese adult female minipigs (133 kg, 50% body fat) were allocated randomly to a 2x2 factorial design with the respective factors being source of protein (WPI (NaturaPro MG2460, MG Nutritionals, Brunswick, Victoria) *vs.* soy protein isolate (SPI) (Profarm 974, ADM, Palm Beach, Queensland)) or level of dietary protein (11: (LP) *vs.* 22% (HP) CP). The WPI contained 46, 30, and 8% β-lactalbumin, GMP and α-lactoglobulin, respectively. After consuming their respective diets *ad libitum* for 10 weeks the surgically prepared pigs were infused intravenously with insulin at 0.6 and 6.0 mU/(kg.min) and blood glucose and amino acids clamped at pre-infusion values by simultaneous infusions of dextrose (50% w/v) and a parenteral amino acid mix (10% w/v), respectively. Composition of the ham region was determined by dual energy X-ray absorptiometry at 0, 4 and 8 weeks (Suster *et al.,* 2003).

Feed intake was lower in pigs fed the HP diet (2070 *vs.* 2352 g/d, P<0.001), particularly in pigs fed WPC (1951 v. 2408 g/d) as indicated by an interaction (P=0.027) between source and level. Pigs consuming the HP diet deposited less weight (231 *vs.* 382 g/d, P=0.045) and had a lower ratio of fat:lean in the ham (0.70 *vs.* 0.76, P=0.026) at 8 weeks than those fed the LP diet. Protein source had no effect on the amount of dextrose infused to maintain euglycemia (108 *vs.* 115 mL/h P=0.59) but the amount infused was lower in the minipigs fed the LP diet (101 *vs.* 125 mL/h, P=0.048). The amount of dextrose required to maintain glycemia was higher at the higher dose of insulin (114 *vs.* 226 mL/h, P<0.001). Protein source had no effect on the amino acid infusion rate required to maintain plasma lysine concentrations (50 *vs.* 50 mL/h, P=0.98) but the amount infused was lower in pigs fed the LP diet (45 *vs.* 55 mL/h, P=0.030). The amino acid infusion rate was higher at the higher dose of insulin (47 *vs.* 103 mL/h, P<0.001). In conclusion, a HP diet reduced feed intake, weight gain and fat deposition and reduced insulin resistance in obese minipigs. The HP diet containing WPI that was enriched in GMP had the greatest effect upon feed intake and weight gain.

References

ANDERSON, G.H. and MOORE, S.E. (2004). *Journal of Nutrition* **134**: 974S–979S.

DUNSHEA, F.R., FERRARI, J.M., KETSES, C., OSTROWSKA, E., MURALITHARAN, M., TATHAM, B.G., MCCAULEY, I., COX, M.L., EASON, P.J. and KERTON, D.J. (2005). *FASEB Journal* **19:** 582.8.

ETZEL, M.R. (2004) *Journal of Nutrition* **134:** 996S–1002S.

SMITHERS, G.W., BALLARD, F.J., COPELAND, A.D., DE SILVA, K.J., DIONYSIUS, D.A., FRANCIS, G.L., GODDARD, C., GRIEVE, P.A., MCINTOSH, G.H., MITCHELL, I.R., PEARCE, R.J. and REGESTER, G.O. (1996). *Journal of Dairy Science* **79:** 1454–1459.

SUSTER, D., LEURY, B.J., OSTROWSKA, E., BUTLER, K.L., KERTON, D.J., WARK, J.D. and DUNSHEA, F.R. (2003). *Livestock Production Science* **84**: 231-242.

Effect of conventional and deep litter housing on pig growth performance and carcass characteristics

M. Trezona*, B.P. Mullan*, J.R. Pluske**, D.W. Pethick** and D.N. D'Souza*

* Department of Agriculture, South Perth, WA 6151. ** Murdoch University, Murdoch, WA 6150.

Anecdotal observations suggest that pigs raised in deep litter systems are fatter than pigs raised in conventional systems. To manage fat deposition in pigs, some producers wean pigs into deep litter and then at about 13 weeks of age move the animals into conventional facilities for finishing. Differences in growth and carcass quality have been reported between pigs raised outdoors and conventionally (Gentry *et al.,* 2002). We would expect the physical and thermal differences between conventional and deep litter housing systems to affect the partitioning of energy for lean and fat deposition during growth. In this study we hypothesised that growth performance and carcass composition would differ for pigs housed conventionally or on deep litter.

Conventional housing consisted of commercial, partially slatted pens for weaner and grower-finisher pigs within an insulated building. The deep litter housing consisted of two shelters, which were divided into four pens and bedded with cereal straw. One hundred and sixty female pigs were stratified by live weight (LW) at weaning and allocated randomly to four treatments. The treatments were: C: conventional housing 3-24 weeks of age; DL: deep litter housing 3-24 weeks; DL-C: deep litter housing 3-13 weeks followed by conventional housing 13-24 weeks; and C-DL: conventional housing 3-13 weeks followed by deep litter housing for 13-24 weeks. There were 10 pigs per pen and four pens per treatment. At 13 weeks all groups of pigs moved pens. The same commercial, cereal-based diets were phase-fed to all treatments. At 24 weeks of age pigs were slaughtered at a commercial abattoir. Twenty-four hours after slaughter, one side of the carcass from 12 pigs per treatment, was collected and analysed for fat and lean content (dual energy X-ray absorptiometry). Data were analysed by ANOVA using Genstat v6.

Table 1. Growth performance and carcass characteristics of pigs raised in different housing treatments.

	C	C-DL	DL	DL-C	lsd[1]	P
LW (kg) 3 Weeks	5.5	5.6	5.5	5.5	0.224	0.967
LW (kg) 11 weeks	34.6	34.1	36.3	35.8	1.23	0.007
LW (kg) 13 weeks	46.6	45.9	48.5	48.4	2.06	0.043
LW (kg) 16 weeks	69.3	64.9	69.8	66.2	2.93	0.009
LW (kg) 24 weeks	123.3	117.2	122.8	117.0	4.43	0.016
Carcass weight (kg)	84.5	82.2	85.2	81.0	3.98	0.074
Dressing %	68.5	70.1	69.4	69.2	2.31	0.434
P2 (mm)[2]	19.2	18.8	21.7	18.7	2.62	0.639
Fat % (side)	29.5	21.0	25.3	25.4	6.27	0.074
Lean % (side)	58.8	63.4	60.7	61.1	3.49	0.091

[1]lsd = least significant difference; [2]carcass weight used as co-variate in analysis

Differences in LW were evident by 11 weeks of age (P=0.007) with pigs housed in deep litter being about 2 kg heavier than pigs housed conventionally. After changing pens at 13 weeks of age, LW was higher for pigs that had remained within the same housing system (P<0.05), than pigs moved into the alternate housing treatment. At 24 weeks, C and DL pigs had higher LW at slaughter (P=0.01) and the trend for carcass weight was similar to LW (P= 0.07). There was a trend for C pigs to have higher percent fat (P=0.07) and lower percent lean (P=0.09) than pigs in the other housing treatments.

There were no treatment differences for carcass weight or P2, suggesting that moving pigs from deep litter housing into conventional housing for finishing is not a valid management strategy for reducing carcass fatness. The results indicate that the reduction in growth that occurs when pigs move into a different housing system is evident at slaughter. Under the current carcass payment system based on P2 and carcass weight, one type of housing treatment did not offer an advantage over other treatments. Contrary to the perception that pigs raised in deep litter are fatter, our results indicate that pigs raised entirely on deep litter have lower percent carcass fat than pigs raised entirely in conventional housing. We conclude that pigs raised in deep litter for the entire growing-finishing period produce a similar carcass to pigs weaned into deep litter and finished in conventional housing.

References

GENTRY, J.G, McGLONE, J.J, BLANTON, Jr. J.R. and MILLER, M.F. (2002). *Journal of Animal Science* **80**:1781-1790.

Australian Pork

The effect of conventional and deep litter housing on belly composition of finished Large White x Landrace gilts

M. Trezona*, B.P. Mullan*, J.R. Pluske**, D.W. Pethick** and D.N. D'Souza*

Department of Agriculture, South Perth, WA 6151. ** Murdoch University, Murdoc, WA 6150.

The inherent differences in the physical and thermal environment of conventional and deep-litter housing systems for pigs are likely to affect the deposition of fat and lean tissue during growth. The differences in thermal conductivity between concrete flooring and straw bedding may affect the deposition of fat. In addition, the ingestion of bedding could also affect fat deposition by diluting the energy content of the diet. Bedding consumption is also likely to alter the digestible amino acid and digestible energy ratio of the diet and the amino acid requirements of pigs, and these requirements may not be accounted for in the formulated diet. In turn, this could cause variation in growth and carcass quality (van Barneveld *et al*., 2003). In this study we hypothesised that the tissue distribution in growing pigs would differ between pigs housed on deep litter and pigs housed conventionally. We also expected the impact of housing type on fat deposition and distribution would be more pronounced during the finishing period because the proportion of fat deposited in the total gain increases with age.

One hundred and sixty female pigs were stratified by weight at three weeks of age and allocated randomly to four treatments. The treatments were: C: conventional housing from weaning to slaughter (24 weeks); DL: deep litter housing 3-24 weeks; DL-C: deep litter housing from 3-13 weeks of age followed by conventional housing for 13-24 weeks; and C-DL: conventional housing from 3-13 weeks of age followed by deep litter housing for 13-24 weeks. At 13 weeks all treatment groups moved pens. Pigs were phase-fed the same commercial, cereal-based diets and were slaughtered at a commercial abattoir at 24 weeks of age. Twenty-four hours after slaughter one side of the carcass, from 12 pigs per treatment, was broken down into shoulder, loin, belly and hind-quarter (ham). The primals were analysed for fat and lean content (dual energy X-ray absorptiometry). The data were analysed by ANOVA using Genstat v6.

Table 1. Carcass characteristics of pigs raised under different housing regimes.

	C	C-DL	DL	DL-C	lsd[1]	P
Final LW (kg)	123	117	123	117	4.43	0.016
Dressing %	68.2	70.2	69.5	69.3	2.31	0.434
P2 (mm)[2]	19.2	18.8	21.7	18.7	2.62	0.639
Proportions:						
Fat:Lean Side	0.52	0.34	0.42	0.43	0.14	0.077
Fat:Lean Shoulder	0.29	0.21	0.26	0.27	0.06	0.100
Fat:Lean Loin	0.78	0.56	0.65	0.63	0.22	0.228
Fat:Lean Belly	0.68	0.44	0.54	0.56	0.15	0.032
Fat:Lean Ham	0.31	0.22	0.27	0.26	0.07	0.106

[1]LSD: least significant difference. [2]Carcass weight used as co-variate in analysis

At 24 weeks, pigs that had remained in the same type of housing throughout the experiment had higher live weights at slaughter (P=0.016) but dressing percentage and P2 were not affected by treatment. Pigs in the C-DL and DL treatments had a lower ratio of fat to lean in the belly primal (P=0.032) than C pigs and there was also a very strong trend for the C-DL and DL pigs to have a lower fat:lean ratio in the carcass side (P=0.077). There were no treatment differences in the ratio of fat:lean of the shoulder, loin and ham primals.

These results suggest that there are differences in fat distribution in the carcasses of pigs grown in different housing systems, particularly in regards to the belly primal. The presence of bedding, through its thermal properties and/or via ingestion and altering of dietary energy, protein and fibre intake, may be primary contributing factors to the variation found in the composition of pork bellies.

References

van BARNEVELD R., EDWARDS, A.C. and CHOCT, M. (2003). *Pig Industry News* **87:**13-15.

Australian Pork

Effect of initial chilling rate and dietary magnesium supplements on colour and quality of pork loins

R.D. Warner, D.N.D'Souza*†, R. Tume**, C. D. Hofmeyr and H. A. Channon

Department of Primary Industries, Weribee. Vic. 3030.; *Department of Agriculture, South Perth, WA 6151.; **CSIRO, Food Science Australia, Qld, 4170. †Current address - Alltech Biotechnology PL, Dandenong South, Vic. 3175.

Some pork markets prefer pinkish-red pork and have a particular aversion to any signs of paleness or pale, soft, exudative (PSE) pork. PSE pork is caused by acute stress around slaughter and can be minimised through Mg supplements in the diet for three to five days before slaughter. Fast chilling also has potential to prevent PSE pork as it reduces protein denaturation associated with paleness and excessive exudation. The aim of this experiment was to investigate the effect of feeding magnesium and meat chilling temperature on the quality of pork from heavy carcasses.

Twenty-four female Large White x Landrace female pigs were allocated randomly in a 2x2 ANOVA to two on-farm treatments and carcass sides to two post-slaughter chilling treatments, in a split plot design. Four days before slaughter, groups of pigs were fed one of two rations, control or Mg (control diet with 32 g of Mg Bioplex/pig/day). Twenty four hours before slaughter, feed was removed and pigs were transported 1 km to the abattoir and left in lairage overnight with access to water. Pigs were stunned using 90% CO_2 in air, exsanguinated and each side was then initially allocated randomly to very fast (-5° C, high fan speed) or slow (10° C, slow fan speed) chilling. At six hours after slaughter, all sides were moved to a chiller running at 2° C with a fast fan speed. Forty eight hours after slaughter, the *Longissimus thoracis* (LT) and *spinalis dorsi* (SD) at the anterior end were removed from all sides and surface lightness (L*) and ultimate pH (pHu) were measured. A portion of the LT was vacuum-packed and stored at 2° C for four weeks. Warner-Bratzler shear force (WBSF) was measured at two days (0 weeks ageing) and after four weeks of ageing. Purge was measured as the percent weight loss in vacuum-packed meat samples over time and side shrink was expressed as the difference between the hot and cold weight of a side.

Table 1. The effects of feeding magnesium and initial chilling rate on the meat quality traits in the *longissimus thoracis* (LT) after 0 days ageing (LT0) and 4 weeks ageing (LT4) and in the *spinalis dorsi* (SD) after 0 days ageing.

Diet	Control		Magnesium		P-Value			
Chilling	Fast	Slow	Fast	Slow	s.e.d.	Mg	Chill	Mg.Chill
LT0 -WBSF (kg)	3.41	3.07	3.34	3.59	0.254	0.319	0.768	0.042
LT4 -WBSF (kg)	2.38	2.27	2.38	2.31	0.161	0.880	0.288	0.809
LT4 -Purge (%)	1.06	0.96	0.95	1.05	0.190	0.971	0.982	0.350
LT0 –L*	47.4	46.2	45.1	45.5	0.896	0.084	0.436	0.087
LT0 -pHu	5.65	5.55	5.67	5.54	0.027	0.689	<0.001	0.424
LT4 -L*	52.82	52.22	52.47	52.36	0.803	0.899	0.086	0.228
LT4 -pHu	5.44	5.45	5.44	5.45	0.011	1.000	0.457	0.925
SD –L*	42.6	42.9	41.5	42.3	0.835	0.298	0.083	0.435
SD -pHu	5.83	5.75	5.84	5.76	0.040	0.640	0.004	0.946

The average hot carcass weight was 75.6 kg (SD=5.28) and the average P2 was 16.4 mm (SD=3.61). Sides subjected to a slow rate of chilling had higher shrink than fast chilled sides (2.86% and 2.71%, respectively, SED = 0.053; P=0.009). When Mg was fed to the pigs there was a tendency (P=0.084) for the meat to have a lower L* (darker) in the LT but this difference disappeared after four weeks ageing. Fast chilling caused the SD and the LT to have a higher pHu and a tendency for a lower L* in the SD two days after slaughter than sides chilled slowly for six hours after slaughter. Pigs not fed Mg and warm chilled had more tender meat (lower WBSF) in the LT at two days after slaughter than pigs fed Mg and warm chilled but there was no difference after four weeks of ageing. Feeding Mg for three days before slaughter tended to produce darker loin meat, which would be more acceptable for some markets. Chilling at an initial –5° C air temperature had minimal effect on muscle colour compared to 10° C, although the ultimate pH of the loin and *spinalis dorsi* muscles was 0.1 units higher with fast chilling. Lightness and pHu differences between treatments were absent after four weeks of ageing. Under commercial conditions where pigs slaughtered could be carrying the halothane gene and where electric prodders or other stressful events may be present, it is likely that Mg feeding and fast chilling may result in a more dramatic benefit in reducing the incidence of pale meat than shown here.

Supported in part by Australian Pork Limited.

Effect of initial chilling rate and electrical stimulation on colour and quality of pork loins

R.D. Warner, D.N.D'Souza*†, R. Tume**, C. D. Hofmeyr and H. A. Channon

Department of Primary Industries, Werribee, Vic. 3030; *Department of Agriculture, South Perth, WA 6151; **CSIRO, Food Science Australia, Qld., 4170; †Current address - Alltech Biotechnology PL, Dandenong South, Vic. 3175.

Colour variation of pork remains an important issue for the pork industry in Australia and worldwide. Consumers in some countries prefer to buy pale pork but the pork must also have acceptable tenderness and no 'weep'. Pork colour and tenderness can be enhanced and problems of drip or weep minimised by manipulating the chilling rate applied to a carcass and by using electrical stimulation to influence post-mortem muscle glycolytic rates and pH fall. The aim of this experiment was to investigate the impact of chilling rate and electrical stimulation on the quality traits of pork.

Twenty-four female Large White x Landrace pigs were allocated randomly to treatments in a 2 (stimulation) x 2 (chilling, allocated to sides within carcass) factorial experiment in a split plot design. Pigs were removed from feed about 24 hrs before slaughter, transported 1 km to the abattoir and left in lairage overnight with access to water. Pigs were stunned using 90% CO_2 in air, exsanguinated and then either electrically stimulated with 300 mA for 30 sec at 5 min. after slaughter (ES) or not stimulated (control). Each side within a carcass was then allocated randomly, either to an initial fast (0° C, high fan speed) or slow (7° C, slow fan speed) chilling rate. *Longissimus thoracis* (LT) pH and temperature were measured every hour until 6 hrs after slaughter and then all sides were moved to a 0° C chiller. Twenty four hours after slaughter the LT and *spinalis dorsi* (SD) at the anterior end were removed from all sides and surface lightness (L*), subjective NPPC colour score (1=pale, 6=dark), exudate score (1=none, 5=extreme) and ultimate pH (pHu) were measured. Warner-Bratzler shear force (WBSF) and purge were measured on the LT. Purge was measured as the percent weight loss in vacuum packed meat samples over time (24 hr) and side shrink was expressed as the difference between the hot and cold weight of the side. LT temperature at pH 6.0 was calculated by linearly interpolating the temperature with respect to pH between the first time pH reached a value less than six and the previous measurement. Data were subjected to 2x2 ANOVA.

Table 1: Effect of electrical stimulation and chilling on side shrink, muscle temperature at pH 6.0 and meat quality traits for the *longissimus thoracis* (LT) and *spinalis dorsi* (SD).

Electrical stimulation	Control		ES			P-Value		
Chilling	Fast	Slow	Fast	Slow	s.e.d.	ES	Chill	ES.Chill
Side shrink (%)	2.85	3.01	2.73	3.10	0.105	0.804	<0.001	0.114
LT -Purge (%)	3.50	4.22	3.74	3.95	0.304	0.931	0.063	0.291
LT -WBSF (kg)	3.44	3.29	2.45	2.63	0.194	<0.001	0.886	0.072
LT -Temp. at pH 6.0	5.89	13.04	30.65	35.61	2.38	<0.001	<0.001	0.461
LT -L*	46.38	46.71	51.02	53.49	0.941	<0.001	0.022	0.073
LT -pHu	5.65	5.58	5.60	5.52	0.030	0.032	0.001	0.891
LT -Colour score	3.42	3.17	2.50	2.25	0.254	0.001	0.090	1.000
LT -Exudate Score	2.50	1.92	3.42	3.25	0.278	<0.001	0.083	0.325
SD -L*	42.31	42.60	43.15	43.89	0.477	0.006	0.169	0.544
SD -pHu	5.98	5.83	5.95	5.85	0.045	0.862	0.001	0.456
SD -Colour score	4.33	4.08	4.17	4.17	0.191	0.809	0.177	0.177
SD -Exudate Score	1.25	1.00	1.42	1.25	0.199	0.175	0.143	0.764

The average hot carcass weight was 60.1 kg (s.d=4.06) and the average P2 was 14.8 mm (s.d=2.72) with neither being influenced by treatments (P>0.05). Sides chilled slowly had higher side shrink and purge tended to be higher than those sides chilled at faster rates. The loin muscle was more tender (lower WBSF) for sides that had been electrically stimulated. Electrical stimulation produced carcasses with a higher LT temperature at pH 6, a higher L* (lighter) in the LT and SD, a lower colour score (paler), a lower exudate score and a lower pHu, all in the LT. Slow chilling increased the LT temperature at pH 6.0 and produced LT muscles with a higher L* and a tendency for a lower colour score. Meat which was slow chilled had a lower ultimate pH in both the LT and SD. Electrical stimulation was successful in producing a paler meat colour in both the LT and the SD, without causing any increase in side shrink or purge and was also beneficial in producing more tender pork in the LT. A slow chilling rate also tended to produce slightly paler meat in the LT but not in the SD, however it did increase side shrink and purge.

Supported in part by Australian Pork Limited.

Effect of abattoir and vendor on colour variation of pork from heavy carcasses

H.A. Channon*, R.D. Warner*, R. Tume**, D.N. D'Souza*** and C.D. Hofmeyr*

* Department of Primary Industries, Werribee, Vic. 3030; **CSIRO, Food Science Australia, Qld. 4173; ***Alltech Biotechnology Pty. Ltd., Dandenong South, Vic. 3175.

Consumers are increasingly demanding and often only buying pork that meets specific colour requirements. Pale coloured pork and 'two toning' are two of the major issues that influence customer purchasing decisions for pork in the Australian domestic and export markets. Two toning refers to the darker coloured *M. spinalis dorsi* muscle that overlies the *M. longissimus thoracis* muscles within the collarbutt primal and anterior end of the loin primal. This study aimed to determine the extent of colour variation of pork in two export supply chains. It was carried out over three consecutive days from two abattoirs located in South Australia and Queensland in July 2003. A total of 120 carcasses from three different vendors per abattoir (90-110 kg hot carcass weight, P2 >13 mm) were randomly selected on the slaughter floor. Muscle temperature was measured at two hours after slaughter in the *M. longissimus thoracis*. Fifteen hours after slaughter all carcasses were transported to a pork boning room in Sydney. Forty eight hours after slaughter, ultimate pH and muscle lightness (L*) were measured after exposure to air for 10 min at 2° C on the *M. longissimus lumborum (*LL*), M. spinalis dorsi* (SD) and flank muscle. The *M. longissimus lumborum* and the collarbutt primal from each carcass were then vacuum packed, transported to Victoria and stored at 2° C for four weeks. Each pack was then opened and colour (LL and SD) and purge (LL only) measurements were made.

Table 1. Effect of abattoir (A) and vendor (V) on temperature (°C) and pH after slaughter and muscle lightness (L*), ultimate pH and purge (%) for the *M. longissimus lumborum (LL), M. spinalis dorsi* (SD) and flank after two days or four weeks of chilled storage.

		Abattoir A			Abattoir B				
	Muscle	V1	V2	V3	V4	V5	V6	s.e.d.	Significance
Temp at 2 h		27.7	27.9	27.8	29.3	29.2	30.7	0.47	A***,V*
L* - 2 days	LL	49.5	48.7	48.5	46.9	49.8	45.9	1.11	A*,V**
	SD	40.7	40.8	40.0	37.8	40.5	38.2	0.66	A***,V***
	Flank	41.9	42.9	41.2	40.2	40.7	42.1	0.61	A**,V**
Ultimate pH	LL	5.50	5.44	5.52	5.54	5.50	5.55	0.036	A*
	SD	5.81	5.84	5.9	5.96	5.99	5.98	0.057	A***
L* - 4 weeks	LL	51.1	51.7	49.9	49.1	52.4	48.8	0.99	V***
	SD	41.1	41.7	40.2	38.2	41.0	39.3	0.655	A***,V***
Purge (4 weeks)	LL	4.70	4.26	3.68	3.22	5.02	3.39	0.373	V***

*P<0.05; **P<0.01; ***P<0.001

Abattoir influenced the muscle temperature two hours after slaughter and muscle lightness in the LL, SD and flank muscles measured two days before slaughter. Differences in chiller regimes used at each abattoir (data not presented) may partly explain these carcass characteristics as well as differences identified between vendor groups for muscle colour measured two days after slaughter. The LL muscle became paler in colour with ageing for four weeks in vacuum bags and differences between vendor groups in muscle lightness were also observed. Vendor group, but not abattoir, influenced purge from the LL muscle following ageing in vacuum bags. The LL muscle was consistently paler (higher L* value) than the SD muscle after both two days and four weeks ageing. This 10 unit difference in L* values between the LL and SD is sufficient for a two toning issue to be identified by customers. Overall, pork from Abattoir B had consistently higher ultimate pH in the SD muscle, exacerbating the 'two toning' issue.

This study demonstrated that colour variation exists between the carcasses from different abattoirs and between carcasses from vendors within abattoirs. Therefore, carcass selection based on carcass weight and fatness alone is not enough to enable processors to supply pork of the required colour specifications to different markets. Considerable variation in meat colour between vendor groups further complicates the ability of processors to produce pork to market requirements. Currently, no differentiation in chiller practices occurs for carcasses destined for different markets. Further research is being carried out to optimise chilling conditions for pork of differing carcass and quality specifications to help improve quality consistency.

Supported in part by Australian Pork Limited.

Use of computer tomography in live pigs as an alternative to chemical carcass assessment

L.R. Giles, K.J. James, L.J. Barker and G.J. Eamens

Department of Primary Industries, Camden, NSW 2570.

While computer tomography (CT) has been used as an alternative to physical dissection in live pigs and carcasses, few studies have investigated whether CT imaging of tissue weight in live pigs could be used as an alternative to chemical carcass analysis. Jopson *et al.* (1995) used a combination of chemical analysis and physical dissection to record tissue weight in pig carcasses and found a close association with recorded tissue weight using CT imaging. The objective of this study was to test the hypothesis that CT imaging of muscle, fat and bone weight in live pigs is closely associated with chemical analysis of carcasses for protein, fat and ash weight.

Forty five male, female and castrate male pigs (hybrid, mainly Large White x Landrace) were allocated at 10 weeks of age and 32.4 kg ± 3.5 (mean ± SD) live weight to individual pens in four rooms maintained at 22° C. The pigs were fed a commercial, pelleted diet *ad libitum* that was adequate in digestible energy, with free amino acids added to maintain an excess to requirements. Water was provided using nipple drinkers. Nine pigs (three males, three females and three castrate males) were anaesthetised with an intravenous injection of Ketamine (10 mg) and Xylazine (0.5 mg) per kg live weight at close to each of five live weights (30, 60, 90, 120 and 150 kg). Cross-sectional images were collected along the whole body at intervals of 10 mm using a Picker (PQ 2000) spiral CT scanner at EMAI. On the day after CT scanning each pig was sedated with an intravenous injection of barbiturate and then exsanguinated and eviscerated to record carcass weight. The left side (including the head, skin and hair) was frozen, minced, mixed, sampled (1 kg) and freeze-dried. Each sample was analysed for nitrogen, fat and ash content. The CT cross-sectional images for each animal were eviscerated electronically and CT tissue volume was assessed using a Voxel Q workstation. Tissue weight was calculated from volume and density measurements based on the Hounsfield unit range (Hounsfield, 1980). The interaction between pig sex and chemical carcass components was not significantly different for CT muscle, fat and bone weight. Hence, the relationship between CT tissue weight and chemical carcass components was analysed independently of animal sex using linear regression (Table 1).

Table 1. Intercepts (a), slopes (b), ± standard errors (s.e.), coefficients of determination (R^2) and residual standard deviations (RSD) of live pig tissue weights recorded using CT imaging as a function of carcass weight and wet[1] chemical carcass components in 45 pigs from 30 to 150 kg live weight.

Dependent variable	Independent variable	a (s.e.)	b (s.e.)	R^2	RSD
CT carcass weight (kg)	Carcass weight (kg)	1.27 (1.93)	1.04 (0.02)	0.978	5.50
CT carcass muscle (kg)	Carcass protein[2] (kg)	-3.76 (3.92)	3.11 (0.24)	0.801	8.66
CT carcass fat (kg)	Carcass fat (kg)	-4.31 (1.12)	0.87 (0.04)	0.902	3.48
CT carcass bone (kg)	Carcass ash (kg)	-0.09 (1.14)	3.10 (0.39)	0.590	2.46

[1] Chemical carcass components corrected for sample moisture content. [2] Nitrogen x 6.25.

The hypothesis that CT imaging of tissue weight in live pigs is closely associated with the weight of chemical carcass components was supported (Table 1). Computer tomography was able to assess carcass weight accurately in the live animal (see Table 1) and CT muscle and fat weight were closely associated with carcass protein (R^2, 0.801) and chemical fat (R^2, 0.902) weight, respectively. Computer tomography assessment of carcass fat weight appeared to underestimate chemical fat weight in the carcass. This underestimation may be associated with the inability of CT imaging to detect all solvent-extracted fat components in bone and muscle. Bone weight had the lowest coefficient of determination (0.590) when regressed against carcass ash weight. Inherent sampling difficulty associated with the mincing of bone during chemical carcass assessment is a likely source of error when measuring carcass ash weight. Measurement of tissue weight in live animals using CT imaging offers a number of advantages over chemical carcass assessment, including retention of the live animal, a degree of automation, reduced labour and analytical costs, and the ability to store cross-sectional tissue images electronically for further analysis.

Supported in part by Australian Pork Ltd. and Pig Improvement Company, Australia.

References

JOPSON, N.B., KOLSTAD, K., SEHESTED, E. and VANGEN, O. (1995). *Proceedings of the Australian Association of Animal Breeding and Genetics* **11:**635-638.

HOUNSFIELD, G.N. (1980). *Journal of Computer Assisted Tomography* **4:** 665-674.

Australian Pork

Pig growth performance and carcass quality is not affected by feedstuff source

P.C.H. Morel, J.A.M Janz, R.W. Purchas, B.H.P. Wilkinson and W.H. Hendriks

Massey University, Palmerston North, New Zealand.

Animal byproducts are commonly used as sources of dietary protein and fat in New Zealand, where a typical finisher pig diet may contain tallow as a fat source and 10% meat and bone meal representing 30% of total dietary protein (Hendriks *et al.*, 2002). Considerable variation exists worldwide regarding the regulation of animal feeds and the safe use of byproduct feedstuffs (Machin, 2005) and the implications of future restrictions or bans on the use of animal byproducts in pig feeds must be considered. As few reports exist, this work was undertaken to compare the effects of diets based on either animal byproducts or plant material on pig growth performance and carcass quality.

Thirty-two female Duroc x (Large White x Landrace) pigs were fed from six weeks after weaning (live weight 11.4 ± 2.7 kg) to an average slaughter weight of 101.9 ± 3.4 kg and age at slaughter of 146.4 ± 8.4 days. Sixteen pigs received a diet with fat and protein sourced from a combination of animal (AN) and plant feedstuffs (barley + blood meal, fish meal, meat and bone meal, skim milk powder, soybean meal, tallow). The remaining sixteen pigs received diets based solely on plant (PL) feedstuffs (barley + soybean meal, soy protein isolate, peas, soybean and linseed oils). Feed intake and live weight were measured weekly throughout the trial on a pen basis during the weaner phase and individually for grower/finisher pigs. Pigs were slaughtered at a commercial abattoir and carcass parameters were collected on the processing line at grading.

Table 1. Effect of feedstuff sources of protein and fat on pig growth performance and carcass quality.

		Feedstuff source			
Growth parameter	Phase	Animal byproducts	Plant material	s.e.m.	P-value
Average daily gain (g)	Weaner	685	664	34.0	0.39
	Grower	899	895	10.1	0.80
	Finisher	890	895	23.8	0.90
Average daily feed intake (kg)	Weaner	1.15	1.10	0.077	0.65
	Grower	1.87	1.92	0.024	0.13
	Finisher	2.85	2.96	0.030	0.09
Gain to feed ratio	Weaner	0.61	0.59	0.022	0.56
	Grower	0.48	0.46	0.006	0.07
	Finisher	0.31	0.30	0.007	0.49

The time required to reach slaughter weight was unaffected by diet (AN: 145.4, PL: 147.9 d ± 2.1 SEM, P=0.40) and no significant differences in overall growth performance were observed. As is common with plant based diets for weaner pigs (Rodenhutscord *et al.,* 2002), the PL diet caused an early lag in growth before weaning, but no significant difference was observed by the end of this phase. Hot carcass weight (AN: 80.0, PL: 80.6 ± 0.90 kg, P=0.63), dressing percentage (AN: 79.0, PL: 78.6 ± 0.49 %, P=0.61), and fat thickness (AN: 11.9, PL: 12.0 ± 0.54 mm, P=0.86) were also unaffected by diet. Animal byproducts can be replaced by plant-based feedstuffs as sources of protein and fat in pig diets without affecting growth performance or carcass quality. and

Support provided by: Foundation for Research, Science and Technology & the New Zealand Pork Industry Board.

References

HENDRIKS, W.H., BUTTS, C.A., THOMAS, D.V., JAMES, K.A.C., MOREL, P.C.H. and VERSTEGEN, M.W.A. (2002). *Asian-Australasian Journal of Animal Science* **15**:1507-1516.

MACHIN, D.H. Accessed 15-04-2005 [On-line: http://www.fao.org/agrippa/publications/522_en.htm].

RODENHUTSCORD, M., ABEL, H. J., FRIEDT, W., WENK, C., FLACHOWSKY, G., AHLGRIMM, H. J., JOHNKE, B., KÜHL, R. and BREVES, G. (2002). *Archives of Animal Nutrition* **56**:67-91.

Feedstuff effects on quality and fatty acid profile of pork

J.A.M Janz, P.C.H. Morel, R.W. Purchas, B.H.P Wilkinson and W.H. Hendriks

Massey University, New Zealand.

Dietary source of protein (animal byproducts *vs.* plant material) does not influence pig growth performance and carcass quality (Morel *et al.*, 2005). In contrast, fat type has a direct impact on pork fatty-acid profile since fatty acids are absorbed unchanged from the monogastric gut (Enser *et al.,* 2000). However, few reports describe the effects of various feedstuffs on meat quality. The aim of this study was to examine whether animal- and plant-based feedstuffs influence pork quality and fatty-acid profiles.

Forty eight hours after birth the *longissimus* muscles of 29 female Duroc x (Large White x Landrace) pigs (slaughter weight 101.9 kg ± 3.9 sd) fed diets containing either animal byproducts (barley + blood meal, fish meal, meat and bone meal, skim milk powder, soybean meal, tallow) or solely plant feedstuffs (barley + soybean meal, soy protein isolate, peas, soybean and linseed oils) were measured for colour, pH, drip loss, cooking loss, Warner-Bratzler shear force and fatty acid profile using standard methods. Before and after slaughter management was identical for all the animals. A linear model with feedstuff source as a fixed effect was fitted to the data.

Table 1. Effect of feedstuff source of protein and fat on attributes of the pork *longissimus* muscle.

	Feedstuff source			
Attribute	Animal byproducts	Plant material	SEM	P-value
L* (colour lightness)	49.2	49.3	0.40	0.86
Chroma (colour intensity)	6.5	6.0	0.29	0.27
Hue angle,° (colour)	22.2	22.9	1.10	0.67
pH, 48 hour	5.53	5.57	0.04	0.48
Cooking loss (%)	29.5	29.7	0.51	0.80
Drip loss (%)	3.9	3.9	0.35	0.89
Shear force (kg)	7.4	7.6	0.39	0.66
PUFA : SFA	0.32	0.52	0.02	<0.01
C18:2 : C18:3	13.6	6.0	0.28	<0.01

None of the meat quality attributes measured in this study were affected by feedstuff source (Table 1). Similarly, Lettner *et al.* (2001) reported no differences for drip loss and taste panel scores for pork from pigs fed meat meal or soy-based diets. The use of a plant-based diet resulted in pork with more favorable fatty acid characteristics than pork from pigs fed a diet containing animal byproducts (Table 1). Pigs in the plant-only group produced pork with a PUFA:SFA ratio that was 63% higher and a C18:2 to C18:3 ratio that was 56% lower than pigs fed the diet based on animal byproducts. The PUFA:SFA ratio (0.32) of the pigs fed the plant-based diet was below the recommended ratio of 0.4, which would be desirable for the cardiovascular health of human consumers (Wood *et. al.,* 2003). Slight refinement of the proportions of dietary soybean and linseed oils could further reduce the linoleic to linolenic acid ratio (6.0) and bring it below the desired maximum ratio of 4.0.

Pork of acceptable quality can be produced by feeding pigs diets containing animal byproducts and/or plant material. A significant shift towards 'healthier' fatty acid characteristics (higher levels of unsaturated fats) was evident when only plant-based feedstuffs were used.

Support provided by: Foundation for Research, Science and Technology & the New Zealand Pork Industry Board.

References

ENSER, M., RICHARDSON, R.I., WOOD, J.D., GILL, B.P. and SHEARD, P.R. (2000). *Meat Science* **55**:201-212.

LETTNER, F., WETSCHEREK, W. and BICKEL, S. (2001). *Die Bodenkultur* **52**:247-254.

MOREL, P.C.H., JANZ, J.A.M., PURCHAS, R.W., WILKINSON, B.H.P. and HENDRIKS, W.H. (2005). In 'Manipulating Pig Production X', p.274. ed J. E.Paterson. (Australasian Pig Science Association: Werribee).

WOOD, J.D., RICHARDSON, R.I., NUTE, G.R., FISHER, A.V., CAMPO, M.M., KASAPIDOU, E., SHEARD, P.R. and ENSER, M. (2003). *Meat Science* **66**:21-32.

Author Index